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Review on Alziemers Disease

Advait Jagdish Rahate[1], Utsav Kumbhamwar[2], Parag Penulwar[3], Namdev Pastapure[4], Monika Bhagwanrao Ghode[5], Mrunali Gautam Wankhede[6]
Final Year Students, New Montfort Institute of Pharmacy, Ashti, Wardha[1,3,4,5,6]
advaitrahate786@gmail.com

Abstract: *Alzheimer's disease is a long-term illness with long prenatal and prodromal phases that last 20 years on average. The disease itself lasts for 8–10 years on average. The disease is thought to affect 10–30% of people over 65, and it happens to 1–3% of people that age. It is thought that more than 95% of people who get Alzheimer's have the sporadic form, which starts late (around age 80–90) and happens when the amyloid β (A^2) protein isn't cleared from the brain's spaces. Many genetic factors that put people at risk for random diseases have been found. Some people get the disease much younger (mean age of about 45 years) because they inherited changes in genes that affect how Aβ is processed. Using biomarkers in cerebrospinal fluid and PET, it is now possible to find Aβ buildup in the developmental and prodromal stages. There are a number of approved drugs that can help with some of the symptoms of Alzheimer's disease, but none of them can change how the illness works. The goal of management is on getting the patient's social networks to help and treating any other illnesses that may be present, like cerebrovascular disease..*

Keywords: Alzheimer's disease, Dementia, Memory loss,Cognitive decline, Neurodegenerative disorder, Amyloid plaques, Tau tangles, Brain atrophy, Risk factors.

I. INTRODUCTION

A lot of research has been done on the descriptive (rather than critical) epidemiology of Alzheimer's disease over the last 30 years. Unfortunately, most of them aren't very useful because the confounding variables of co-morbidities aren't always clear. This is especially true for cerebrovascular disease, which has been the main factor in descriptive epidemiological studies so far. There have been a lot of descriptive studies on "dementia," which give us an idea of how common cognitive impairment is in older people. However, the stated estimates of the incidence and prevalence of Alzheimer's disease, not dementia in general, should be taken with a grain of salt. The issue is the same when it comes to possible links with risk factors.

Luckily, new technologies are now available that can specifically detect Alzheimer's disease in living patients (rather than studies done after the fact), such as molecular PET imaging and levels of biomarkers A^2 and tau in the cerebrospinal fluid (CSF). These will help to get a better idea of how common and how often Alzheimer's disease happens. See FIG. 2 for the most up-to-date projections of the annual risk of sporadic Alzheimer's disease. Since Alzheimer's lasts on average for 10 years, the total mean incidence of 1-3 percent is in line with the overall prevalence of 10-30 percent in people over 65. 1–4. There aren't many accurate statistics on how common Alzheimer's disease is in people over the age of 90, but we need them in order to figure out how Alzheimer's disease is connected to the normal ageing process in the brain. Many people say that getting Alzheimer's is a natural part of getting older and that everyone over the age of 90 will show signs of preclinical, prodromal, or clinical Alzheimer's disease.

Post-mortem studies, on the other hand, show that the number of people who have Alzheimer's disease may drop off after age 98, while the number of people who have other neurological diseases, like TAR DNA-binding protein 43 (TDP43)-related hippocampal sclerosis, rises5 (FIG. 3). If this turns out to be true, it would put Alzheimer's disease in line with other neurodegenerative diseases, like Parkinson's disease, which have a clear peak frequency between the ages of 70 and 90, after which it drops sharply6–8. Alzheimer's disease and other types of dementia affect women more than men. For example, 66% of dementia deaths in Australia are women9). It hasn't been proven yet whether this is because guys die more often from causes other than dementia or Alzheimer's disease. Our only way to learn more about

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"genes versus environments" in the cause of Alzheimer's disease is to accurately find out how common it is by using new technologies (like Aβ PET imaging and Aβ levels in the CSF) along with analytical epidemiology methods10,11. Random Alzheimer's disease seems to happen at about the same rate in all of the world's populations, according to the latest evidence12. If this result is confirmed by more research, it will make the case for genes over environment stronger. Diabetes mellitus is one of the risk factors for Alzheimer's disease that may be able to be changed.

