Alzheimer's Disease

Alzheimer' s Disease (AD) is by far the most common type of dementia. AD starts very gradually, sometimes insidiously, and progresses slowly but steadily. In 1907 a German Psychiatrist, Alois Alzheimer, first described the changes caused by this condition.

The disability experienced can vary from one person to another and may fluctuate from day to day. It is also important to note that symptoms can sometimes become worse in times of stress, fatigue or ill health.

Common difficulties experienced may include:

- memory loss
- impaired judgement
- personality changes
- difficulties with speech and conversation
- difficulty with decision-making
- wandering
- repetitive questions
- sleep disturbance
- depression
- suspiciousness
- aggressive behaviour
- incontinence

This list is by no means exhaustive and symptoms vary over time as the disease progresses and different areas of the brain are affected. The onset of AD generally occurs in later life (65 +) and its incidence increases with age. In some cases the disability associated with AD may be improved by drug treatments but not everyone with AD is suitable for such treatments.

Medication works by boosting levels of a chemical called acetycholine which is deficient in people with AD. It is important to remember that not everyone with AD who are offered medication responds positively.

Causes of Alzheimer's Disease

Unfortunately the cause of AD is largely unknown. Increasing age and family history are known to be risk factors. The impact of genetics on AD is quite complex and only a small number of families have a very strong genetic component to their AD.

There are a few families with autosomal dominant transmission, which means that the person with a parent with AD has a 50% chance of getting it. However, this is extremely rare and is characterised by early onset, usually before the age of 50.

A recent discovery has been that specific versions of the gene for a protein called Apolipoprotein E (alleles for ALP) that an individual carries, are related to the risk of developing sporadic (non-familial) AD. The gene for Apolipoprotein E is located on chromosone 19. However, not everyone with ApoE 4 will get a dementia so testing for Apo E 4 genotype is not predictive of AD.

Other possible non-genetic risk factors for AD include head injury with loss of consciousness, gender, women are more commonly affected than men and a lower level of education. However, research somewhat contests the association between educational level and AD. It has been noted that the lower risk of AD for people from a higher

educational background may be an artifact, since there is a likelihood that those with more education have a higher baseline level of functioning and therefore do not meet criteria for AD as readily as others.

Older people with Down's Syndrome are at risk of developing AD due to having a triple copy of chromosome 21, the chromosome on which the amyloid precursor protein (APP) is found. In fact it is well established that all persons with Down's Syndrome aged over 40 will have the neuropathology for AD.

The importance of early diagnosis

The diagnosis of AD is primarily made on the basis of symptoms. Diagnosis usually means that the person meets the criteria for a diagnosis of probable AD and this diagnosis is usually only 80 to 90% accurate. More definitive diagnosis requires the examination of the brain tissue either by brain biopsy or after death to demonstrate the characteristic features under microscope. Brain biopsy is rarely recommended in view of the risks and discomforts associated with the procedure.

Currently there is no single test that can definitely diagnose AD and the diagnosis is made after careful clinical consultation. However, it is most important to exclude other forms of dementia that may be treatable and even reversible. For this reason seeking an early diagnosis is very important and can help to empower the individual to take control of their legal and financial affairs and become actively involved in their care plans.

Neuropathology of Alzheimer's Disease

The diagnostic characteristic features of AD are plaques and tangles found on post mortem in the brain. Plaques are abnormal forms of protein. Amyloid is a medical term used to describe the protein deposited in the brain tissue and in blood vessels of people with AD. The specific amyloid protein in AD is called "beta amyloid" which is derived from a larger protein called amyloid precursor protein (APP). APP is normally produced by a number of different types of cells in the body its exact function remains unknown.

Dying brain cells can be found surrounding amyloid deposits in parts of the brain, which are important to memory and other cognitive functioning. Some believe that these amyloid deposits are the key factors leading to the development of AD.

Another characteristic of AD are tangles. The term tangle describes the appearance of dense proteins found within neuronal cells. These tangles can appear as bundles of threadlike structures. One particular protein component of tangles is called Tau. Tau protein found within tangles in AD differs from Tau protein found in normal brains because it has been chemically modified.
There is now an on-going debate among scientists over, which is more important in relation to the aetiology of AD, plaques or tangles. Some researchers argue that tangles correlate more clearly with the clinical features of AD, while others maintain that an increased production of amyloid protein represents a critical early step and that tangles mark the death of neurones from exposure to amyloid protein or other toxic compounds.