View metadata, citation and similar papers at core.ac.uk

brought to you by CORE

provided by DigitalCommons@University of Nebraska

University of Nebraska - Lincoln

DigitalCommons@University of Nebraska - Lincoln

Library Philosophy and Practice (e-journal)

Libraries at University of Nebraska-Lincoln

2020

Bibliometric review on Classification of Alzheimer's Disease

Nishad Vyas
Symbiosis International University, nishad.vyas@sitpune.edu.in

Keta Modi
Symbiosis International University, modi.keta@sitpune.edu.in

Saanil Khanna
Symbiosis International University, saanil.khanna@sitpune.edu.in

Chinmay Katpatal
Symbiosis International University, katpatal.chinmay@sitpune.edu.in

Mrinal Bachute Dr
Symbiosis International University, mrinal.bachute@sitpune.edu.in

Follow this and additional works at: https://digitalcommons.unl.edu/libphilprac

 Part of the Library and Information Science Commons

Vyas, Nishad; Modi, Keta; Khanna, Saanil; Katpatal, Chinmay; and Bachute, Mrinal Dr, "Bibliometric review on Classification of Alzheimer's Disease" (2020). *Library Philosophy and Practice (e-journal)*. 4843.
https://digitalcommons.unl.edu/libphilprac/4843

Bibliometric review of Classification of Alzheimer's Disease

ABSTRACT

Alzheimer's Disease (AD) is said to be the most prevailing Neurogenerative Disease. The neurogenerative disease is stated in a gathering of disorders that affect the brain. Deep Learning, the avant-garde machine learning approach is well prominent and this approach has shown wonderful performance compared to traditional machine learning techniques to identify convoluted structures in complicated high dimensionality data especially within the realm of computer vision. The application of the Deep learning approach is to have an early observation and mechanized classification of Alzheimer's Disease. Nowadays, it has gained a great amount of recognition as neuro-imaging techniques have generated an extensive range of multimodal neuro-imaging data. Deep learning Techniques and neuro-imaging data for diagnostic classification of Alzheimer's Disease was carried out and has been scientifically reviewed by a publication. Scopus and Google Scholar hunt was used to find some useful papers on deep learning techniques for AD. These papers were judged, assessed, and classified with the algorithms and neuro-imaging techniques and then the discovery was summarized. The objective of this project is to discover the early symptoms of the disease which are varying from age to age while classifying different stages of it along the way and enhance the prediction and accuracy to our extent.

1. INTRODUCTION

Alzheimer's disease is further manageable if the symptoms of Mild Cognitive Impairment (MCI) are in early stage. This disease is fundamentally associated with the fluctuations in brain which causes the deficiency of brain structure including the failing of neurons. In accordance with the Neurologists statements, there is no cure for this disorder. On the contrary unearthing early symptoms can help in early diagnosis of the disease. There are certain treatments which can put back the symptoms if they are looked after within the premature phases of the disease. Hence, an early identification of AD may be a major aspect for the patients affected by it. This disease is misperceived with standard ageing outcomes and hence Electroencephalography (EEG) has been present and defined as a constructive

technique which will help to smoothen the progress of premature diagnosis while an efficient reason is it's simple to practice.

EEG is employed to image brain activities and it registers the brain signal by means of electrodes affixed to the scalp. In addition to this, advanced neuroimaging techniques like resonance Imaging (MRI) and Positron Emission Tomography (TMI) have also been advanced and utilized within the diagnosis of AD. Deep learning methodologies like Convolutional Neural Network (CNN) or Recurrent Neural Network (RNN) which uses neuro-imaging data exclusive of extracting pre-processing for attribute selection have produced a greater accuracy for AD classification. Analysis of AD applying structural MRI and hybrid arrangement for creating a distinction among NC, MCI, and AD patients. EEG are regularly exercised for positive correlation of brain functionalities, which may stand utilized for monitoring brain motion, Non-linear analysis of EEG data displays the unique attributes to discover the diagnosis of neurological disorders, like Alzheimer, Epilepsy, and Parkinson’s. Numerous irregularities obtained in EEG signals of patients affected by AD embrace decelerating of EEG signals, moderated complexity of EEG signals and disconcertion in EEG synchrony.

