ECRONICON OPEN ACCESS

EC NEUROLOGY
Research Article

What to do with Senile Dementia now that Alzheimer's Disease is a Fiction: Is there any Plausible Genetic Explanation in the Light of and/or in Connection with Fischer's Disease?

Fred C C Peng[1]* and Virginia M Peng[2]

[1]*Department of Neurosurgery and Neurological Institute, Taipei Veterans General Hospital, Taiwan*

[2]*Ristumeikan University, Japan*

***Corresponding Author**: Fred C C Peng, Department of Neurosurgery and Neurological Institute, Taipei Veterans General Hospital, Taiwan.

Received: May 11, 2018; **Published:** July 20, 2018

Abstract

Several notions of AD exist among researchers today, resulting from each one of them inventing his/her own meaning of AD: (1) Dementia of any form is AD; (2) Dementia without any vascular disorder is AD (in contrast with vascular dementia); (3) Dementia caused by lesions in the hippocampus is AD; (4) Dementia with atrophy other than in frontal-temporal lobes is AD; (5) Dementia caused by amyloid beta to form plaques, one-to-one, is AD; (6) Dementia with plaques and tangles of whatever origin is AD (the so-called two hallmarks); (7) Tauopathy is added as another one-to-one cause-effect correlation as AD. (8) Is Vascular Dementia a solution to replace AD, then? This article is to probe the reasons why such has become the case.

When compared with historical facts in time and space, first of all, these notions are unfounded misconceptions. Auguste's autopsied materials reveal that she had four vascular disorders: Diabetes, Decubitus Angina, Arteriosclerosis, and Stupor. The autopsy also showed her evenly atrophied brain--cortical and subcortical-as well as internal and external hydrocephali. However, Oskar Fischer's voluminous publications in 1907-10 indicate that dementia can be caused by lesion in the brain of vascular and/or non-vascular origin. He pointed out that "There were no cases with tangles without plaques", suggesting that plaques and tangles might be two phases of one pathogenesis. For this idea, he posited a dichotomy: simple dementia and presbyophrenic dementia. Such being the case, what else in neurological disorders can cause or result in senile dementia? DLB, PSP, HD, SCA, EPILEPSY, MS, PD and PiD (Pick Disease), are just a few such cases. To these we must add FD (Fischer's Disease) in place of AD as well as Autism, although we do not have time to illustrate each one of these neurological disorders which eventually lead to dementia for the patients.

With so many pathological origins of senile dementia, however, there is one common cause: apoptosis in the nervous system. Our presentation will therefore raise three issues: (1) Experts on each of these neurological conditions carve their own sphere (or territory) of health care without regards to patients of other brain disorders. Is this territoriality of health care management appropriate or cost-effective? (2) We will illustrate a case of AD which in our view was misdiagnosed but can be better treated as a case of FD. (3) As public health management includes many domains, such as Nursing, Therapeutics (including language therapy in autism), and Social adjustment (including adaptation to the external environment), is there a plausible way to coordinate patients of each such form of senile dementia in a unified public health care management?

The conclusion lies in abandoning AD as a fiction by coming to grips with the following seriously: (1) FD and (2) dementia in the light of Fischer's dichotomy without inventing new terms, as there is no one-to-one cause-effect correlation with dementia because it is not a disease.

Keywords: *Senile Dementia; Alzheimer's Disease; Fischer's Disease*

Introduction

This article is based on what the first author has in the past few years published in books and articles which are concerned with two related questions: (1) Does AD really exist? [1]. (2) If not, what should we do then? Our suggestion will now be that it should be abandoned, as it has been a hindrance of progress for hundreds of neuroscientists when they need to be concerned seriously with the notion of dementia, as it is not a disease and has been in existence for as long as mankind, without inventing new terms for contrast with such a fiction called AD.

In this article, therefore, we shall attempt to explicate these questions for an answer in order to set the record straight, as many additional notions of AD have been invented in the literature, which only further aggravate the confusion. For this reason, we shall set up three related tasks with which to tackle the issues involved: (1) The evidence that AD is a fiction, and therefore it is not a disease. (2) The rush to invent new meanings of AD is a wild goose chase which must be stopped. (3) The importance of FD in place of AD is urgently needed in the light of Fischer's dichotomy without inventing new terms. But keep in mind that our point of departure is that dementia is not a disease; nor is it equivalent to AD, for it has been in existence for thousands of years.