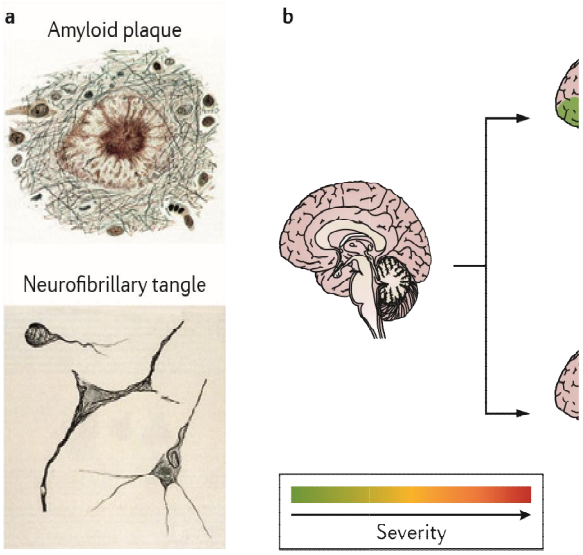

Fig 1.1 The pathological evolution of Alzheimer's disease.

II. MECHANISMS/PATHOPHYSIOLOGY

Studying how Alzheimer's disease works in people will likely reveal new information about how the illness starts, how to diagnose it, and how to treat it (TABLE 1). At this point, there is strong proof that three things—Aβ, APOE, and tau—play a role in Alzheimer's disease. Alzheimer's disease is characterised by abnormal protein deposits in neurones (called neuro fibrillary tangles) and in the extracellular space (called cerebrovascular, diffuse, and neuritic plaques) (FIG. 4). These deposits are synaptic loss and selective neuronal death, as well as a decrease in certain neurotransmitters. A in dementia with Alzheimer's There is a lot of evidence to back the idea that A² is a peptide that causes Alzheimer's disease. The main part of plaques, Aβ, is cut from APP into a group of peptides that are all different in length (between 38 and 43 amino acids) and have some minor differences in how they work16,17. Also, isoforms that have been shortened or changed at the N-terminus have been found18. Studies on proteolytic processing have shown that Aβ is a common byproduct of APP metabolism and is made in large amounts by neurones and other cell types throughout a person's lifetime. APP's role in neurones is still unknown, but it may have something to do with synapse plasticity. Several pieces of evidence point to Aβ building up and changing shape to forms with a high ²±sheet structure as being important to the development of Alzheimer's disease19. A² is thought to play a big role in Alzheimer's disease based on research that looks at people who have a genetic form that starts early.There are changes in three different genes (APP, PSEN1, and PSEN2) in more than half of people with DIAD20. The majority of mutations lead to too much Aβ being made, especially Aβ42, which is made up of 42 amino acids and is more likely to clump together16. Most APP mutations change how APP is processed, which makes the plasma level of A²42 to A²40 higher in people who have these mutations21,22. Furthermore, changes in PSEN1 and PSEN2 lead to higher A²42/A²40 ratios23. The Dominantly Inherited Alzheimer Network (DIAN) study24 and a meta-analysis25 both showed that the type of mutation and the Aβ42/Aβ40 ratio can be used to predict the average age at which dementia starts. The rise in the Aβ42/Aβ40 ratio can be seen in the culture supernatants of cells that were treated with mutant APP or PSEN1 constructs and in living mouse models21,26. The biochemistry of Aβ deposits in 30 DIAD types also shows that all of them have more A²42 deposits than A²40 deposits (REF. 27). It may be most important to note that people with these

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mutations have higher Aβ42/A²40 ratios in their plasma and higher levels of Aβ42 production in their central nervous system (CNS)20,28. Even though DIAD is not common, the fact that changes in three different genes cause similar changes in the ratio of Aβ products shows that there is one final common pathway in the development of Alzheimer's disease.

