Alzheimer’s Disease and Osteopathy

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Introduction

Alzheimer’s disease is the most common form of dementia (Alzheimer’s Association, 2013b). People that are afflicted with Alzheimer’s disease would show a characteristic gradual memory loss, declines in thinking abilities, and together with the occurrence of personality changes (Fisher_Center_For_Alzheimer's_Research_Foundation, 2012). At the moment, no cure is available for this progressive neurodegenerative disease. Alzheimer’s disease is most prevalent for seniors over the age of 65. However, early onset Alzheimer’s disease can affect individuals as early as in their 30’s (National_Institutes_of_Health, 2012a). Alzheimer’s disease is frequently undiagnosed. The reason is that there are currently no detectable biomarkers that can be used for diagnosis of the disease.

As a result, early symptoms of Alzheimer’s disease such as failure in forming new memory, to later ones including confusion, mood swings, language breakdown and long term memory loss can all be easily disregarded as features of aging (Waldemar et al., 2007). At present, current diagnosis is primarily based on behavioural and cognitive assessment. It is important to note that cognitive difficulties could surface up to eight years before a person fulfills the clinical criteria for Alzheimer’s disease diagnosis (Backman et al., 2004). This makes early diagnosis of Alzheimer’s disease difficult.

Osteopathy is a concept of osteopathy that was founded by Andrew Taylor Still. He believed in a therapeutics system that utilizes natural forces in healing instead of having the therapy based on drugs and chemical agents (Still, 1902). In later days, Leo Page also suggested that the practice of osteopathy with the goal of maintaining and restoring structural integrity and physiological function (Parsons and Marcer, 2006). Nowadays, osteopathy has its focus in emphasizing the relationship between body structure and function, their integrity and ability to heal itself. Manual osteopathy has now a therapy approach that is non-invasive and utilizes manual manipulation and modalities to help correct somatic dysfunctions to restore health and body balance.

With Alzheimer’s disease being an illness that has as yet a cure or reliable preventive measure, this thesis aims to examine how osteopathy could potentially be beneficial in the alleviation and in support of the healing of the disease.
**Pathology of Alzheimer’s disease**

The central nervous system is the organ system that is affected in Alzheimer’s disease. The exact causes and mechanisms behind Alzheimer’s disease are still elusive. In terms of cellular structural and functional changes, amyloid plaques and neurofibrillary tangles are two classic histopathological features in the brains of Alzheimer’s disease patients (Tiraboschi et al., 2004). Amyloid plaques and neurofibrillary tangles are both due to accumulation of aggregations of beta-amyloid and tau proteins, respectively, in the brains of patients with Alzheimer’s disease (Hashimoto et al., 2003; Muchowski and Wacker, 2005).

![Figure 1. Structural changes of nerve cells and the brain in Alzheimer’s disease](American_Health_Assistance_Foundation, 2012b)
Amyloid plaques formation is one of the hallmarks of Alzheimer's disease (American_Health_Assistance_Foundation, 2012b) (Figure 2). Their accumulation is found between neurons in the brain. Beta-Amyloid is a proteolytic product cleaved from the amyloid precursor protein (APP) (De Strooper, 2010). In Alzheimer's disease, beta-amyloid fragments aggregate into fibrils and form amyloid plaques in the brain.

Another type of aggregation in Alzheimer’s disease is the neurofibrillary tangles that are found as insoluble fibers in neurons (American_Health_Assistance_Foundation, 2012c) (Figure 2). They are also characteristics of the lesions that are found in the brain with Alzheimer’s disease (Brion et al., 2001). Tau is a protein that forms microtubules in the cells. Tau is present mainly in the axons (neuronal processes that conduct nerve impulses between nerve cells) of neurons with its role in maintaining the stability of microtubules (Duan et al., 2012). Microtubules are structures that help with nutrient and substance transportation. In Alzheimer's disease, the tau protein is abnormal and this contributes to the collapse of the microtubule structure (American_Health_Assistance_Foundation, 2012c).

