INDIAN SOCIETY OF HYPERTENSION
World Hypertension League
CURRENT MEDICAL CONCEPTS
HTNJ
CrossMark

Review Article

Hypertension as a Cause of Dementia

Subhash Kaul

Department of Neurologist, Krishna Institute of Medical Sciences, Secunderabad, Telangana, India.

Abstract

Several observational studies have demonstrated a link between high blood pressure (BP) and memory decline. The association is more with midlife rather than late-life hypertension and with systolic rather than diastolic BP. Hypertension by causing vessel wall thickening, reduced luminal diameter and vascular obstruction can lead to vascular dementia. Brain infarcts by decreasing the brain reserve lower the threshold for neurofibrillary tangles and amyloid plaques to produce Alzheimer's disease. Longer and intensive lowering of systolic BP has been shown to reduce the prevalence of mild cognitive impairment and dementia.

Key words: Dementia, midlife, hypertension

Introduction

Much attention has been paid to kidneys and heart as targets of end-organ damage due to hypertension. However, both large and small artery brain vasculature are probably more vulnerable to this end-organ damage. Many prospective studies have addressed the relationship between high blood pressure (BP) and memory decline. The overall impression does suggest that midlife hypertension has a strong association with late-life dementia of both vascular dementia (VaD) and Alzheimer's disease (AD) type. The aim of this article is to review the available literature on the subject of hypertension as a cause of dementia.

Hypertension Dementia Studies

Many studies have examined the relationship between midlife hypertension and late-life dementia.[1-4] The Honolulu-Asia Aging Study (HAAS) studied this relationship in 3703 Japanese-American men aged 45–68 years who were followed prospectively for 26 years.[5] A strong association between midlife hypertension (>160/95) and dementia (both AD and VaD) was established. Other studies also found similar results[4] high systolic BP (SBP) combined with an elevated total cholesterol level, further increased the risk for AD or VaD.[6]

As opposed to midlife high BP, the association between late-life high BP and dementia is not very robust. Only two studies have identified an association between late-life hypertension and dementia.[7,8] The first among these was a community-based cohort of 1270 participants (aged >75 years) followed for 6 years, out of whom 339 subjects developed dementia.[7] Subjects with very high SBP (>180 mmHg) were at 1½ times at risk to develop dementia, whereas those with high diastolic BP (DBP) (>90 mmHg) were not associated with an increased risk. Surprisingly, low DBP (<65 mmHg) was associated with an increased risk of dementia. Second study comprising 382 subjects described an association of both elevated SBP and DBP with a subsequent diagnosis of AD and VaD.[8]

In another study comprising 2356 participants, the relationship between BP and the risk for dementia across a range of older ages (>65 years) was studied over 8 years.[9] The youngest age group showed a significant association between high SBP (>160 mmHg) and all-cause dementia. The risk estimates for dementia associated with SBP declined with advancing age. It, therefore, seems that longer exposure to systolic hypertension has the strongest association with dementia.

Mechanism of Hypertension-Dementia Connection

Persistently, high BP may cause vessel wall thickening, reduced luminal diameter, and occlusion of large as well as microvessels of the brain.[10] A single strategically located infarct can cause acute

Address for correspondence:
Subhash Kaul, Senior Consultant, Department of Neurologist, Krishna Institute of Medical Sciences, Secunderabad - 500 003, Telangana, India. E-mail: subashkaul@hotmail.com

Received: 05-05-2019; Accepted: 20-05-2019
doi: 10.15713/ins.johtn.0162

Innovative Nature Science Publishers

VaD. Accumulation of multiple relatively silent infarcts over the course of time can also eventually lead to VaD. The presence of cerebral infarcts also lowers the brain reserve and the threshold for neurofibrillary tangles and amyloid plaques to precipitate AD.[11-13] Hypertension has also been proposed to cause ischemic damage to the cornu ammonis 1 hippocampal sector, which is the prime site for AD-type neurofibrillary degeneration.[14] The autopsy follow-up of 243 participants demonstrated that elevated SBP in midlife was associated with vasculopathic changes, a lower brain weight, and greater number of neuritic (ß-amyloid) plaques in both the neocortex and the hippocampus.[15] A neuroimaging study has demonstrated an association between midlife untreated hypertension and hippocampal atrophy.[16] There is also evidence to suggest that hypoxia-induced factors may strengthen amyloidogenic mechanisms by inducing BACE (beta-site amyloid precursor protein cleaving enzyme) a protein associated with the production of ß-amyloid, resulting in expression of AD.[17,18] This evidence suggests that hypertension by causing cerebral ischemia may lead to the development of both VaD and AD. Of note, many studies have shown an association of AD with atherosclerosis, high cholesterol, diabetes mellitus, and obesity, all of which often coexist with hypertension.[19-22]

