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Review Article

Lifestyles and Their Impact on Dementia

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Abstract: Dementia is the loss of cognitive functioning, with a significant disabling effect on a person's daily life and activities. The 2015 World Alzheimer Report estimated that 46.8 million people worldwide are living with dementia, and this number is projected to increase to 82 million by 2030 and to 152 million by 2050. The primary dementias are the most common and include those due to neurodegeneration (like Alzheimer's Disease) and due to vascular causes (such as post-stroke). Secondary dementias result from other causes, such as vitamin deficiencies, infectious diseases, and brain tumors. Dementia usually has no cure, and the treatment is symptomatic. The value of adopting healthy lifestyles has a significant impact on reducing the risk as well as the progression of dementia. This manuscript will primarily focus on the role of healthy lifestyles on Alzheimer's disease.

Keywords: Dementia, Alzheimer's disease, smoking, lifestyles, exercise, alcohol, diet, obesity.

INTRODUCTION

Dementia is a complex process, usually occurring in individuals after the age of 65 years [1]. Since the aging population is growing, the incidence and prevalence of dementia is expected to steadily increase worldwide [2]. The 2015 World Alzheimer Report estimated that 46.8 million people worldwide are living with dementia, and this number is projected to increase to 82 million by 2030 and to 152 million by 2050 [3]. Primary dementias can be broadly classified as neuro-degenerative or vascular [4]. In neurodegenerative dementias, Alzheimer's disease (AD) is the most common type, and accounts for 50–70% of dementia cases [5]. The annual healthcare expenditure for AD (in the US) patients was $277B in 2018 and is expected to go up to $1100B annually by 2050 [6]. AD is characterized by pathologies: β-amyloid plaque deposition and neurofibrillary tangles of hyperphosphorylated tau [7]. This disorder continues to increase in low- and middle-income countries [8]. Its etiology is unclear but appears to be related to a complex interplay between genetic and environmental factors [9]. AD produces cognitive impairment and functional decline and often leads to institutionalization [10, 11]. There is no cure for AD, and treatment remains symptomatic [12]. The quality of life of these patients is markedly reduced and AD remains a major cause of death [13]. The second most common neurodegenerative dementia is Lewy body dementia, and this comprises 4%-8% of all dementias [14-16]. Vascular dementia is the second most common form of dementia after Alzheimer's disease and affects almost a third of people over age 70 [17]. Its underlying cause is vascular – usually an infarct or a hemorrhage [18] and its prevalence ranges from 2.4% to 23.7% [19]. Vascular dementia is not characterized neuropathologically by an accumulation of abnormal proteins [20]. Vascular dementias are however progressive and degenerative [21]. Dementia that is mixed (AD and vascular) account for 4.1–21.6% of all dementias [22]. Secondary dementias result from other causes, such as vitamin deficiencies, infectious diseases, and brain tumors [23]. This manuscript will be dealing primarily with AD.

DISCUSSION

Five healthy lifestyles have been recognized as having a major impact on morbidity and mortality [24]. These behaviors are non-smoking, abstinence or low to moderate alcohol intake, a normal body mass index, regular exercise, and a prudent quality of diet [24]. According to Yanping *et al*. adherence to these lifestyles can greatly improve survival and provide several extra years of life, both in men and women [24].

The dietary guidelines for Americans recommend that the diet be well balanced, mostly plant-based, rich in fruits and vegetables, whole grains, fish,

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low in sugar and salt, and with the occasional intake of lean meats [25]. It should limit or eliminate trans-fats, saturated fats, fried foods, sodium, red meat, refined carbohydrates, and sugar-sweetened beverages [25]. These five lifestyles and their impact on dementia are discussed below:

Smoking

Smoking is the leading preventable cause of death worldwide [26]. The World Health Organization estimates that more than 7 million people die each year because of tobacco smoking, worldwide [27]. The high mortality is the result of smoking related increased risk and progression of several major diseases including dementia [28]. Smokers have a significantly higher risk of both neurodegenerative and vascular dementia [29-31]. Anstey *et al*. found that current smoking increased the risk of incident AD by 79% and by 27% for any dementia [32]. A meta-analysis of 37 prospective studies from Zhong *et al*. reported that smokers had a RR of 1.30 for dementia, 1.40 for AD, and 1.38 for vascular dementia [33]. More recently, Li *et al*. in a meta-analysis of 34 prospective studies reported that current smoking increased the risk of dementia (RR = 1.61 [34]. Never smokers have a lower risk of AD (HR 0.82) [35]. Exposure to passive smoke increases the risk of cognitive damage in almost a quarter of individuals [36] and may be associated with dementia [37]. Exposure to tobacco smoke, while in the womb, can cause harm to fetal brain development [38]. Quitting smoking decreases the risk of dementia. In a prospective Korean National Health Screening Cohort (N = 46,140 men), dementia risk was reduced after smoking cessation for 4 years or more (Hazard Ratio = 0.81) [39, 40]. A recent study from Japan (N = 12,489) reported that after 3 or more years of abstinence, the risk of incident dementia equaled that of never smokers [41].

Alcohol

Alcohol is protective for some diseases if it is taken in moderation - intake to 2 drinks or less in a day for men and 1 drink or less in a day for women [42]. However, excessive drinking (>60 g/day in men and >40 g/day in women), significantly increases the risk of short-term harm, such as injury and violence, as well as the risk of long-term chronic health problems, such as some types of cancer [43]. The relationship between alcohol and dementia is somewhat controversial [44]. Animal and cell culture studies [45-47] and several cross-sectional studies, cohort studies, and case-control studies have suggested a beneficial effect of low to moderate intake of alcohol on the brain and its function [48]8. A meta-analytic study by Anstey *et al*. have also confirmed the protective effects of alcohol on dementia [49]. However, heavy drinking is harmful to the brain [50-52]. In a 5-year follow-up study of 13,342 men and women, Piumati et al reported that cognitive function declined more with alcohol intake of 12 units per week when compared to those who drank less [53]. Schwarzinger *et al.* in a retrospective cohort study of 31 million patients discharged from French hospitals (1,109,343 with dementia) showed that patients with alcohol abuse disorder have a significant increase in dementia risk [54]. In the 23-year UK Whitehall study (9087 participants), abstinence or heavy drinking were both associated with a higher risk of dementia [55]. Overall, the association between alcohol intake and dementia, including AD, appears to be J or U-shaped with low or moderate amounts of alcohol being safe or even protective while high intake increasing neuronal cell death and neurodegeneration[56-58]. However, alcohol drinking should not be started to protect against cognitive decline [59].

Obesity

The worldwide prevalence of obesity (BMI>30kg/m2) nearly tripled between 1975 and 2016 and is still on the rise [60]. Obesity is associated with cardiovascular diseases, diabetes, osteoarthritis, and some cancers (such as endometrial, breast, ovarian, prostate, liver, gallbladder, kidney, and colon)[61-64]. Obesity is also associated with reduced brain function [65]. There is increased cognitive impairment and even dementia [66]. In a review of 19 longitudinal studies including 589,649 people aged 35 to 65 years, followed up for up to 42 years, increased midlife BMI was associated with late life dementia with a RR=1.33 [67]. There was no association between being underweight and dementia [67]. This was also reported in another metanalysis (1.3 million adults aged ≥18 years) where a higher body mass, assessed >20 years before the diagnosis of dementia, increased the dementia risk (HR = 1.16) [68]. These studies indicated that clinically apparent dementia is related to midlife obesity, and reports of no connection between obesity and dementia at a later age are confounded by a long preclinical period of the latter, during which time weight loss is common [69,70]. A more recent study (Whitehall II study of more than 10,000 men and women) confirmed that obesity (BMI ≥30 kg/m2) at a midlife age of 50 years, is a risk factor for dementia [71]. Several other studies have reported a connection between BMI calculated obesity and AD [72-74]. Visceral obesity is also strongly linked with AD [75, 76]. Whitmer *et al.* reported in a longitudinal study of 6,583 individuals, that those with the largest abdominal diameter appeared to have a three-fold risk of developing dementia when compared with those with the smallest diameter [75]. Lifestyle changes, including a reduction in body weight, can play an important preventive and therapeutic role in AD [77]**.** It is estimated in an Australian study that in 2050, dementia in old age can be reduced by 10% if midlife obesity is reduced by 20% [78]. A meta-analysis of seven RCTs (468 participants) and 13 longitudinal studies (551 participants) of overweight and obese adults without dementia, (mean age 50 years), showed that there was a significant improvement in attention and memory, executive function, and language in different categories of evaluation,

associated with weight loss [79]. Obesity induces adipokine dysregulation leading to central nervous system inflammation [80-82]. The resultant increase in microglia causes reduced synaptic plasticity and impaired neurogenesis [83]. Microglia also interferes with insulin action [83] and can result in Aβ accumulation and reduce the tau protein degradation seen in AD [84]. Obesity is also a well-known risk factor for DM [85], dyslipidemia [86], cardiovascular diseases [87], and other cerebrovascular diseases [88] - all known risk factors for AD.