Both of the above cellular structural changes in neurons would contribute to neuronal dysfunction, such as the loss of loss of synapse and synaptic function of neurons (nerve cells), and finally leading to neuronal cell death (Donev et al., 2009).

Figure 2. Formation of amyloid plaques and neurofibrillary tangles in Alzheimer’s disease (American_Health_Assistance_Foundation, 2012b)
Cellular structural changes would translate as gross morphological changes of brain structures. An overall shrinkage of brain tissue can be observed in Alzheimer’s disease (American Health Assistance Foundation, 2012c). Comparing the normal and the Alzheimer’s brain (as shown in Figure 3 below), we can see that the grooves or furrows in the brain (called sulci) are widened and that the folds of the brain (called gyrus) are shrunk. This demonstrates an overall reduction of brain volume. The ventricle of the brain (chambers of the brain that contains cerebrospinal fluid) is enlarged in the Alzheimer’s brain. Many areas of the brain including those that are responsible for memory and language are affected.

![Figure 3. Brain structural changes in Alzheimer’s disease](American_Health_Assistance_Foundation, 2012c)

**Chronological Progression of Symptoms and Long Term Complications**

Early symptoms of Alzheimer’s disease include:

- Confusion
- Disturbances in short-term memory
- Problems with attention and spatial orientation
- Changes in personality
- Language difficulties
- Unexplained mood swings

There are multiple stages of Alzheimer’s disease that follows the progression of Alzheimer’s disease (American Health Assistance Foundation, 2012a). According to the American Health
Assistance Foundation, it can generally be divided as mild, moderate and severe. It is important to note that Alzheimer’s disease does not affect everyone the same way, nevertheless their symptoms usually progress along the stages listed here:

Stage 1 (Mild):

- Early stage in the illness
- Can last from 2 to 4 years
- Symptoms:
  - Less energetic and spontaneous
  - Minor memory loss and mood swings
  - Slow to learn and react
  - May become withdrawn, avoid people and new places and prefer the familiar
  - Confusion
  - Difficulty in organizing and planning
  - Easily get lost
  - Poor judgment
  - Difficulty in performing routine tasks
  - Have difficulties in communication and the understanding of written material
  - Become angry and frustrated

Stage 2 (Moderate):

- The longest stage
- Can last for 2 to 10 years
- Symptoms:
  - Become clearly disabled
  - Can still perform simple tasks independently, but may need assistance with more complicated activities
  - Forget recent events and their personal history
  - More disoriented and disconnected from reality
  - Confusing memories of the distant past with the present
  - Reduced ability in comprehending the current situation, date and time
  - May have trouble recognizing familiar people
  - Speech problems (understanding, reading and writing are more difficult, and the individual may invent words)
  - May no longer be safe alone and can wander
- Awareness of their own loss of control may lead to depression, irritability and restlessness or apathy and withdrawal
- May experience sleep disturbances
- Have more trouble eating, grooming and dressing

**Stage 3 (Severe):**

- Final stage
- Can last from 1 to 3 years
- Symptoms:
  - May lose the ability to feed themselves, speak, recognize people and control bodily functions, such as swallowing or bowel and bladder control
  - Memory worsens
  - May become almost non-existent
  - Sleep often
  - Grunting or moaning can be common
  - Constant care is needed
  - Weakened physical state, patients may become vulnerable to other illnesses, skin infections, and respiratory problems (particularly when they lose the ability to move around)

Other than the symptoms and complications listed above, there are also other complications that can occur along with Alzheimer’s disease (Healthline_Networks, 2013). Surprisingly, the most common cause of death for patients afflicted with Alzheimer’s disease is aspiration pneumonia. Aspiration pneumonia is the inflammation of the lungs and airways to the lungs (bronchial tubes) from breathing in foreign material (PubMed_Health, 2012). This can occur to patients with Alzheimer’s disease when they experience difficulty in swallow right (Healthline_Networks, 2013). This would lead to swallowing of food or liquid down the trachea (windpipe) instead of the esophagus (food pipe), causing blockage, damage, injury, and/or infection.
Significant Causative Factors for Alzheimer’s Disease

By far, the exact cause of Alzheimer’s disease is still unknown. However, with increasing research focused in the understanding of the disease, some factors have been listed as correlated to the progression of the disease.