In so doing, we shall conclude, given the fact of AD as a fiction, that (senile) dementia is not a unified brain disorder, least of all a disease for which Kraepelin invented the term Alzheimer's Disease in 1910 to start the wild goose chase for more than a century, because dementia ensues in differing manifestations of behavioral alterations, depending on where in the brain the cause/origin of dementia, i.e. apoptosis begins. Put differently, we shall raise differing neurological disorders in some details, where dementia is bound to manifest, even though dementia is not a disease. The reason is that depending on the initial location of apoptosis, the resulting behavioral alterations, which are collectively called (senile) dementia, most obvious in language disorder, will vary. But we must caution the reader to keep in mind that since to us dementia is not a disease, it has been defined by us as totality of the effects of wear and tear owing to aging which is the ongoing process of wear and tear.

In so doing, however, we must add that dementia is one overall manifestation of wear and tear most obvious in the alterations of brain functions of memory and cognition. Therefore, we think that a subtle distinction must be made between dementia and aging; that is, dementia is a significant result of aging, referring mostly to the weakening of mental activities while aging not only covers such altered behaviors but also includes disorders, such as prostate enlargement for men or colon cancer for men and women, including non-neurological disorders, such as breast cancer for women. and cataract for men and women as well as joint weakening because of ligamentous deterioration. However, given such a distinction, we shall in the following concentrate only on dementia, without getting too much involved in aging for our discussions.

Discussion

Alzheimer's Disease is now a Fiction

Although the first author has already published the idea that AD is a fiction, we purposely chose this statement from his recent article [2] to be in direct contrast with the theme of the Congress which is Dealing with the Challenges thrown in by Alzheimer's Disease and Dementia, International Conference on AD and Dementia. The statement, therefore, is intended to be a follow-up of his previous article [3] in order to emphasize that AD is indeed a fiction.

In that second article, he summarizes his previous publications regarding Alzheimer's academic status in time and space from 1901 in Frankfurt to his eventual position in Munich, during which time he never treated Auguste D who was admitted to the Asylum in Frankfurt. See [4] for more details.

Without knowing or checking the historical facts, two major deviations have come up in the literature: (1) Vascular dementia as a contrast with AD which is taken to be equivalent to or synonymous with (senile) dementia, because AD was popularly but mistakenly claimed to be caused by non-vascular disorders; and then (2) Frontotemporal dementia which stems from Pick's Disease, also as a contrast with AD. It was, for some reasons, thought that AD was confined to the frontal disorders only, without realizing that Auguste's brain at autopsy was evenly atrophied, cortically and subcortically including even the cerebellum.

To these two notions of dementia was then added another notion, known as MCI (Mild Cognitive Impairment) which takes two steps: that is, (a) the first step is the initial mild cognitive impairment which is then claimed to (b) progress to the second step which leads eventually to AD. But strangely enough the claimers of this notion had never checked the literature to realize that Oskar Fischer in 1906-10 already stated a dichotomy of simple dementia (without glandular necroses) and presbyophrenic dementia (with glandular necroses). Therefore, MCI has now turned out to be nothing but a poor reinvention of Fischer's dichotomy.

All these deviations stem in the main from Kraepelin's invention in 1910 when he defined in figure 143a, b, c "fibrillary patterns in Alzheimer's Disease as the most serious form of senile dementia", as if Alzheimer had prepared them for presentation in 1906 at Tübingen. These three microscopic preparations of fibrillary patterns from Kraepelin's textbook published in 1910 have therefore kept Kraepelin's definition of AD active, unfortunately, for more than a century even though Alzheimer did nothing of that sort; rather they were taken (or plagiarized) from Perusini's dozens of microscopic preparations.