1.1 HISTORICAL BACKGROUND

Alois Alzheimer, a German doctor, revealed the first instance of Alzheimer's disease in 1907. He initially observed Auguste Deter, a 51-year-elderly person, in 1901. Auguste's spouse Karl carried her to a psychological emergency clinic after she started displaying bizarre conduct, including hiding items, undermining neighbors, and blaming her husband of adultery. She additionally lost the capacity to do day by day exercises for example, cooking and housework. Auguste went under Alzheimer's consideration at a psychological clinic in Frankfurt. There he noticed and recorded her standards of conduct: she could talk yet not keep in touch with her own name, she could name items, for example, a pencil yet not the food she was eating, she was obliging occasionally however noisy and hostile at other times. He determined Auguste to have "presenile dementia".

Upon her passing in 1906, Alzheimer's biopsy of her mind uncovered diffuse cortical decay and "specific changes in cortical cell bunches". Alzheimer depicted plaques and tangles of nerve filaments which scientists would distinguish in the 1980's as beta amyloid plaques and neurofibrillary tangles of tau. That year, Alzheimer gave a presentation on Auguste at a German psychiatry conference, asserting these cortical injuries to be the reason of her

indications. He distributed a research paper the following year, and a psychiatry textbook in 1910 named the problem 'Alzheimer's Disease.' The clinical symptomatic measures for AD were normalized in the U.S. in 1984. They were updated in 2011 and 2018 to make separate judgments for the preclinical, mild cognitive impairment (MCI) and dementia phases of AD furthermore, to perceive the role of biomarkers in AD analysis.

1.2 PATHOGENESIS

AD is a perplexing, multifactorial, neurodegenerative infection, coming about because of convoluted communications of one's hereditary makeup, education, age, and climate. Numerous speculations have established the framework to pick up agreement of the etiology of the infection, with one of the eldest being the cholinergic speculation. This theory depends on the way that AD patients show decrease in action of choline acetyltransferase and acetylcholinesterase in the cerebral cortex contrasted with the typical cerebrum. Postmortem brain tissue from patients with AD affirmed the decreased neurotransmitter pathway activity, uncovering that degeneration of cholinergic neurons and loss of cholinergic neurotransmission essentially contributes to the cognitive impairment found in those with AD.

The Tau speculation has likewise been proposed, considering AD histopathology uncovers intraneuronal neurofibrillary sores comprised of tau proteins. Tau proteins are essentially found in neurons and are engaged with the assembly and stabilization of the neuronal microtubule organization. Tau protein becomes obsessive when the phosphorylation guideline gets unchecked and hyperphosphorylated tau proteins polymerize into fibers and become neurofibrillary tangles. This prompts breakdown of the underlying and administrative activities of the cytoskeleton and at that point prompts irregular morphology, axonal vehicle, also, synaptic capacity of neurons, consequently prompting neurodegeneration.