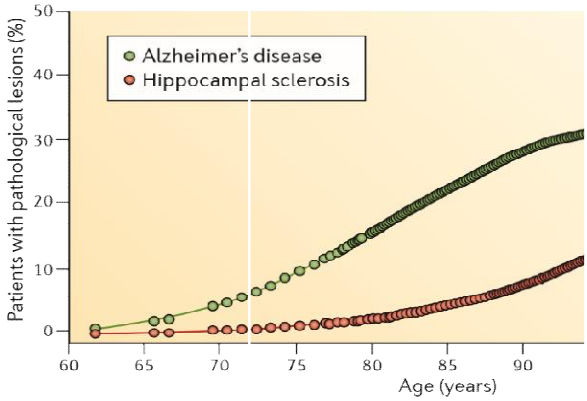

Fig 1.2 Co-morbidities with Alzheimer's disease in advanced ages

Along with the changes in DIAD, people with Down syndrome have an extra copy of chromosome 21, which includes APP. This causes their bodies to make more Aβ, and by the time they are 35, all of these people have clinical changes that look like Alzheimer's disease29. Also, a change in APP that lowers the production of Aβ has been shown to protect against late-onset Alzheimer's disease in a very impressive way30. Human A kinetics: making A, moving A, and making sure A is clear. It has been possible to measure marked proteins in the brain and spinal cord to learn more about how Aβ moves in Alzheimer's disease31,32. The stable isotope tagging kinetics (SILK) method was used to find out how much A² was made and removed from the CNS31. The results showed that CSF A² levels changed very quickly in healthy people, with an A² half-life of about 9 hours. When drugs that target Aβ formation were tested, they were shown to lower the production of Aβ33. Newer research suggests that people with even very mild sporadic Alzheimer's disease have less A² clearance in the brain, but their average generation doesn't change much32. It's still not clear what causes this drop in Aβ clearance, but it might have something to do with APOE and the fact that microglial cells and macrophages aren't breaking down the extracellular Aβ clumps properly.

III. APOE AND ALZHEIMER'S DISEASE

The biggest genetic risk factor for getting Alzheimer's disease is APOE on chromosome 19. Triglyceride-rich lipoproteins are broken down normally with the help of APOE. The first thing that was shown to connect APOE to Alzheimer's disease pathology was APOE immunoreactivity in Aβ deposits and neurofibrillary tangles, which are typical of Alzheimer's disease pathology34. Also, differences in the APOE transcriptional regulatory area have been linked to Alzheimer's disease35.

Two hundred amino acids make up APOE. In people, there are three common isoforms that are only different by one or two amino acids each: APOE2 (Cys112 and Cys158), APOE3 (Cys112 and Arg158), and APOE4 (Arg112 and Arg158). Each allele is found in 7% of people with APOE2, 78% of people with APOE3, and 15% of people with APOE4 who are from Europe36. The changes to the amino acids affect APOE's overall charge and structure. This changes how it binds to cell receptors and lipoprotein particles, and it may also change how stable it is and how fast it is made and cleared out. A lot of APOE is found in the brain, where it is mostly made by astrocytes and microglia. Some APOE production can happen in neurons37 under certain situations. APOE only comes from inside the blood–brain barrier38 and is found in the cerebrospinal fluid (CSF) at levels of about 5µg per ml. Population studies have shown that APOE4 raises the risk of getting Alzheimer's disease (one gene raises the risk three times, and two alleles raise the risk twelve times)39 and is linked to getting Alzheimer's disease earlier in life.

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40, 41. On the other hand, APOE2 lowers the chance of getting Alzheimer's disease. 42, 43. It is thought that the APOE4 allele is responsible for about half of all cases of spontaneous Alzheimer's disease44. Researchers have found that different types of human APOE can lower the amount of Aβ in neuritic plaques and delay the start of Alzheimer's disease in mouse models45–48. This is because APOE2 is less common than APOE3 and APOE4. Also, human APOE3 lowers the amount of A² that is deposited.