Exercise

Physical activity (PA) is defined as any bodily movement produced by the contraction of skeletal muscles resulting in a substantial increase in resting energy expenditure [89]. Exercise is defined as 'any sport or activity that works large groups of muscles, is continually maintained and performed rhythmically [90]. Physical activity, including exercise, is beneficial to people suffering from Alzheimer's disease or other dementias [91]. Exercise helps stabilize and improve cognitive function [92-96]. Zheng *et al*. evaluated 11 studies involving 1497 participants and found that in patients with mild cognitive impairment, aerobic exercise improved global cognitive ability and memory [97]. Northey *et al*. after analyzed 36 studies and found that physical exercise, in people over the age of 50, improved cognitive function, irrespective of their baseline cognitive status [94]. Panza and colleagues in a meta-analysis of nineteen studies, which included 1,145 subjects, reported that exercise training may delay the decline in cognitive function that occurs in individuals who are at risk of or have developed AD [98]. In patients aged 60 or more, Falck *et al*. in a systematic review and meta-analysis of 48 studies, also concluded that exercise was associated with an improvement in cognitive function in this population [95]. Chen *et al*. recently performed a meta-analysis of 33 RCT studies and concluded that exercise interventions improve executive function [99]. In patients with AD, exercise also helps in decreasing neuropsychiatric symptoms (such as depression, confusion, apathy) [100] and induces a slower decline in activities of daily living[101]. These patients function better and notice a reduction in falls [100]. Patients with AD experience fewer side effects and demonstrate better adherence to medications, with exercise [102,103]. This also helps reduce the caregiver burden [101]. Exercise helps preserve neurogenesis [104] and neuroplasticity [105]. Exercise also helps improve comorbid diseases such as diabetes, hypertension, obesity, stress, depression, and inflammation, which are also risk factors for dementia [106].

Diet

It is estimated that almost 10% of the global burden of disease is related to improper diet [107]. It is also one of the leading causes of excess mortality [108]. Diet also affects cognition [109-125]. High caloric intake may result in obesity and increase the odds of cognitive impairment, as discussed above. Maintaining a normal BMI and avoiding visceral obesity by caloric restriction and exercise is therefore important. Several authors have reviewed the connection between the quality of diet and the risk or progression o of dementia [109] and found that plant-based diets, especially those rich in green leafy vegetables, soybeans, nuts, and omega-3 polyunsaturated fatty acids, along with a low intake of saturated fats, animal-derived proteins, and refined sugars may reduce cognitive decline and dementia [110-112]. Both the Mediterranean diet and the DASH diet may help preserve cognition [113-115]. AD is strongly connected with diabetes, obesity, and cardiovascular disease and the benefits of these diets may be partially modulated by its mitigating effects on these [116]. On the other hand, dietary intakes following a more inflammatory dietary pattern – higher intakes of red and processed meat, peas, legumes, and fried food, and lower intakes of whole grains increase inflammation[117] and are associated with a higher decline in several aspects of cognition, such as episodic memory, semantic memory, working memory and executive function[118]. Some studies have noted that the intake of folate, B vitamins, vitamin C, D, E, and selenium, may beneficially modify cognitive impairment and dementia [119-124]. Further, some multi-nutrient supplementation (docosahexaenoic acid and eicosatetraenoic acid; uridine monophosphate; choline; vitamins B12, B6, C, E, and folic acid; phospholipids; and selenium) may slow cognitive decline [125]. The World Health Organization, however, does not recommend dietary supplements for the management of dementia [126].

CONCLUSION

There is strong clinical evidence for the development and progression of dementia because of unhealthy lifestyles. Conversely, adherence to five healthy behaviors can, not only reduce the risk of dementia but also improve cognition. These five healthy behaviors are not smoking, alcohol intake in moderation, regular exercise, proper diet, and avoidance of obesity. These lifestyles help reduce several major diseases and the benefit thus accrued contributes towards the reduction in dementia.

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