Figure 1: The amyloid (Aß) cascade hypothesis

These earlier speculations made ready to the broadly acknowledged hypothesis for the pathogenesis of AD: the amyloid cascade hypothesis. This hypothesis credits clinical sequelae of the sickness to the overproduction or diminished freedom of amyloid beta (Ab) peptides, which at that point prompts expanded statement of Ab, besides, prompting neuronal harm (Figure 1). The length of Ab shifts relying upon the posttranslational cleavage pattern of the transmembrane amyloid forerunner protein (APP). Ab is produced by cleavage of APP by means of one or the other b-or g-secretases, coming about in the scandalous insoluble Ab fibrils. Two principal types of Ab polymers assume an immediate part in the pathology of AD: Ab40 and Ab42. Ab40/Ab42 at that point oligomerizes, ventures to synaptic clefts, and meddles with synaptic flagging. These in the end further polymerize into insoluble amyloid fibrils that total into amyloid plaques. Inside the plaques, Ab peptides in b-sheet compliance polymerize into basically unmistakable structures, including fibrillar, protofibers and polymorphic oligomers. It is the deposition of these plaques diffusely all through the cerebrum that lead to microglial enactment, cytokine discharge, reactive astrocytosis, and an in general inflammatory reaction. These underlying changes lead to synaptic and neuronal loss and inevitable gross cerebral decay. Then again, should APP be handled by a-secretase in the solid grown-up, solvent b-amyloid is delivered, which has been connected to assume a part in neuronal versatility/endurance, is defensive against excitotoxicity, is significant for

ahead of schedule CNS improvement, and has been demonstrated to be significant for advancing neural connection development. The hereditary qualities of AD ought to likewise be considered to assume a persuasive part in the pathogenesis, close by irritation, apoptosis, and plaque development. The APP quality situated on 21q21, referenced above, was the primary found causative quality of AD. Advances in hereditary research have recognized two unmistakable types of AD: Familial Alzheimer's Disease (FAD) and Sporadic Alzheimer Disease (SAD), with the last making up the dominant part of cases. Significant advances during the 1990s and mid 2000s uncovered that FAD is the consequence of autosomal predominant changes in APP, PSEN1 and PSEN2 qualities, situated on chromosome 21, 14, and 1, separately. Even more explicitly, PSEN1 and PSEN2 contain the vital amino corrosive deposits needed for the reactant dynamic site of gamma-secretase. Certain transformations of these qualities lead to expanded creation of Ab peptide and neurodegeneration. Undeniably more regularly, the genetic danger factor for SAD was recognized as the sort e4 allele on chromosome 19, of the gene for apolipoprotein E (APOE), a low-thickness lipoprotein transporter dwell. APOE is available in approximately 50- 60% of patients with AD contrasted with 20-25% in sound older grown-ups without the historical backdrop of familial AD.

1.3 TESTING

AD remains a clinical diagnosis and is reliant on a detailed history, cognitive assessment, and physical exam. Structural imaging is also an important component of the assessment for AD. Atrophy of the medial temporal lobes on magnetic resonance imaging (MRI) is considered a diagnostic marker for the mild cognitive impairment stage of AD. Similarly, hypometabolism in the parieto-temporal association area, posterior cingulate and precuneus on fluorodeoxyglucose PET imaging is associated with AD.

2. PRELIMINARY DATA COLLECTION

Publication databases are often accessed using the library portals of the schools or by individually registering on the websites. Several methods are available to retrieve data from the quality databases like Scopus, Clarivate, Science Direct, Mendeley, Research gate, Google Scholar etc. Scopus database accessed on 19th September has been considered during this paper because it is that the largest among the peer reviewed databases. List of the keywords used is given within the following section.

2.1 Important keywords

The important keywords required to carry the search were “Alzheimer’s”, “Machine Learning” and “MRI”. The other keywords are enumerated in table 1.

Table 1 List of Keywords

Keywords	Number of Publications
Alzheimer's Disease	126
Machine Learning	106
Magnetic Resonance Imaging	99
Neurodegenerative Diseases	69
Neuroimaging	57
Brain	50
Major Clinical Study	48
Mild Cognitive Impairment	47
Diagnostic Imaging	38
Deep Learning	35
Support Vector Machine	34
Feature Extraction	33
Cognitive Defect	27
Computer Assisted Diagnosis	26
Image Processing	26
Convolutional Neural Network	22
Disease Classification	22
Learning Algorithms	20

The search was further restricted to English publications only. There were 169 English publications. Table 2. shows the publications of different language.

