Normal ageing of the brain

The growth cycle is well known to all of us: when a baby is born, it is completely dependent on the mother; next, the child learns to recognize family members and express its need, then goes to school and university and, finally, grows up to be a healthy adult. Health remains stable for many years, till the onset of decline in bodily functions. The ageing process occurs in every living species, so also in human beings: graying of hair, wrinkling of the skin, and hardening of arteries, aches and pains in joints, weakening of eyesight. The aged often complain that their memory power has decreased over the past few years. While they recall events of the past, they tend to forget more recent events. Remembering names and finding the “right word” seem to be a problem but, as they have discovered, the words do come back later when they stop trying too hard. It is well known that learning gets harder as one grows older. These are the features of advancing years, which are to be expected, but are of no consequence, as they do not interfere with daily life. This basically implies that minor forgetfulness, such as forgetting where one has kept the keys, is of no consequence. Sometimes people get very concerned about minor forgetfulness that is completely normal in old age, and confuse it with Alzheimer’s disease (AD).

This growth cycle of development from birth to teenage to adulthood and the gradual decline with age is depicted in the following graph:

In some people, increasing age is accompanied by a loss of intellectual capability so marked that it becomes a disease. This very rapid decline is shown in the above graph as Alzheimer’s disease.

Between the inevitable consequences of ageing and Alzheimer’s disease is the “grey area” (shaded grey in the diagram on page 56) in which some people suffer loss of intellectual functions that is more than mild and yet not severe enough to be considered Alzheimer’s disease. Scientists are unsure of the terminology vis-à-vis these patients. Many terms are in use, for example, minimal cognitive impairment or benign senescence of old age. This is an area of intense research to determine whether these cases will eventually progress to Alzheimer’s disease and, if so, who will progress and why, or which of these cases remain in the “grey area”.

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Dementia vs Alzheimer’s disease

There is widespread misunderstanding that dementia and AD are the same. Many people use the terms interchangeably. But truly speaking, dementia is the end result of damage to the brain resulting in loss of intellectual function. There are many different causes of dementia, only one of which is AD.

Dementia

Dementia is a decline of intellectual function (medically called decline of cognition). Intellectual function has many components (memory, geographical orientation, problem solving, giving advice, calculations, etc). The most important component of intellectual function is memory, so people complain of “memory loss”. Thus, if a person is affected by dementia, he/she has deficits in many components of intellectual function. Each of these components can be checked separately by doctors or psychologists.

There are many causes of decline in intellectual function, i.e. many conditions can lead to dementia in a person, for example, iodine deficiency, brain tuberculosis, head trauma, multiple strokes, Alzheimer’s disease, and several other causes. Some of the causes of dementia can be prevented (iodine deficiency) or optimally treated (brain tuberculosis).

Alzheimer’s disease

Alzheimer’s disease is the commonest cause of dementia world-wide except in some communities where causes are endemic e.g. iodine deficiency disorders. The relative proportion of AD as the cause of dementia varies from 60 to 80% in different communities. The loss of intellectual function gradually progresses in AD, leading to increasing levels of disability to the point the person becomes bedridden and incapable of any meaningful function.

What happens to the brain in AD?

The brain shrinks in size. This shrinkage can be seen by doing a computerized tomography (CT) or magnetic resonance imaging (MRI) scan of the brain. But shrinkage of the brain is not diagnostic of AD. Many normal people, particularly the elderly have shrunken brains on CT/MRI but function completely normally. Thus the CT/MRI findings have to be clinically correlated.

Some abnormal proteins accumulate in the brain (e.g. beta amyloid, tau). But it has still not been established if the accumulation of these proteins “cause” AD. Moreover removing these proteins through chelating agents does not improve the person’s condition.

How is AD diagnosed?

The person should be evaluated by an experienced doctor who is familiar with memory disorders. The doctor should take a detailed history from family about the onset, progression and current condition in terms of intellectual function. Specific examples of patient’s deficits should be obtained.

The second step is “interacting” with the patient. This should be conducted in a calm
environment that is not stressful to the patient; the intention should be to obtain an objective status of the patient. It is inappropriate to subject the patient to a school examination-like situation.

The next step is to conduct select psychometric tests that have been validated to the culture of the patient, for example, it is inappropriate to ask an illiterate person to spell words backwards. Deficits in psychometric tests cannot be used as the only basis of diagnosis of AD. The findings must be correlated with the patient condition.

The next step is to conduct some blood tests, an electroencephalogram (EEG) and then radiological tests (CT/MRI) of the brain. With the availability of modern diagnostic tools, a study of the brain fluid (cerebrospinal fluid) through removing some of this fluid by a lumbar puncture is usually not needed unless some infection is being considered. There is almost no need for a brain biopsy, except in very specific conditions.

The final diagnosis is made by combining all the above information. Lastly, when in doubt, doctors will examine the patient periodically every three to four months. If the patient has AD, his/her condition will deteriorate over time.

**Pseudodementia (depression)**

Depression (an exaggerated form of sadness) often presents as “memory loss”. This is because the person suffering from depression is unable to concentrate on what is going on in the environment and thus is not able to register the circumstances. When the person tries to recall what had been said or happened there is no “memory”. This is incorrectly considered “dementia”. Truly this should be called “pseudodementia”, that is a false dementia and an inappropriate conclusion of dementia.