Query	Pathogenesis	Pathophysiology	Biomarkers	Pathology	Clinical and cognitive
Assay	Genetic testing of risk factors and protective factors	Aβ, tau and APOE metabolism in the brain, FDG PET and functional connectivity	Biochemical measures in the CSF	Aβ and tau PET imaging, and structural MRI	CDR-SB and neurological examination, and psychometrics
Result	Mutations in *PSEN1*, *PSEN2*, *APP* or *APOE* allele (2, 3 or 4)	Overproduction or impaired clearance of Aβ and aggregation tau in the brain Hypometabolism in the parieto-occipital cortex	Decreased Aβ42 levels, and of increased T-tau and P-tau levels in the CSF	Aβ aggregation, tau aggregation, and hippocampal and cortical atrophy	Memory, attention, executive cognitive dysfunction, functional impairment and dementia staging

Table No 1.1 Diagnostic And Clinical Tests For Alzheimer's Disease

Pathological diagnosis	T-tau levels (in the CSF)	P-tau levels (in the CSF)‡	Abundant isoform§
Primary tauopathies			
Frontotemporal lobar degeneration associated with *TAU* (also known as *MAPT*) mutations	Normal	Normal P-tau$_{181}$	3R, 3R plus 4R, or 4R
Argyrophilic grain disease	NA	NA	4R
Sporadic multiple system tauopathy	Normal	Normal P-tau$_{181}$	4R
Pick's disease	NA	NA	3R
Progressive supranuclear palsy	Normal	Normal P-tau$_{181}$	4R
Corticobasal degeneration	Normal-to-mild increase	Normal P-tau$_{181}$	4R
Secondary tauopathies			
Alzheimer's disease	Moderate increase	Moderate increase of both P-tau$_{181}$ and P-tau$_{231}$	3R and 4R
Creutzfeldt–Jakob disease	Very marked increase	Normal P-tau$_{181}$ and normal-to-mild increase of P-tau$_{231}$	NA

Table No 1.2 Diagnostic And Clinical Tests For Alzheimer's Disease

in mouse models that rely on the dose, with the least amount of deposition seen when two APOE3 alleles are present48. Overall, this shows that the type and amount of APOE in the CNS are very important for Aβ buildup and nerve cell death. There have been a lot of studies using cell culture and transgenic animals to look into how APOE4 might be linked to Alzheimer's disease. Among these are studies that look into how APOE4 acts as a pathological chaperone for Aβ, affecting the clearance and deposition of Aβ and ultimately leading to plaque formation46,49–52; studies that look into changes in tau phosphorylation and neurofibrillary tangle formation49,53; and studies that look into changes in

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lipid metabolism, which stops neurites from extending54–56. The APOE4 version doesn't change the production of Aβ50, but it can greatly increase the buildup of Aβ in animal models of Alzheimer's disease46. In line with this, Aβ40 clearance from the CNS to plasma is slowed down in a way that is unique to each allele (ApoE4>ApoE3 or ApoE2), showing that APOE plays a part in Aβ clearance, with the isoform57 determining how well it works.

