



Alchajmerova bolest: nekad i sad, perspektive u budućnosti Alzheimer's Disease, then and now, Perspectives in the Future

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Apstrakt

Alchajmerova bolest, kao načešći uzrok demencije širom sveta, multifaktorijalne je etiologije. Statistički podaci pokazuju eksponencijalni porast broja slučajeva Alchajmerove bolesti, naglašavajući potrebu za razvojem efikasnog lečenja.

Iako je demencija opisana u drevnim tekstovima tokom veka, naše znanje o njenim osnovnim uzrocima je staro više od veka. Alchajmer je objavio svoju sada poznatu studiju slučaja pre više od 110 godina, a naše savremeno razumevanje bolesti, koja nosi njegovo ime, i njenih psihopatoloških posledica, zaista je počelo da se ubrzava tek 1980. godine. Nakon tog perioda svedoci smo velikog broja istraživanja, kako osnovnih, tako i translacionih, o uzrocima, karakteristikama i mogućnostima tretmana za Alchajmerovu bolest i druge demencije.

Dok su ranije procene stadijuma bolesti bile fokusirane na progresivno pogoršanje kliničkog funkcionalisanja, osamdesetih godina prošlog veka započela su temeljna profilisanja neuropsihološkog deficit-a. Devedesetih godina, u nastavku napora i eksplozije istraživanja, počinje identifikacija specifičnih kognitivnih mehanizama na koje utiče različit neuropatološki supstrat. Dve hiljadite godine bacaju fokus na proučavanje prodromalnih stadijuma neurodegenerativne bolesti pre pojave potpunog sindroma demencije (npr. blagog kognitivnog poremećaja).

Snimanje mozga pozitron emisionom tomografijom i studije biomarkera cerebrospinalne tečnosti istakli su drugu fazu, predkliničku fazu Alchajmerove bolesti, te i zaključak da kaskada detektibilnih bioloških abnormalnosti itekako prethodi kognitivnom deficitu i njegovom padu. U protekloj deceniji razvoj imidžinga, detektovanja tečnih biomarkera patofiziologije Alchajmerove bolesti daju nam mogućnost dijagnostikovanja i nekoliko faza u predkliničkoj fazi Alchajmerove bolesti.

Dosadašnji lekovi koji su u upotrebi obezbeđuju samo simptomatsko poboljšanje, ali nemaju uticaja na modifikaciju patofiziološkog supstrata bolesti. Glavna strategija istraživanja terapije za Alchajmerovu bolest zasnovana je na amiloidu u tauu, koji bi mogli da budu ključ za lečenje Alchajmerove bolesti u bliskoj budućnosti.

Abstract

Alzheimer's disease, as the most common cause of dementia worldwide, has a multifactorial etiology. Statistical data show an exponential increase in the number of Alzheimer's disease cases, emphasizing the need for the development of effective treatment.

Although dementia has been described in ancient texts for centuries, our knowledge of its underlying causes is more than a century old. Alzheimer published his now-famous case study more than 110 years ago, and it wasn't until 1980 that our modern understanding of the disease that bears his name and its psychopathological consequences really began to accelerate. After that period, we witnessed a large amount of research, both basic and translational, on the causes, characteristics and possibilities of treatment for Alzheimer's disease and other types of dementia.

While earlier assessments of disease stage focused on progressive deterioration of clinical functioning, in the 1980s, thorough profiling of neuropsychological deficits began. In the 1990s, following the efforts and explosion of research, the identification of specific cognitive mechanisms that are influenced by different neuropathological substrates began. The two thousand years have focused on the study of prodromal stages of neurodegenerative disease before the onset of a full dementia syndrome (e.g. mild cognitive impairment).

Brain imaging with positron emission tomography and studies of cerebrospinal fluid biomarkers highlighted the second phase, the preclinical phase of Alzheimer's disease, and the conclusion that the cascade of detectable biological abnormalities precedes the cognitive deficit and its decline. In the past decade, the development of imaging and detection of liquid biomarkers of the pathophysiology of Alzheimer's disease give us the possibility of diagnosing several stages in the preclinical phase of Alzheimer's disease.

The drugs currently in use provide only symptomatic improvement, but have no effect on the modification of the pathophysiological substrate of the disease. A major research strategy for Alzheimer's disease therapy is based on amyloid in tau, which could be the key to Alzheimer's disease treatment in the near future.