Contributory causes – age

The biggest risk factor, and an unavoidable one, for Alzheimer’s disease is aging (Ferri et al., 2005). Alzheimer’s disease is not observed in young individuals. Alzheimer’s disease is most prevalent for seniors over the age of 65. Early onset Alzheimer’s disease can affect individuals as early as in their 30’s (National Institutes of Health, 2012a).

Contributory causes – dietary

More recently, diet has been associated with the cause of Alzheimer’s disease. There is the suggestion that Alzheimer’s disease could be a metabolic disease similar to diabetes (Dillow, 2012). There is even suggestion of referring Alzheimer’s disease as type 3 diabetes. Evidence has shown that type 2 diabetes is common in the elderly and it is associated with higher risk of Alzheimer’s disease (Samaras and Sachdev, 2012).

It is still unclear on how diabetes is linked with cognitive decline in Alzheimer’s disease. It has been proposed that hyperglycaemia (high blood sugar condition), hyperlipidaemia (high blood lipid condition), hypertension (high blood pressure condition), and low-grade systemic inflammation are linked to structural changes and volume loss in the brain, as well as cognitive decline via macrovascular pathways (Kloppenborg et al., 2008; Reijmer et al., 2010).

Cholesterol is one of the known risk factor for Alzheimer’s disease (Grant et al., 2002). This can be contributed by high dietary intake of dietary sugar and saturated fat. Interestingly, lowering of cholesterol levels appears to reduce the risk of developing Alzheimer’s disease.

Contributory causes – environmental

Aluminum is linked to Alzheimer’s disease as one of the primary environmental factors (Grant et al., 2002). Aluminum causes neurotoxicity in patients undergoing long-term hemodialysis for chronic renal failure. Relative risk for developing Alzheimer’s disease also increases in communities with high water aluminum concentrations.
Other than aluminum, Alzheimer’s disease affected brain was found to have problems in metal trafficking causing redistribution of metals into inappropriate compartments of the nerve cells (Ayton et al., 2012). For the metals that are implicated, these include iron, copper and zinc. Long term elevation of these transition elements in the body may potentially contribute to Alzheimer’s disease (Koh et al., 1996; Lovell et al., 1998; Mantyh et al., 1993).

**Exciting causes – anger, grief, emotional trauma, stress responses**

Stress has been increasingly recognized as one of the causative factors that contribute to Alzheimer’s disease. Chronic psychosocial stress plays an important role in Alzheimer’s disease (Alkadhi, 2012). In a rat model, researchers have demonstrated that even mild chronic psychosocial stress can push towards Alzheimer’s disease. It seems that the stress hormone such as cortisol can affect the hippocampus and hypothalamus of the brain, leading to neuroinflammation and neuronal dysfunction favouring Alzheimer’s disease development (Ricci et al., 2012). In addition, individuals with genetic predisposition to depression may also have increased vulnerability to Alzheimer’s disease (Aznar and Knudsen, 2011).

All of the above suggested that anger, grief, emotional trauma or stress could all potentially contribute to development of Alzheimer’s disease.

**Specific causes - infection**

Viral infections is another environmental factor for Alzheimer’s disease (Grant et al., 2002). Herpes virus has been linked to Alzheimer’s disease. It appears that human herpes virus type 6 (HHV6) is present in a higher percentage of Alzheimer’s brains that that of age-matched controls.