However, while attempting to employ something of their own, these three deviations only result in two failures: (1) negligence to check the historical facts as to whether AD really exists or not; (2) eagerness on the part of each such claimer to carve territoriality as if (senile) dementia is a disease, when it is not, although Demenz in German is a Krankheit and dementia in English is NOT a disease; but an illness. See [5] for a detailed explanation why Demenz as Krankheit in German is equivalent to dementia in English but not at all a disease in English. Thus, the claim that Vascular dementia, Frontotemporal dementia, or MCI is a new idea to characterize a disease in English is false, even though each one of them attempts to keep AD alive for a distinction from its own.

The Wild Goose Chase

Why have such unfounded inventions come out in the literature to become so popular? Even in the list of topics for the 12th International Congress on Controversy in Neurology, held in Warsaw on April, 2018, a couple of topics of AD remain listed, although there is also a suggestion that the name of Alzheimer's Disease be deleted as it impedes the progress of research on dementia. The founder of CONy, Prof. Amos Korcyn, should be congratulated for including the deletion owing to the input from [3] and personal communications in emails with him. We believe that there are two major factors of perpetuation of AD even though it is a fiction:

(1) The annual meeting of the Alzheimer's Association is the first major cause of perpetuation; it has many branches each one of which encourages thousands of enthusiastic researcher to attend, as may be evidenced by the one held in Toronto, 2016, which attracted five thousand enthusiastic attendants to discuss Amyloid Beta and Tauopathy as a one-to-one cause of AD.

(2) In 2005, Peng published a booklet, entitled Alzheimer's Disease: What Is It After All?, in an attempt to set the record straight because Alzheimer did not discover a new disease in 1906. His purpose is to prove: (a) that dementia is not a disease but an illness, the confusion being in part linguistic, as English has four terms, Ailment, Disease, Illness, and Sickness while many others, German included, have only one term; (b) that dementia is caused by the progressive brain atrophy of one kind or another, resulting in a cluster of behavioral alterations collectively called dementia; and (c) that dementia can be observed and tested, as it manifests as an illness in varying forms of behavioral alterations, because it is the effect of brain atrophy due to wear and tear - apoptosis - that is the cause.

Unaware of these vital historical facts, new trends have been added to compete, such as the following claims: (1) Dementia of any form is AD; (2) Dementia without any vascular disorder is AD (in contrast with vascular dementia); (3) Dementia caused by lesions in the hippocampus is AD; (4) Dementia with atrophy other than in frontal-temporal lobes is AD; (5) Dementia caused by amyloid beta to form plaques, one-to-one, is AD; (6) Dementia with plaques and tangles of whatever origin is AD (the so-called two hallmarks); (7) Tauopathy is added as another one-to-one cause-effect correlation as AD.

Our question now is this: What is the fundamental cause or motivation for these neuroscientists to invent new terms as AD? We think the main motivation is twofold: (1) that Alzheimer did not discover a new disease in 1907 and yet the false claim of his discovery has come down in the literature to spread for more than a century as a result of setting up the Alzheimer's Association as described above; but (2) that those who have believed the false claim confused AD as claimed with the real neurological disorder called senile dementia which has been in existence for many, many centuries, as will be shown below, a confusion owing to the total unawareness or denial of its long history in existence.

Innovation is good, if and only if it reflects reality based on historical facts. But none of the new inventions comes close to such facts. Let us therefore attend to the notion of (senile) dementia as to whether it was a "new idea" starting from 1901 when Auguste D. was admitted to the Asylum in Frankfurt or it had been known long before that. For this, we shall cite a famous historical fact, going back to 246 BC-210 BC) during the reign of China's first Emperor known by his title of Chin Shih Huang (秦始皇) who has been noted for connecting the Great Walls of China, among others, by defeating the six warring states, one-by-one, in China, each one of which had built its own wall to guard against invasion.

The point we wish to raise here is that after conquering each one of the warring states the Emperor decided to find a way to live an eternal life as he began to realize that every human aged and then died. So, he sent a man known in the history as 徐福 to look for "the pills for eternal life" (不老不死丹). The agent did leave China in search of the pills but he never came back to China. What we want to point out is that the notion of aging which leads to senile dementia and death was already known to the Emperor among others in 246 BC and not at all a new discovery in 1907, least of all discovered by Alzheimer as claimed by Kraepelin in 1910,

Vascular Dementia as a solution: Unaware of those "inventions", thinking that AD was caused by a non-vascular disorder of the nervous system, this idea of vascular dementia was then proposed by a group of neurologists and neuropsychiatrists in Europe, notably Sweden, and Canada; they now form an organization, called Vascog (The International Society of Vascular Dementia) which is intended to replace AD - or even the Alzheimer's Association.