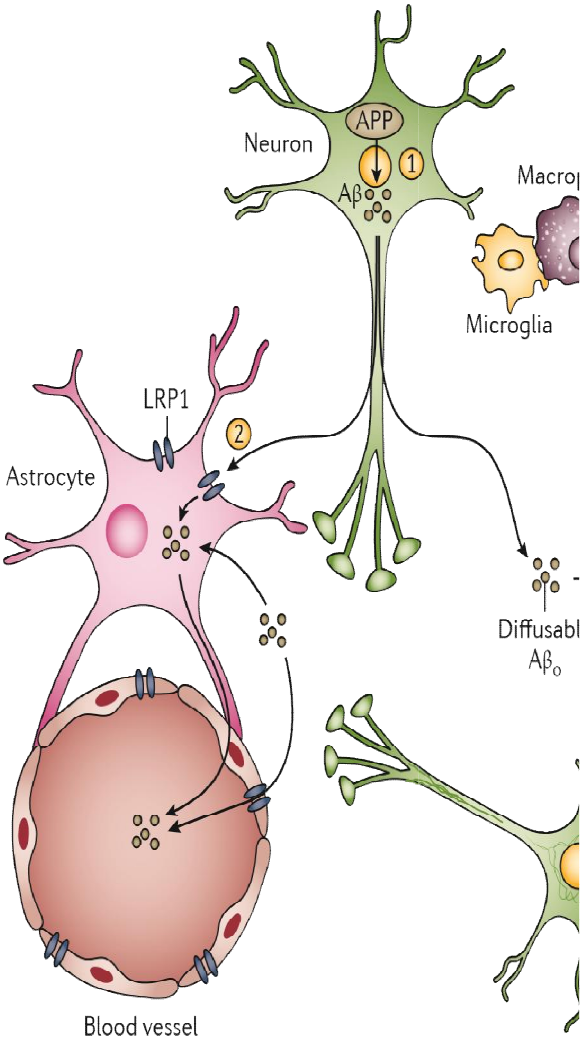

Fig no 1.3 Pathways leading to plaques and tangles form the basis of the amyloid-β theory of Alzheimer's disease.

IV. DIAGNOSIS, SCREENING AND PREVENTION

Findings It's hard to tell if someone has Alzheimer's disease just by looking at them60. This is true both in the early stages, when people only have mild memory problems, and in the later stages, when they have dementia. In fact, 35% of people who were hospitalised and thought to have Alzheimer's disease actually had negative A² PET scans and were wrongly diagnosed with the disease61. Brain diseases like cerebrovascular disease and hippocampal sclerosis make this problem even worse. A SPECT scan. Pittsburgh compound B (PiB) was created. It is a radioactive (carbon 11) version of the fluorescent amyloid dye thioflavin±T62 that can cross the blood–brain barrier and binds to fibrillar Aβ very strongly. This made it possible to image Aβ in living things with PET63–65 Scientists have learnt a lot about the link between Aβ levels and cognitive loss and neurodegeneration in the early, early-stage, and late stages of Alzheimer's

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disease over the past ten years thanks to studies using PiB. These studies have shown that Aβ buildup starts decades before dementia and happens before cognitive loss and brain atrophy. They have also shown that genetic factoraffect these connections.

A PET scan can show how quickly someone with mild cognitive impairment will move to dementia due to Alzheimer's disease in longitudinal studiesA² PET imaging has made it possible to do tests on people with Alzheimer's disease who aren't showing any symptoms yet and has made it easier to choose the right people for tests on people with early-stage or mild Alzheimer's disease. In the early stages of Alzheimer's, there are tests that can show if A is building up in the brain, which are used to diagnose the disease. These tests use PET imaging or CSF analysis. If you have a positive Aβ PET scan, it means that your CSF has low amounts of A²42. You can find both of these things up to 15 years before you start showing signs of Alzheimer's disease-related dementia.