Another common cause that presents as “pseudodementia” is anxiety. With increasing awareness about AD and dementia, people begin to test themselves repeatedly to see if their memory is intact. This repeated testing of oneself leads to tremendous anxiety if there are any errors. This is what happens in an examination setting among children.

Anxiety and depression are two of the common causes of “pseudodementia” in a community setting, particularly among older people.

**Are all communities equally affected?**

In the last decade of the twentieth century, there was a belief that AD does not occur in developing countries. Researchers thus set out to conduct scientifically appropriate cross-national studies comparing select developing countries with developed countries. Studies comparing a community in India with a community in the United States of America, a community in Nigeria with a community in the United States and comparing the Japanese population with people of Japanese origin leaving in Hawaii were conducted. These studies conclusively found that AD did occur both in India and Nigeria and also in Japan but the risk of getting AD in these three countries was much lower than in Hawaii or mainland United States.

After rigorous investigations, it was found that these differences were indeed valid. The investigators from India developed a hypothesis that perhaps a gene-environment interaction is the cause of AD. This theory is based on the observation that although the genetic pool of the population under study both in India and the United States was similar, the difference in risk could be due to the lower cholesterol level in the Indian population. High cholesterol, the main risk factor for cerebrovascular disease has also been implicated as a risk factor for AD. Thus the investigators hypothesized that the lower cholesterol in the Indian, Nigerian and Japanese population reduced the risk of AD.
Another interesting hypothesis is the use of spices in certain countries. These condiments have “anti-oxidant” properties. There are numerous reports on the benefits of garlic and turmeric.

An interesting observation about lower risk in select countries is the fact that the risk of getting AD can change among migrants within two generations of migration. The basis of this change, despite maintaining the same genetic pool, could be the increasing level of cholesterol that frequently happens as migrants adopt the food habits of people living in developed countries, where the food consumed is high in cholesterol.

What are the risk factors for AD?

There is great fear among family members of patient with AD, particularly the children. The question most commonly asked is “What is my risk of getting AD?” It should be noted that only about 2% cases of AD are directly inherited from parents, that is if one of the parents has AD, children are likely to get it. This mode of inheritance is called autosomal dominant.

Another genetic contribution to the risk of AD is the genetic pool (apolipoprotein E4) combined with a high cholesterol level. The apolipoprotein gene is transmitted in the family but the cholesterol level is mediated by the diet of a person. This combination is called gene-environment interaction as discussed above.

There are certain “external” risk factors that traditionally cause strokes such as high cholesterol, cigarette smoking, diabetes, high blood pressure, sedentary lifestyle and obesity, etc. However, it is now believed that the same risk factors can cause AD independent of strokes.

Current treatment and care

It should be noted that currently there is no complete cure for AD, although a substantial amount of research is in progress.

There are certain medications that can enhance the intellectual functioning of the brain. These medications (choline esterase inhibitors) neutralise a brain chemical that is believed to be one of the factors implicated in the cause of AD. However, these medications are beneficial only in limited number of cases and have a marginal degree of benefit. They also have some side-effects. Thus the current recommendation is to try this medication for about three months. If they are beneficial they could be continued, if not they should be discontinued.

There are certain distressing symptoms such as restlessness, talking constantly, use of foul language, sexual misdemeanour, and not sleeping at night, etc. These symptoms can easily be controlled by giving small doses of psychotropic medication. It should be noted that sedatives are relatively contraindicated in patients with AD as they cause severe drowsiness.

Caring for a patient with AD imposes a huge burden on the family, particularly in a nuclear family. The burden of caring invariably falls on the lady of the house. Care of the care-giver is a very important issue and every effort must be made to ensure that the care-giver gets sufficient rest and time to relax and to get away from the duty of caring. A practical solution is to have a trained care-giver at home. Training programmes for such domestic helpers need to be developed. Another solution is to divide the care-giving activity among all the family members.

Old age homes, as exist in western countries, have a very limited role in our part of the world. Most elderly people expect to stay at home and to be cared for by their children. Moreover even if old age homes were to be built, it will be very expensive to keep a person there. Moreover, there is limited...
love and compassion in old age homes, which only a family can provide. The role of old age homes should therefore be limited to people who have no family members or for the elderly who are abused.

What you can do to protect yourself from Alzheimer’s disease

The most important thing to do is to lead a completely normal and healthy life. If one lives in fear of getting AD, one cannot enjoy the present and at the same time cannot prevent the future. The fact that there is no cure for AD should not make a person depressed or anxious. Worrying about the future affects the “mind”, which then affects the brain and the body.

Traditional practices such as yoga and meditation have been practised for centuries. These practices are good for the body and mind and are strongly encouraged. There is increasing scientific evidence of their benefits.

Control of risk factors such as cholesterol, blood sugar, blood pressure and obesity is highly desirable. Not only will this reduce the risk of AD but also reduce the risk of strokes, heart attack and numerous other diseases.

Lastly, maintaining a healthy lifestyle from the early years in life will contribute to a healthy old age, free of disease. A healthy lifestyle consists of nutritious diet, rich in fruits and vegetables; plenty of exercise appropriate for age; management of stress; good sleeping habits; and to gainfully engage with friends and family.

References and bibliography

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