Dementia and Antihypertensive Therapy; Observational Studies

The data from observational studies are mixed, but in general there seems to be a trend in reduction of both VaD and AD prevalence with antihypertensive therapy. The Rotterdam Study, a community cohort of 6416 nondemented subjects followed up for 2.2 years, reported a significant association between antihypertensive therapy and reduction in VaD but not AD.[23] However, Baltimore Longitudinal Study of aging, and the cache county study found that the use of Antihypertensive (AH) therapy was associated with a reduced risk of developing AD.[24,25] A 5-year follow-up study on a community sample of 1617 African Americans demonstrated 40% reduction in risk of dementia by the use of medications that control vascular risk factors.[26]

The HAAS was conducted on a sample of Asian American men between 1965 and 1995.[5] The relationship between the use of AH drugs and hippocampal atrophy was analyzed in a random sample of 543 participants.[16] The risk of hippocampal atrophy was increased in patients who never received AH drugs compared to those who received AH therapy. A further report of 848 participants from the HAAS, who had a history of mid-life hypertension and were dementia-free in 1991 showed that longer duration of AH treatment was associated with a reduced risk of AD and VaD. Each year of AH therapy was associated with a 6% reduction in the risk for dementia, compared with those never treated.[27]

On the contrary, two population studies, which had enrolled patients >65 years of age, failed to show any association between AH therapy and dementia. The East Boston cohort (n = 634 subjects >65 years of age) and the Canadian study of health and aging (n = 3238 subjects, >65 years of age), with follow-up periods of 4 and 5 years respectively, demonstrated no benefit for AH treatment.[28,29]

Dementia and Antihypertensive Therapy; Randomized Controlled Trials

Several large trials on hypertension have evaluated the effects of antihypertensive drugs on cognition with mixed results. Most of these trials were primarily designed to study the effect of AH medication on cardiovascular and stroke outcomes. Cognitive outcomes were measured only as a secondary outcome.

Systolic Hypertension in the Elderly Program (SHEP)

This double-blind placebo-controlled trial (1991) included 4736 patients with a mean age of 72 years.[30] Active treatment consisted of the diuretic chlorthalidone, with the possible addition of atenolol or reserpine. SHEP failed to demonstrate a significant effect of antihypertensive treatment on the incidence of dementia, despite between-group BP differences of >10 mmHg SBP, and >4 mmHg DBP.

Medical Research Council (MRC) Treatment Trial of Hypertension Study

In this prospectively planned MRC trial of treatment in 2584 patients (age 65–74) with hypertension, subjects were randomized to a diuretic, ß-blocker, or placebo.[31] There was a mean fall in SBP following treatment of 33.5 mmHg in the diuretic group, 30.9 mmHg in the ß-blocker group, and 16.4 mmHg in the placebo group. Subjects were followed up for 54 months, and no significant difference in cognition was found between the two groups.

Systolic Hypertension in Europe Study (Syst-Eur)

The VaD project included in the Syst-Eur demonstrated for the 1st time a reduction in the incidence of dementia following AH treatment.[32] Participants with age above 60 years (n = 2418) with isolated systolic hypertension were randomized to placebo or a dihydropyridine-calcium channel blocker (DHP-CCB) nitrendipine with or without an angiotensin-converting enzyme (ACE) inhibitor (enalapril), or a diuretic (hydrochlorothiazide) or both drugs to achieve adequate BP control. The trial was stopped prematurely because active treatment resulted in a 42% reduction in the primary endpoint of fatal and nonfatal stroke. Nitrendipine was the only antihypertensive used in 60% of patients in the active treatment group. The incidence of dementia was reduced by 50% in the treatment group. All patients enrolled in this trial were invited to continue or commence the study medication for a further follow-up period of 2 years (Syst-Eur).[33] Follow-up showed that the long-term antihypertensive therapy reduced the incidence of dementia by 55% from 7.4

to 3.3 cases/1000 patient years. Both the incidence of AD and VaD were reduced. These results indicate that the treatment of 1000 patients for 5 years can prevent 20 cases of dementia.