Table 2 Trends in publishing language

Publication Type	Publication Type

English	169
Chinese	3
Total	172

Researchers have published 70.34% papers in journals, 18.60% papers in conference proceedings and 11.04% in book series as in table 3.

Table 3 Publication type

Publication Type	Number of Publication	Percentage from 169
Journal	118	70.34%
Conference Proceeding	32	18.60%
Book Series	19	11.04%

2.2 Highlights of preliminary data

In this paper the preliminary investigation is completed supported the keywords that extracted 169 publications from the Scopus database. Documents like journal papers, conference proceedings and book series were retrieved from 2018 to 2021 for the research area of Alzheimer's disease classification. The publication count per annum is as shown in table 4. Analysis supported number of publications per annum is shown in figure 1.

Table 4 Year wise publication count

Publication Type	Publication Type
2021	3
2020	53
2019	71
2018	42
Total	**169**

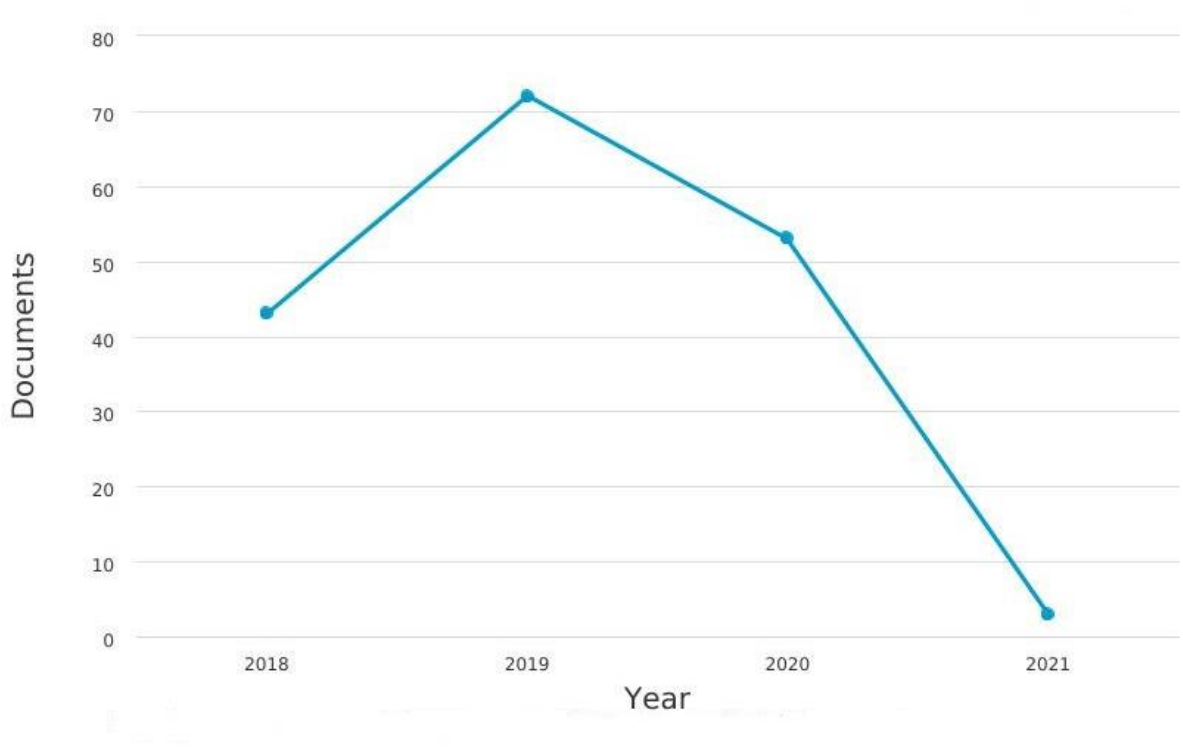

Figure 2 Yearly publishing trends (2018-21)

2.3 Data investigation

Through the conducted bibliometric review reveals about the typography of literatures available in the next section which will highlight the distinctness of the available literature and using the geographical dispersion and the contributions by different authors, where the papers were published and the affiliation statistics.