Both tests can help confirm that someone has Alzheimer's disease, but Aβ PET imaging may be better for tracking changes in the A² burden over time and is more closely linked to the level of cognitive damage in the early stages of Alzheimer's disease80. On the other hand, A²42 levels Table 2 | The range of molecules found in main and secondary tauopathies *Diagnosis of disease The amounts of T-tau and P-tau in the cerebrospinal fluid (CSF) are high. Main tauopathies Degeneration of the frontotemporal lobar area linked to TAU (also called MAPT) genes Normal All right P-tau181 3R, 3R + 4R, or 4R Disease of argyrophilic grains NA NA 4R Multiple system tauopathy that comes and goes Normal All right Pick's disease cause by P-tau181 4R NA NA 3R Getting worse supranuclear palsy Normal All right 4.P-tau181 Loss of corticobasal tissue Normal to mild growth Not abnormal P-tau181 4R Other tauopathies Having Alzheimer's Moderate growth Both P-tau181 and P-tau231 3R and 4R showed a moderate rise. The disease Creutzfeldt–Jakob A very clear rise Normal levels of P-tau181 and a normal to mild rise in P tau231 NA 3R means three times; 4R means four times; CSF means cerebrospinal fluid; NA means not analysed (no accurate information available); P-tau means phosphorylated tau; and T-tau means total tau. Tau clumping and building up inside cells can happen either directly, where tau lesions are the main sign of a disease, or as a result of other changes, like when A builds up (like in Alzheimer's disease) or PrPSc (an abnormal form of the cellular prion protein) builds up (like in Creutzfeldt–Jakob disease). It's interesting that T-tau levels rise in the CSF in secondary tauopathies. Certain changes that happen after tau is translated, like phosphorylation on acids 181 and 231 (P-tau181 and P-tau231, respectively), are very different in these situations. ¦ The relative amounts of the 3R and 4R tau isoforms change a lot, but the amounts of the other tau isoforms don't change at all. PRIMER 6 | 2015 | THIS BOOK Visit www.nature.com/nrdp Copyright 2015 by Macmillan Publishers Limited. Some people think that the CSF might be more sensitive in the early stages of the disease81. Making A² PET ligands that are labelled with fluorine-18 available to the public has made it easier to identify Alzheimer's disease in the clinic and before it gets worse. 68,82–84. Aβ PET imaging and histopathological analysis side by side in Phase III trials showed high sensitivity (88–98%) and specificity (88–100%) for finding mild or frequent Aβ plaques in the nerves83–85. Large-scale studies of Alzheimer's disease, like the Alzheimer's Disease Neuroimaging Initiative (ADNI) and the Australian Imaging, Biomarkers, and Lifestyle study of ageing (AIBL)87, have also added good PET ligands for imaging tau aggre gates.

These studies will show how Aβ buildup is related to tau aggregation and what role each plays in cognitive loss and neurodegeneration. The CSF sample for Alzheimer's disease. The CSF reflects the biology of the brain, and it can easily talk to the interstitial fluid in the brain. In clinical neurology, lumbar puncture is a popular way to get cerebrospinal fluid (CSF). Several biomarkers for Alzheimer's disease have been found, such as Aβ42, T¬tau, and P¬tau. The most common tests are those that look for P-tau at Thr181 (REF. 88). If we want to know how the levels of these biomarkers change in CSF, we need to know how they do it. This is because these biomarkers can be used in clinical studies to look into the molecular disease mechanisms in Alzheimer's disease directly in humans and in clinical trials to find and watch the biochemical effects of drug candidates (TABLE 3). The amount of Aβ42 in cerebrospinal fluid (CSF) has been repeatedly linked to the number of plaques found after death89 and the retention of amyloid ligand on Aβ PET imaging. This suggests that this biomarker shows where the pep tide is deposited in the brain. Not only do CSF levels of T±tau rise in Alzheimer's disease, but they also rise in neurodegenerative diseases that don't have tau damage, like Creutzfeldt–Jakob disease92. This suggests that T±tau shows how badly neurones and axons are dying in general. Aside from tau, there are more visinin-like protein 1 (VLP1)93 and neurofilament light polypeptide (NFL)94 in the

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cerebrospinal fluid (CSF) of people with Alzheimer's disease. These proteins help predict both the rate of clinical decline and the rate of neurodegeneration91,94. Also, the finding that tau can be released from cultured cells95 and mouse neurons96 makes me wonder if people can also release tau from whole neurones into the CSF. This needs to be confirmed, though. It's also interesting that tau secretion would explain why all young, healthy people have amounts of tau that can be measured in their CSF. Last but not least, it has been shown that the amount of P±tau in the cerebrospinal fluid (CSF) is linked to neurofibrillary tau disease after death97, the rate of hippocampal atrophy in the brain98, and rapid clinical progression99. Low levels of CSF A²42 and high levels of T±tau and P¬tau are often referred to as the "Alzheimer's disease CSF profile." This profile is 85–90% sensitive and specific for Alzheimer's disease, and when analysed together, it performs better as a diagnostic tool than any of the CSF biomarkers alone.