**Specific causes - poison**

Metal poisoning could potentially contribute to development of Alzheimer’s disease. Please refer to the section “Contributory causes – environmental” above for information concerning metals and their implications in Alzheimer’s disease.
Specific causes - injury

Moderate and severe traumatic brain injury has been linked to increased risk of developing Alzheimer’s disease (Alzheimer's Association, 2013a). Increased risk was noted for older adults who had history of moderate traumatic brain injury, with a 2.3 times higher risk of developing Alzheimer’s disease than people with no history of such injury. With severe traumatic brain injury, the risk increased to 4.5 times.

Although exercise in general has been shown to be beneficial against Alzheimer’s disease (Radak et al., 2010), sports that caused repetitive cerebral concussions such as professional football players can lead to onset of dementia-related syndromes such as Alzheimer’s disease (Guskiewicz et al., 2005).

Predisposing cause – hereditary, genetic, congenital

There is a genetic element in the development of Alzheimer’s disease (National Institutes of Health, 2012b). The genetic component of Alzheimer’s disease is more critical for early onset types than late onset types.

Early onset Alzheimer's disease is defined as those occur in people from the age of 30 to 60. It only represents less than 5 percent of all people who have Alzheimer's. Most cases are inherited and are commonly known as “familial” Alzheimer's disease. It can be caused by any of the single-gene mutations reported so far on chromosomes 21, 14, and 1. The abnormal proteins that are made from these mutations include the abnormal amyloid precursor protein (APP), abnormal presenilin 1, and abnormal presenilin 2.

Late onset Alzheimer's disease are the predominant form of cases reported. It is defined as being developed after the age of 60. The causes of late onset Alzheimer's are still unclear. It is believed that there include a combination of genetic, environmental, and lifestyle factors that influence a person's risk for developing the disease. The single-gene mutations mentioned above for early onset Alzheimer’s disease do not seem to be involved in late onset version of the disease. Apolipoprotein E (APOE) has been reported as a genetic risk factor for late onset Alzheimer’s disease. APOE encodes a protein that is involved in transporting cholesterol and other types of fat in the bloodstream.
Alzheimer’s Disease and Osteopathy

In view of the fact that Alzheimer’s disease has yet a cure, natural and complementary medicine has been playing key supporting roles from the angles of prevention and symptomatic alleviation of associated pain and discomfort.

The roles of alternative medicine in the treatment of Alzheimer’s disease have been diverse. For example, dietary modification and supplementation can be effective in prevention by reducing the damaging oxidative stress in the body. Psychological consultation and mental activity based training could improve cognitive performance, supporting both prevention and recovery. Acupuncture and Traditional Chinese Medicine could assist by potential stimulating and improving the nervous system, while modulating the side effects of chemical based drug treatments. Social interaction and physical activity are also modalities with beneficial effects.

In the realm of osteopathy, osteopathic practitioners can play important roles in the management of Alzheimer’s disease. In the early text of osteopathy, the improvement of general blood circulation and the continued control of the blood supply to the brain and the correlative drainage has been considered a task for osteopathic treatment (Riggs, 1901). Interestingly, there was the suggestion that mental disease could be related to the disturbed innervations or vascular channel to the brain (McConnell and Teall, 1906). Building on this idea, manual osteopathic techniques with the goal of improving circulation and neural transmission for the brain, including the reduction of cervical and brachial obstruction, could be beneficial in restoring this concept of “innervations” and “vascular channel to the brain”.

The various osteopathic treatment techniques can aid in the correction of somatic dysfunctions and visceral damage in Alzheimer’s patient. Potentially, adjustments could restore body balance aiding circulation and the nervous system. Osteopathic techniques such as muscle energy techniques, cranio-sacral techniques, myofascial release and joint mobilization could all be beneficial.

Further research and investigation would be needed to further evaluate the efficacy of manual osteopathic modalities in aiding the management of Alzheimer’s disease. Manual osteopathy nevertheless opens up new ground for the care of patients of Alzheimer’s disease.
Reference


impairment in retired professional football players. Neurosurgery 57, 719-726; discussion 719-726.