3. BIBLIOMETRIC ANALYSIS

To perform the bibliometric analysis two ways were used

- Geographical region analysis
- Statistics of affiliations, subject area, author statistics and document type.

3.1 Geographical region analysis

The geographical regions of attentiveness of the published papers are shown in figure 2 which is drawn using GPS Visualizer tool from gpsvisualizer.com. Indian and Chinese

publications are of the maximum number. Figure 3 shows the contribution in publications by different countries.

Figure 3 Geographical locations of the study

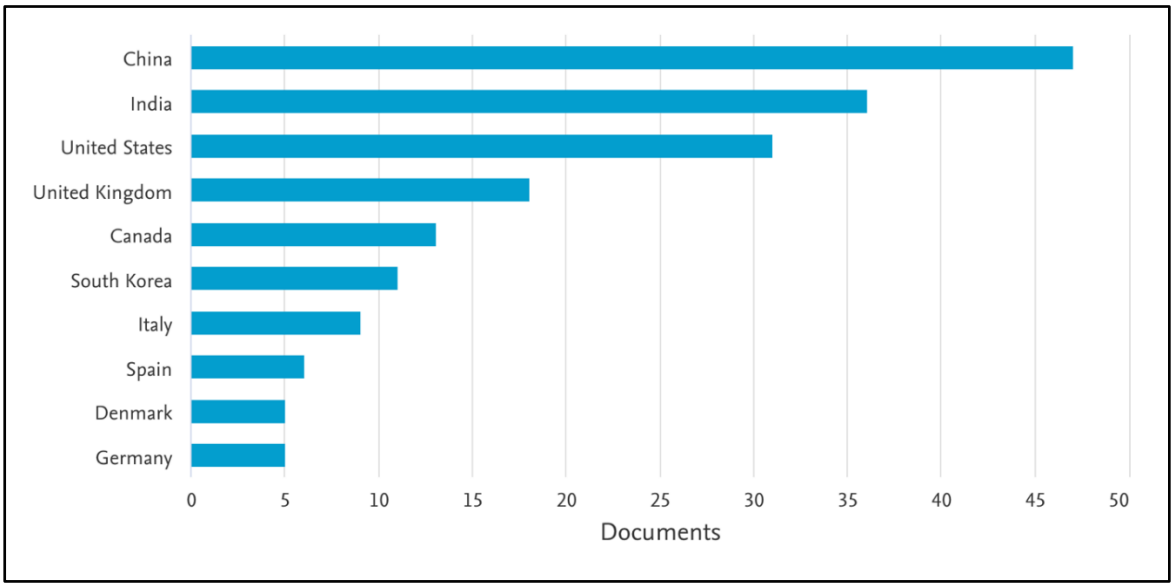

Figure 4: Analysis by country / Region

3.2 Analysis based on subject area

Categorization based on subject area is shown in figure 4. The analysis reveals that maximum number of research papers are published from the area of Computer science, Engineering followed by Medicine, Neuroscience and Mathematics.

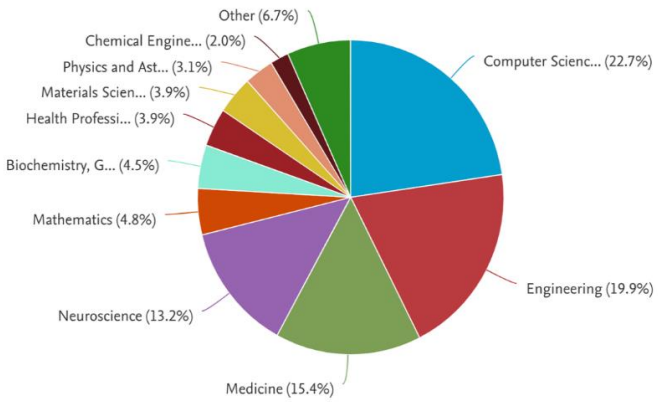

Figure 5 Analysis based on Subject area

3.3 Analysis based on affiliation

Contributions by different Universities worldwide in publishing research papers in the field of classification of Alzheimer's disease is shown in figure 5. The research area of classification of Alzheimer's disease dominated by the Indian and Chinese Universities listed in figure 5. Top ten Universities publishing in this field have been shown.