These biomarkers help tell Alzheimer's disease apart from other important diagnoses, like depression and Parkinson's disease. P±tau levels also help a lot with telling Alzheimer's disease apart from other dementias, like frontotemporal lobar dementia and Lewy body dementia98,102. The ability of these CSF biomarkers to identify has been proven in cohorts103 that were confirmed by autopsies. They were just as good at telling the difference between groups as studies that only used clinical diagnoses. It's not clear how useful it is to check CSF tau levels to find people who have Alzheimer's disease in the early stages since there is a strong link between Aβ42 and Aβ PET59,80, and these tests have almost the same diagnosis accuracy for Alzheimer's disease.

The main thing that T¬tau and P¬tau seem to do, though, is help predict development. Aβ biomarkers, like CSF Aβ42 or Aβ PET, become positive many years before the disease shows any symptoms. However, high levels of CSF T¬tau and P¬tau make it easier to predict when the disease will get worse during a time window that is useful for patients104–106. So, the new criteria from the International Working Group for Alzheimer's disease say that people with Alzheimer's should be diagnosed by having low levels of CSF A²42 and either high levels of T¬tau or high levels of P¬tau.

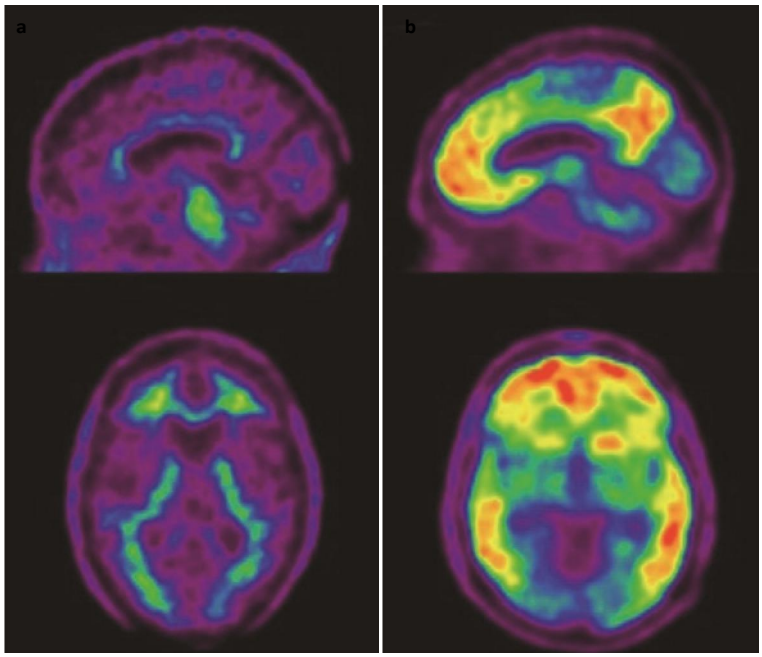

Fig no 1.4 Amyloid-β PET imaging.

Researchers published the first study on CSF biomarkers in the early stages of sporadic Alzheimer's disease in 2003. They found that CSF Aβ42 levels dropped but not Aβ40 levels did in older patients who were cognitively normal but developed dementia during a 3-year follow-up periodPeople with low amounts of CSF A²42 were eight times more likely to develop dementia, but none of the people with high levels of CSF A²42 developed dementia. In this group of

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people, CSF tau levels did not show a link to future dementia107,108. A later study showed that low levels of CSF A²42, but not changes in tau, could predict dementia more than 8 years before it Other early-stage studies back up the idea that average CSF Aβ42 levels are a better indicator of future cognitive decline than Ttau or P-tau levelsThere are more mixed results from research that looked at CSF in people with DIAD. These studies are based on looking at biomarkers that are linked to the estimated age of start in people with APP or PSEN1 mutations who don't have any symptoms. In the first study, mutation carriers who were more than 10 years before the expected age of onset had marked increases in the CSF levels of Ttau and Ptau but no change in A²42 levels. A bigger followup study, on the other hand, found marked changes in