SBP Intervention Trial (SPRINT)

A substudy of SPRINT MIND project showed that at 1 year, mean systolic BP was 121.4 mmHg in the intensive-treatment group and 136.2 mmHg in the standard group. There was a "significantly lower rate" of mild cognitive impairment (MCI) and a "non-significant reduction" in the primary outcome of probable dementia in the intensive treatment group. The secondary outcome of a combined outcome of MCI plus probable dementia was significantly lower in the intensive versus standard treatment group. The SPRINT MIND magnetic resonance imaging (MRI) trial found that treating with a systolic BP target <120 mmHg also reduced the rise in cerebral white matter lesion on MRI scans but found no significant change in total brain volume.[34]

Perindopril Protection against Recurrent Stroke Study (PROGRESS)

The PROGRESS study was a randomized, double-blind, and placebo-controlled clinical trial on 6105 patients from 19 countries (mean age 64 years).[35] The active treatment group received the ACE inhibitor perindopril with or without the diuretic indapamide. Following a mean follow-up period of 3.9 years, active treatment reduced the risk of cognitive decline in the whole population by 12% in the active treatment group. The effect was similar in hypertensive or non-hypertensive subjects. Combination therapy with perindopril and indapamide was more effective than monotherapy with perindopril alone in reducing the risk of dementia due to greater reduction of BP in former.

Study on Cognition and Prognosis in the Elderly (SCOPE)

The SCOPE study evaluated the effect of angiotensin receptor blocker candesartan, with or without diuretic, in 4964 non-demented (MMSE score >24) elderly (mean age 76 years), hypertensive patients. After 3.7 years of follow-up, there was no significant difference between the two groups for cognitive function and dementia.[36]

Hypertension in the Very Elderly Trial-cognitive Function Assessment (HYVET-COG)

The HYVET was designed to assess the risks and benefits of treatment of hypertension in the very elderly patients and included a cognitive assessment, the HYVET-COG.[37] Non-demented ($n = 3336$) patients with hypertension aged >80 years of age were randomly assigned to receive 1.5 mg slow release indapamide, with the option of 2–4 mg perindopril, or placebo. No statistical differences were found between treatment and placebo groups with regard to cognitive decline or dementia.

Do Antihypertensive Agents Vary in their Effect on Dementia?

CCB

A Cochrane review of the clinical efficacy of nimodipine in treating dementia, found benefit associated with nimodipine (90 mg/day at 12 weeks) compared with placebo on cognitive function, and this benefit was similar for AD and VaD.[38] Lipophilic CCBs cross the blood–brain barrier with ease enabling more local effects within the brain. It is hypothesized that DHP-CCBs exert these effects by correcting the cerebral hypoperfusion that can precede clinical symptoms of both AD and VaD. DHP-CCBs also appear to antagonize the ß-amyloid-induced vasoconstriction associated with AD.[39] The aging brain loses its ability to efficiently regulate intracellular calcium levels, leading to cell death[40] and contributing to the development of AD[41] It is hypothesized that DHP-CCBs may alter this disruption.[42]

ACE Inhibitors

Findings from the Syst-Eur, PROGRESS and HYVET trials suggest that ACE inhibitors with and without diuretics seem to reduce cognitive decline, especially in stroke-related dementia. Only lipophilic ACE inhibitors, capable of crossing the blood–brain barrier (e.g., captopril and perindopril) is associated with decreased rates of cognitive impairment and dementia.[43] Other postulated mechanisms to reduce the risk of cognitive decline include modulation of cerebral blood flow, pleiotropic effects on the musculoskeletal system and nervous system, or effects on inflammation and oxygen-free radicals.[44-46] The gradual increase of white matter intensities on MRI, which play a part in pathogenesis of both AD and VaD, is also believed to be modified by ACE inhibitors.[47]

Beta-blockers and Thiazides

A study of patients with cognitive impairment and dementia at baseline found that lipophilic central nervous system ß-blocker use was associated with poorer cognitive scores.[48] Studies have shown that adrenergic signaling plays a role in the retrieval of intermediate-term contextual memories because the hippocampus receives dense input from adrenergic terminals.[49] Theoretically, this process could be affected by the use of ß-blockers with adverse effects on cognition.