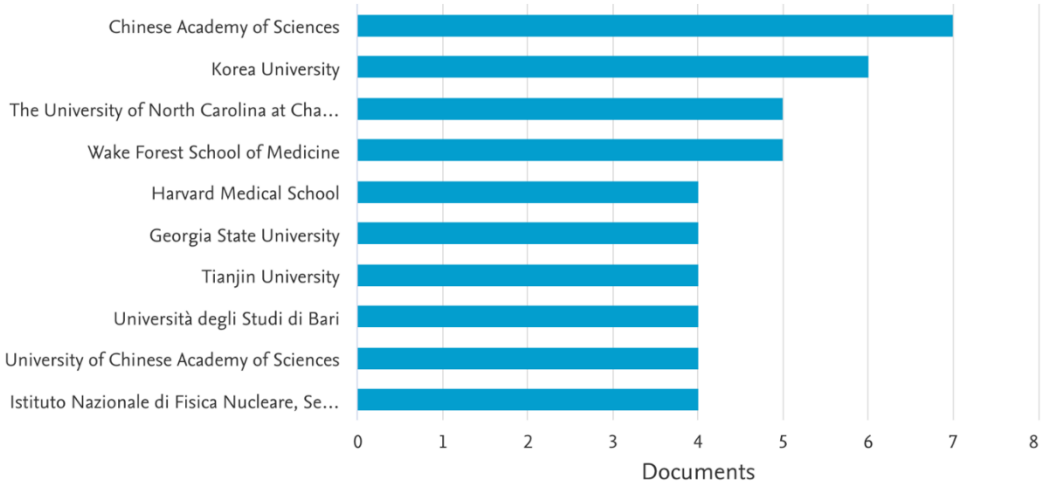

Figure 6 Analysis based on Affiliation

3.4 Analysis based on number of publications per author

Key authors contributing to the field of classification of Alzheimer's disease are depicted in figure 6**.** First ten authors were considered from the available accessed data from the Scopus database.