Peng attended a couple of such meetings initially but decided to drop out because his view of eliminating AD as a fiction has become an irritation and it contradicts their fundamental principle of vascular disorders as the base of dementia by hanging on to AD in contrast with their notion of vascular dementia. Put differently, if they allow Peng's views to compete in their organization, conflicting views are bound to emerge. Therefore, Peng has decided not to attend Vascog again, as he was prohibited to present a paper at the meeting in Toronto.

Fronto-Temporal Dementia: Likewise, this idea was inspired by a group of German neuroscientists who thought that Pick's disease has a different lesion site and that there should/could be another lesion site which they believe also causes dementia as a disease, starting with the fronto-temporal regions instead.

At the same time, the proponents of this idea also wanted to contrast fronto-temporal dementia with AD because they also believe that AD is caused by non-vascular lesion in the brain, as recommended by DSM IV. Thus, they too have organized an association to hold annual meetings to compete with Vascog without realizing that Fischer's disease has both vascular and/or non-vascular disorders in origin and that Auguste's autopsied brain revealed an evenly atrophied brain, who also had four vascular disorders, as well as Internal and External hydrocephali.

Mild Cognitive Impairment: While the two previous organizations hold on to AD as a contrast to their view, the proponents of Mild Cognitive Impairment (MCI) directly involves AD as the terminal part of its pathological development. As it has turned out, owing to the unawareness of Fischer's dichotomy of simple dementia and presbyophrenic dementia, they have used Ronald Reagan's admission before his death that he had been inflicted by AD to prove their point; that is, they claim that Reagan, while still in office, had mild cognitive impairment which, after his departure from the White House, got worse, leading to his admission of suffering from AD.

Our view of Reagan's illness is that he was a perfect example of Fischer's dichotomy of Simple Dementia (without glandular necrosis), while still in office, and Presbyophrenic Dementia (with glandular necrosis), before his death after he had left the White House. Our view raises a pertinent puzzle here.

We want to ask one important question: That is, since Kraepelin did NOT dare include plaques in his definition of AD, we want to know if fibrillary patterns are the same things as Fischer's glandular necroses which characterize his Presbyophrenic Dementia? We think the answer to this question, one way or the other, holds the key to our views as illustrated further above. (1) If the same, then there is NO Alzheimer's Disease, because glandular necroses had been described by Fischer before 1910. (2) If not the same, then why should there have been two hallmarks, Plaques and Tangles, to claim that Alzheimer had discovered them as two unique findings which could not be classified in line with Kraepelin's dementia praecox, and therefore Alzheimer had discovered a new disease?

There was an interesting real example of this Simple Dementia of Fischer's Dichotomy. That is, Prince Charles of the Great Britain visited the USA and President Reagan hosted a formal state dinner at the White House to honor him. He then introduced Prince Charles as Prince David, a scene of the formal dinner party at the White House that was televised throughout the world.

After Reagan's departure from the White House, his conditions of dementia got worse, as reported by his former underlings who came to visit him in California, until finally either Reagan himself or Nancy admitted that he had been diagnosed to have AD. We think that Reagan had Fischer's Disease instead, and not at all AD. To prove our point, we can illustrate below an even better example of the false claim of AD to prove that what many people think patients suffer and die of AD is not AD at all but rather Fischer's Disease.

Illustrations of Other Neurological Disorders

In "Dying with Alzheimer's Disease" [6], the author, Caren Leid, describes in details her mother's illness which was diagnosed to be AD of which her mother died gradually in four major steps according to her fairly detailed description:

First Step: "Forgetful" early stage

- Insidious/gradual
- Recent memory loss
- Time/space disorientation
- Mood swings
- Slower/withdrawal/denial
- Impaired judgment
- Subtle language dysfunction
- Continues to worsen

Second Step: "Confusion" early middle stage

- Obvious memory deficits
- Need for supervision in specialized activities
- Language/communication problems
- Anxiety/restlessness
- Problem behaviour becomes more severe
- Usually most difficult period for client