Thiazide and loop diuretics reduce potassium concentration in brain, which is associated with increased oxidative stress. Increased inflammation, platelet aggregation, and vasoconstriction, all of which are potential contributors to AD pathogenesis.[50-52] On the other hand, potassium-sparing diuretics do not adversely affect cognition and may, in fact, improve it.[25]

Summary

Midlife hypertension is a significant risk factor for the later development of both AD and VaD. There is less evidence for this connection with late-life hypertension. Accumulating evidence suggests that intensive treatment of high SBP with AH medications may lower the incidence of dementia in individuals at risk, particularly if treated for a long duration. One has to careful to avoid hypotension in elderly which in itself has been shown to be associated with dementia.

References

1. Whitmer RA, Sidney S, Selby J, Johnston SC, Yaffe K. Midlife cardiovascular risk factors and risk of dementia in late life. Neurology 2005;64:277-81.
2. Wu C, Zhou D, Wen C, Zhang L, Como P, Qiao Y. Relationship between blood pressure and Alzheimer's disease in Linxian County, China. Life Sci 2003;72:1125-33.
3. Yamada M, Kasagi F, Sasaki H, Masunari N, Mimori Y, Suzuki G. Association between dementia and midlife risk factors: The radiation effects research foundation adult health study. J Am Geriatr Soc 2003;51:410-4.
4. Kivipelto M, Helkala EL, Laakso MP, Hänninen T, Hallikainen M, Alhainen K, *et al*. Midlife vascular risk factors and Alzheimer's disease in later life: Longitudinal, population based study. BMJ 2001;322:1447-51.
5. Launer LJ, Ross GW, Petrovitch H, Masaki K, Foley D, White LR, *et al*. Midlife blood pressure and dementia: The Honolulu-Asia aging study. Neurobiol Aging 2000;21:49-55.
6. Kivipelto M, Helkala EL, Laakso MP, Hänninen T, Hallikainen M, Alhainen K, *et al*. Apolipoprotein E epsilon4 allele, elevated midlife total cholesterol level, and high midlife systolic blood pressure are independent risk factors for late-life Alzheimer disease. Ann Intern Med 2002;137:149-55.
7. Qiu C, von Strauss E, Fastbom J, Winblad B, Fratiglioni L. Low blood pressure and risk of dementia in the Kungsholmen project: A 6-year follow-up study. Arch Neurol 2003;60:223-8.
8. Skoog I, Lernfelt B, Landahl S, Palmertz B, Andreasson LA, Nilsson L, *et al*. 15-year longitudinal study of blood pressure and dementia. Lancet 1996;347:1141-5.
9. Li G, Rhew IC, Shofer JB, Kukull WA, Breitner JC, Peskind E, *et al*. Age-varying association between blood pressure and risk of dementia in those aged 65 and older: A community-based prospective cohort study. J Am Geriatr Soc 2007;55:1161-7.
10. Swales JD. Pharmacological treatment of hypertension. Lancet 1994;344:380-5.
11. Kalaria RN. The role of cerebral ischemia in Alzheimer's disease. Neurobiol Aging 2000;21:321-30.
12. Kokmen E, Whisnant JP, O'Fallon WM, Chu CP, Beard CM. Dementia after ischemic stroke: A population-based study in Rochester, Minnesota (1960-1984). Neurology 1996;46:154-9.
13. Tatemichi TK, Paik M, Bagiella E, Desmond DW, Stern Y, Sano M, *et al*. Risk of dementia after stroke in a hospitalized cohort: Results of a longitudinal study. Neurology 1994; 44:1885-91.