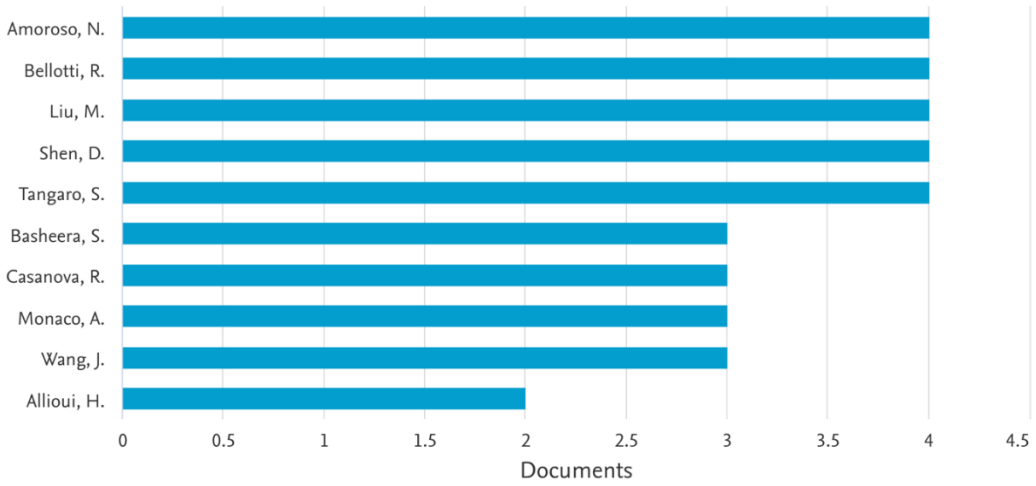

Figure 7 Analysis based on publication per author

3.5 Journal Statistics

The publication source types are shown in figure 7. It is clear from figure 7 that 29.8% publications are conference proceedings, 65.5% publications are from journal, 1.2% from book series and 3.5% are review publications.

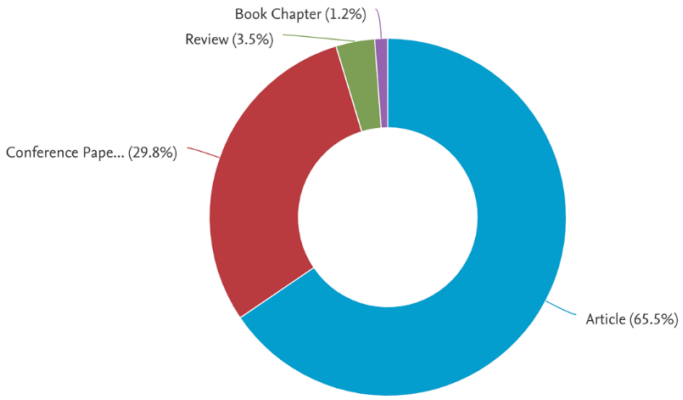

Figure 8 Analysis based on document type

3.6 Statistics by document source

Figure 8 shows the statistics for publications in Classification of Alzheimer's disease. From the available extracted data, it is apparent that maximum number of publications are from the Advances in Intelligent systems and computing journal.