Third Step: "Severe Dementia" stage

- Obviously disabled cognitively
- Full-time supervision needed
- Marked personality/behaviour problems
- Disorientation to person
- Communication very difficult
- Psychosis
- Physical disorders appear
- Can still reminisce

Final Step: "Terminal" late stage

- Almost total loss of intelligence/physical functioning
- Few words spoken/understood
- Emaciation/susceptible to infection
- Death

Resemblances of the Stages of Misdiagnosed AD and Fischer's Disease

The Forgetful Early Stage corresponds to Fischer's Simple Dementia and the "Terminal" Late Stage corresponds to Fischer's Presbyophrenic Dementia. The two stages in between, "Confusion" Early Middle Stage and "Severe" Dementia Stage characterize vividly the Progressive deterioration or Proliferation in Fischer's dichotomy from Simple Dementia to Presbyophrenic Dementia.

Conclusion

In view of the discussions above, it should be clear now that there is no Alzheimer's Disease as invented by Kraepelin. For one thing, he defined it as Fibrillary Patterns without referring even to Plaques, because Plaques had already been described by people before Alzheimer's 1907 barely two page publication. The cutting edge of this conclusive point is Alzheimer's own dishonesty: Kraepelin's invention of AD in 1910 gave Alzheimer a wrong sense of confidence by claiming in 1911 three unbelievable publications as evidence in support of his acceptance of Kraepelin's proclamation of this Alzheimer's Disease: That is, he cites [7,8], both of which describe the same patient, by Bonfiglio and Perusini, but in greater detail by the latter, as well as [9] which is Perusini's detailed description of Auguste, as "three new cases" in support of his discovery of a new disease. in 1906.

Most practitioners in the medical field are not aware of such fabrications; some even claim Alzheimer's Disease re-discovered [10].

Is there then a plausible genetic explanation of dementia which is not a disease but has been in existence for as long as mankind, even though the neuroscientists prior to 1901 had thought otherwise? We think there should be such a plausible genetic explanation. We will now plan to team up with a couple of neuroscientists at the Taipei Veterans General Hospital, The Neurological Institute, to look into this intriguing probe for a plausible explanation in the future, because the onset of dementia varies from one race to another or even from one person to another in the same race.

Put differently, we shall raise differing neurological disorders in some details, where dementia is bound to manifest, in order to determine the location of apoptosis where dementia starts, even though dementia is not a disease, that is, what is the cause of each anatomo-neurophysiological apoptosis that ensues in (senile) dementia. The reason is that depending on the initial location of apoptosis, the resulting behavioral alterations, which are collectively called (senile) dementia, most obvious in language disorder, will vary.

Bibliography

1. Peng FCC. "Does Alzheimer's Disease Really Exist?" Taipei: Ho-Chi Book Publishing Company (2003).
2. Peng FCC. "Dementia in Parkinson's Disease Revisited: In the Light of Fischer's Disease". *EC Neurology* 6.2 (2017): 39-53.
3. Peng FCC. "Is Alzheimer's Disease A Fiction?" *Clinical Research and Trials* 2 (2016): 108-111.
4. Peng FCC. "Alzheimer' Disease: What Is It After All?" Taipei: Ho-Chi Book Publishing Company (2005).
5. Peng FCC. "Alzheimer's Disease Is A Fiction?" *Clinical Research and Trials* 2 (2017): 48-52.
6. Caren Leid. "Dying with Alzheimer's Disease". *EC Neurology* 4.1 (2016): 27-29.
7. Bonfiglio F. "Di speciali reperti in un caso di probabile sifilide cerebrale". *Rivistra Sperimentale di Freniatri* 344 (1908): 196-206.
8. Perusini G. "Sul valore nosografico di alcuni reperti istopatologici caratteristici per la Senilita". *Rivista Italiana di Neuropatologia, Psichiatria ed Eletroterpia* 4 (1911): 145-213.
9. Gaetano Perusini's detailed description of Auguste D.
10. Bick KL and Amaducci L. "Alois Alzheimer and Gaetano Perusini: Alzheimer's First Case Rediscovered". Padova Laviana Press (1989).

Volume 10 Issue 8 August 2018