14. Schuff N, Capizzano AA, Du AT, Amend DL, O'Neill J, Norman D, *et al*. Different patterns of N-acetylaspartate loss in subcortical ischemic vascular dementia and AD. Neurology 2003;61:358-64.
15. Petrovitch H, White LR, Izmirilian G, Ross GW, Havlik RJ, Markesbery W, *et al*. Midlife blood pressure and neuritic plaques, neurofibrillary tangles, and brain weight at death: The HAAS. Honolulu-Asia aging study. Neurobiol Aging 2000;21:57-62.
16. Korf ES, White LR, Scheltens P, Launer LJ. Midlife blood pressure and the risk of hippocampal atrophy: The Honolulu Asia aging study. Hypertension 2004;44:29-34.
17. Zhang X, Zhou K, Wang R, Cui J, Lipton SA, Liao FF, *et al*. Hypoxia-inducible factor 1alpha (HIF-1alpha)-mediated hypoxia increases BACE1 expression and beta-amyloid generation. J Biol Chem 2007;282:10873-80.
18. Sun X, He G, Qing H, Zhou W, Dobie F, Cai F, *et al*. Hypoxia facilitates Alzheimer's disease pathogenesis by up-regulating BACE1 gene expression. Proc Natl Acad Sci U S A 2006;103:18727-32.
19. Hofman A, Ott A, Breteler MM, Bots ML, Slooter AJ, van Harskamp F, *et al*. Atherosclerosis, apolipoprotein E, and prevalence of dementia and Alzheimer's disease in the Rotterdam study. Lancet 1997;349:151-4.
20. Peila R, Rodriguez BL, Launer LJ, Honolulu-Asia Aging Study. Type 2 diabetes, APOE gene, and the risk for dementia and related pathologies: The Honolulu-Asia aging study. Diabetes 2002;51:1256-62.
21. Romas SN, Tang MX, Berglund L, Mayeux R. APOE genotype, plasma lipids, lipoproteins, and AD in community elderly. Neurology 1999;53:517-21.
22. Chandra V, Pandav R. Gene-environment interaction in Alzheimer's disease: A potential role for cholesterol. Neuroepidemiology 1998;17:225-32.
23. In't Veld BA, Ruitenberg A, Hofman A, Stricker BH, Breteler MM. Antihypertensive drugs and incidence of dementia: The Rotterdam study. Neurobiol Aging 2001;22:407-12.
24. Yasar S, Corrada M, Brookmeyer R, Kawas C. Calcium channel blockers and risk of AD: The Baltimore longitudinal study of aging. Neurobiol Aging 2005;26:157-63.
25. Khachaturian AS, Zandi PP, Lyketsos CG, Hayden KM, Skoog I, Norton MC, *et al*. Antihypertensive medication use and incident Alzheimer disease: The cache county study. Arch Neurol 2006;63:686-92.
26. Murray MD, Lane KA, Gao S, Evans RM, Unverzagt FW, Hall KS, *et al*. Preservation of cognitive function with antihypertensive medications: A longitudinal analysis of a community-based sample of African Americans. Arch Intern Med 2002;162:2090-6.
27. Peila R, White LR, Masaki K, Petrovitch H, Launer LJ. Reducing the risk of dementia: Efficacy of long-term treatment of hypertension. Stroke 2006;37:1165-70.
28. Morris MC, Scherr PA, Hebert LE, Glynn RJ, Bennett DA, Evans DA. Association of incident Alzheimer disease and blood pressure measured from 13 years before to 2 years after diagnosis in a large community study. Arch Neurol 2001;58:1640-6.
29. Lindsay J, Laurin D, Verreault R, Hébert R, Helliwell B, Hill GB, *et al*. Risk factors for Alzheimer's disease: A prospective analysis from the Canadian study of health and aging. Am J Epidemiol 2002;156:445-53.
30. Prevention of stroke by antihypertensive drug treatment in older persons with isolated systolic hypertension. Final results of the systolic hypertension in the elderly program (SHEP). SHEP cooperative research group. JAMA 1991;265:3255-64.