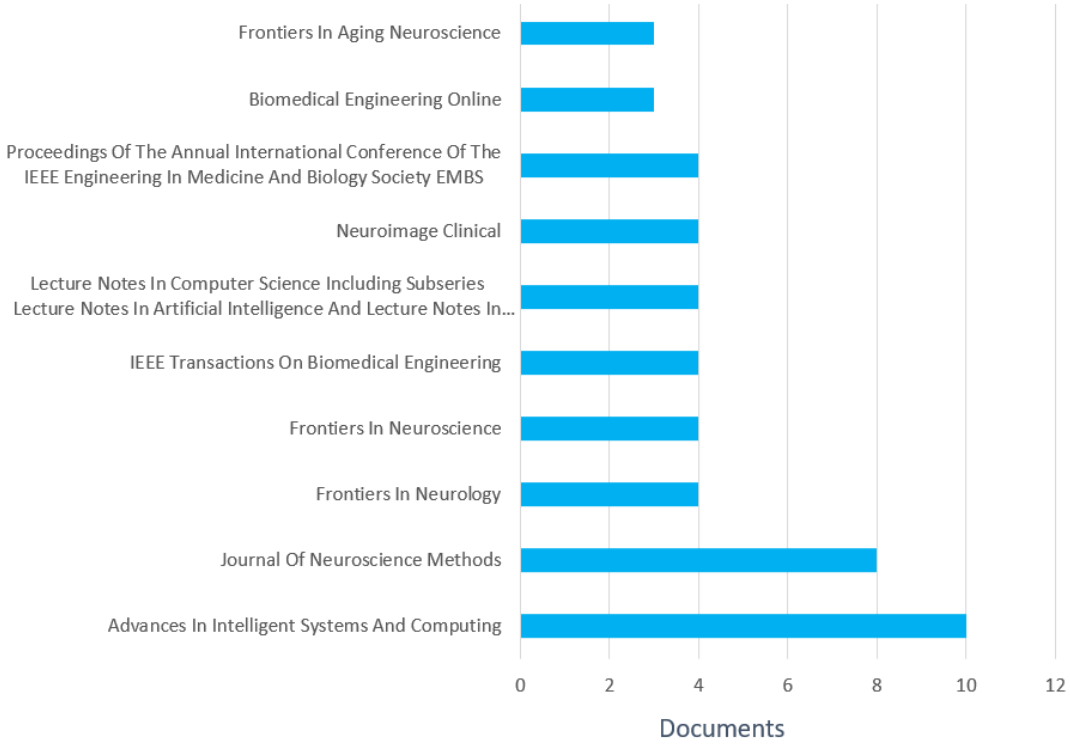

Figure 9 Analysis by document source

4. LIMITATIONS OF THIS STUDY

Different combinations of keywords were entered to explore the Scopus database for the aim of bibliometric review. A couple of important journals and articles which were not available in Scopus database could not be incorporated during this study.

5. CONCLUSIONS

Great advancement in computer intelligence and deep learning techniques, with the help of increase in computational power over the years, medical imaging sector has had a huge positive impact on classification and segmentation of neurodegenerative diseases. With new computer aided detection techniques for image analysis of MRI's and EEG's on the rise, early detection of neurodegenerative diseases such as Alzheimer's Disease has become more accurate and has helped doctors to get hold of the symptoms at an early stage . Although deep learning methods have a high impact on the quantitative chemical analysis of brain MRI, there's still difficulty find a strong, generic method. In this paper we reviewed various studies of brain structure and different classification techniques used for detection of Alzheimer's disease for both MRI and EEG, which when used together along with a deep learning technique can significantly improve diagnostic accuracy.

References

1. Nilesh Kulkarni, Vinayak Bairagi. "Introduction", Elsevier BV, 2018
2. www.j-alz.com
3. www.springerprofessional.de
4. Alzheimer A. Uber eine eigenartige Erkrankung der Hirnrinde. Zentralbl. Nervenh. Psych. 1907;18:177-9.
5. Yang HD, Kim DH, Lee SB, Young LD. History of Alzheimer's Disease. Dementia and Neurocognitive Disorders. 2016 Dec 1;15(4):115-21.
6. Soria Lopez JA, Gonzalez HM, and Leger GC. Chapter 13 – Alzheimer's disease. Handbook of Clinical Neurology 2019; 167: 231-255.
7. Glenner GG, Wong CW. Alzheimer's disease: initial report of the purification and characterization of a novel cerebrovascular amyloid protein. Biochemical and Biophysical Research Communications. 1984 May 16;120(3):885-90.
8. Brion JP, Couck AM, Passareiro E, Flament-Durand J. Neurofibrillary tangles of Alzheimer's disease: an immunohistochemical study. Journal of Submicroscopic Cytology. 1985 Jan 1;17(1):89-96.
9. McKhann G, Drachman D, Folstein M, Katzman R, Price D, Stadlan EM. Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDA Work Group* under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. Neurology. 1984 Jul 1;34(7):939-44.
10. McKhann GM, Knopman DS, Chertkow H, Hyman BT, Jack Jr CR, Kawas CH, et al. The diagnosis of dementia due to Alzheimer's disease: recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. Alzheimer's & Dementia. 2011 May;7(3):2639.
11. Jack Jr CR, Bennett DA, Blennow K, Carrillo MC, Dunn B, Haeberlein SB, et al. NIA-AA research framework: toward a biological definition of Alzheimer's disease. Alzheimer's & Dementia. 2018 Apr;14(4):535-62.
12. Barage SH, Sonawane KD. Amyloid cascade hypothesis: Pathogenesis and therapeutic strategies in Alzheimer's disease. Neuropeptides. 2015 Aug 1;52:1-8.
13. Shao W, Peng D, Wang X. Genetics of Alzheimer's disease: From pathogenesis to clinical usage. Journal of Clinical Neuroscience. 2017 Nov 1;45:1-8.