Principle	Method	Cohort	Biomarkers	Comment
Diagnostics				
Diagnosis of Alzheimer's disease in clinical routine	CSF samples as part of the clinical work-up	Patients with MCI or mild-to-moderate dementia	Aβ42 Aβ42/Aβ40 ratio T-tau P-tau	Aβ42 is the first biomarker that becomes positive during disease progression High T-tau and P-tau levels are more predictive than Aβ42 levels for predicting the progression of cognitive deficits during a clinically relevant time window (1–2 years)
Enrichment of patients with Alzheimer's disease in clinical trials	CSF samples taken during the screening period, before enrolment into a trial*	Phase II and Phase III trials, including patients with early-onset Alzheimer's disease dementia or MCI		
Theragnostics				
Provide evidence of target engagement in humans	CSF samples taken before study initiation, at time points during the trial and at the end of the study Currently, all samples are analysed in one batch at the end of the trial. With technical improvements in assays, it will be possible for real-time evaluations to guide adaptive clinical trial design‡	Phase I trials on healthy volunteers or patients with Alzheimer's disease Phase II and Phase III trials on patients with early-onset Alzheimer's disease dementia or MCI	Aβ42 Aβ40 sAPPβ Aβ oligomers	Amyloid biomarkers may provide evidence for target engagement of an Aβ-specific drug, such as BACE1 inhibitors. Direction of change may depend on the mechanism of action A change in amyloid biomarkers indicates target engagement but does not predict corresponding downstream drug effects or symptomatic effects A change in Aβ oligomers may provide evidence of target engagement for Aβ immunotherapy regimes (in patients with Alzheimer's disease)
Provide evidence of downstream drug effects on neurodegeneration and molecular	CSF samples taken at study initiation and at the end of the study, and if possible at time points during the trial	Phase II and Phase III trials on patients with early-onset Alzheimer's disease dementia	T-tau P-tau	Reduction in T-tau levels suggests that the drug affects the intensity of the neurodegenerative process, whereas a decrease in P-tau

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pathology	All samples analysed in one batch at the end of the trial‡	or MCI		levels affects tau phosphorylation or possibly tangle formation
			H-FABP VLP1 SNAP25 Neurogranin	Additional biomarkers that reflect neuronal and synaptic degeneration, but are not directly involved in Alzheimer's disease pathogenesis, may provide independent evidence for downstream drug effects
Longitudinal clinical studies				
Examine the temporal evolution of Alzheimer's disease via biomarkers Identify which biomarker changes first during the preclinical phase of Alzheimer's disease and at what time point	CSF samples taken at multiple time points during the study Other Alzheimer's disease biomarkers (MRI measures, and Aβ and tau PET imaging) and cognitive function evaluated at the same visits	Elderly population followed longitudinally until a significant proportion develop cognitive symptoms or dementia	Aβ42 and Aβ40 Aβ oligomers T-tau and P-tau H-FABP and VLP1 SNAP25 and neurogranin Inflammatory and glial biomarkers	These biomarker data will provide information on the time course for, and inter-relation between, pathogenetic events during the preclinical and clinical course of Alzheimer's disease, and how biomarker changes correlate with cognitive deterioration

Table no 1.3 Applications for cerebrospinal fluid biomarkers for Alzheimer's disease

V. CONCLUSION

In conclusion, Alzheimer's disease is a complicated neurological disorder that gets worse over time and has a big effect on memory, thinking, and daily life. As of now, there is no cure, but study has helped us learn more about what causes it, including genetic, environmental, and lifestyle factors. Cognitive tests and brain images are two important diagnostic tools that help find the disease early on, which leads to better treatment and care plans.At the moment, treatments focus on managing symptoms and slowing the progression of the disease. However, there is hope for future breakthroughs thanks to potential research into drug therapies, lifestyle changes, and early detection methods. Also, caring is still a big problem, which shows how important it is to have complete support systems for both patients and their families.More study and clinical trials on Alzheimer's are being done to help us learn more about the disease. Eventually, we hope that better treatments or even ways to prevent it will be found. Even though there are problems, people with Alzheimer's are still determined to find answers, make life better, and eventually find a fix.

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