31. Prince MJ, Bird AS, Blizard RA, Mann AH. Is the cognitive function of older patients affected by antihypertensive treatment? Results from 54 months of the medical research council's trial of hypertension in older adults. BMJ 1996;312:801-5.
32. Forette F, Seux ML, Staessen JA, Thijs L, Birkenhäger WH, Babarskiene MR, *et al*. Prevention of dementia in randomised double-blind placebo-controlled systolic hypertension in Europe (Syst-Eur) trial. Lancet 1998;352:1347-51.
33. Forette F, Seux ML, Staessen JA, Thijs L, Babarskiene MR, Babeanu S, *et al*. The prevention of dementia with antihypertensive treatment: New evidence from the systolic hypertension in Europe (Syst-Eur) study. Arch Intern Med 2002;162:2046-52.
34. AAIC. Alzheimer's Association International Conference in Chicago, USA. Chicago: AAIC; 2018.
35. Tzourio C, Anderson C, Chapman N, Woodward M, Neal B, MacMahon S, *et al*. Effects of blood pressure lowering with perindopril and indapamide therapy on dementia and cognitive decline in patients with cerebrovascular disease. Arch Intern Med 2003;163:1069-75.
36. Lithell H, Hansson L, Skoog I, Elmfeldt D, Hofman A, Olofsson B, *et al*. The study on cognition and prognosis in the elderly (SCOPE): Principal results of a randomized double-blind intervention trial. J Hypertens 2003;21:875-86.
37. Peters R, Beckett N, Forette F, Tuomilehto J, Clarke R, Ritchie C, *et al*. Incident dementia and blood pressure lowering in the hypertension in the very elderly trial cognitive function assessment (HYVET-COG): A double-blind, placebo controlled trial. Lancet Neurol 2008;7:683-9.
38. López-Arrieta JM, Birks J. Nimodipine for primary degenerative, mixed and vascular dementia. Cochrane Database Syst Rev 2002;3:CD000147.
39. Paris D, Quadros A, Humphrey J, Patel N, Crescentini R, Crawford F, *et al*. Nilvadipine antagonizes both abeta vasoactivity in isolated arteries, and the reduced cerebral blood flow in APPsw transgenic mice. Brain Res 2004;999:53-61.
40. Khachaturian ZS. Calcium hypothesis of Alzheimer's disease and brain aging. Ann N Y Acad Sci 1994;747:1-11.
41. Kawahara M, Kuroda Y. Intracellular calcium changes in neuronal cells induced by Alzheimer's beta-amyloid protein are blocked by estradiol and cholesterol. Cell Mol Neurobiol 2001;21:1-3.
42. Pascale A, Etcheberrigaray R. Calcium alterations in Alzheimer's disease: Pathophysiology, models and therapeutic opportunities. Pharmacol Res 1999;39:81-8.
43. Ohrui T, Matsui T, Yamaya M, Arai H, Ebihara S, Maruyama M, *et al*. Angiotensin-converting enzyme inhibitors and incidence of Alzheimer's disease in Japan. J Am Geriatr Soc 2004;52:649-50.
44. Lipsitz LA, Gagnon M, Vyas M, Iloputaife I, Kiely DK, Sorond F, *et al*. Antihypertensive therapy increases cerebral blood flow and carotid distensibility in hypertensive elderly subjects. Hypertension 2005;45:216-21.
45. Cesari M, Kritchevsky SB, Baumgartner RN, Atkinson HH, Penninx BW, Lenchik L, *et al*. Sarcopenia, obesity, and inflammation--results from the trial of angiotensin converting enzyme inhibition and novel cardiovascular risk factors study. Am J Clin Nutr 2005;82:428-34.
46. von Haehling S, Sandek A, Anker SD. Pleiotropic effects of angiotensin-converting enzyme inhibitors and the future of cachexia therapy. J Am Geriatr Soc 2005;53:2030-1.
47. Dufouil C, Chalmers J, Coskun O, Besançon V, Bousser MG, Guillon P, *et al*. Effects of blood pressure lowering on cerebral white matter hyperintensities in patients with stroke: The PROGRESS (perindopril protection against recurrent stroke study) magnetic resonance imaging sub study. Circulation 2005;112:1644-50.
48. Gliebus G, Lippa CF. The influence of beta-blockers on delayed memory function in people with cognitive impairment. Am J Alzheimers Dis Other Demen 2007;22:57-61.
49. Murchison CF, Zhang XY, Zhang WP, Ouyang M, Lee A, Thomas SA. A distinct role for norepinephrine in memory retrieval. Cell 2004;117:131-43.
50. Ishimitsu T, Tobian L, Sugimoto K, Everson T. High potassium diets reduce vascular and plasma lipid peroxides in stroke-prone spontaneously hypertensive rats. Clin Exp Hypertens 1996;18:659-73.
51. Young DB, Ma G. Vascular protective effects of potassium. Semin Nephrol 1999;19:477-86.
52. Chen WT, Brace RA, Scott JB, Anderson DK, Haddy FJ. The mechanism of the vasodilator action of potassium. Proc Soc Exp Biol Med 1972;140:820-4.

How to cite this article: Kaul S. Hypertension as a Cause of Dementia. Hypertens 2019;5(3):99-103.

Source of support: Nil, **Conflicts of interest:** None