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Iatrogenic Alzheimer's disease In Recipients Of Cadaveric Pituitary-Derived Growth Hormone

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Abstract

Iatrogenic Alzheimer's disease (AD) refers to cases of Alzheimer's disease caused inadvertently by medical treatment or diagnostic procedures. One significant source of iatrogenic AD has been linked to the administration of cadaveric pituitary-derived growth hormone (c-hGH) used in treating growth hormone deficiencies before synthetic alternatives became available. This case study explores the development of AD in individuals treated with c-hGH, highlighting the pathogenesis, clinical features, and preventive measures that have evolved in response to this medical history.

Keywords: Iatrogenic Alzheimer's disease:; Cadaveric pituitary-derived growth hormone (c-hGH);Growth hormone deficiency; Prion diseases

Introduction

Before the introduction of recombinant human growth hormone (rhGH) in the mid-1980s, growth hormone therapy relied on extraction from the pituitary glands of cadavers. While this method was effective in treating conditions like growth hormone deficiency, it also introduced significant risks, including the transmission of prion diseases. Prion diseases, caused by misfolded proteins called prions, include Creutzfeldt-Jakob disease (CJD) and have been implicated in other neurodegenerative conditions such as Alzheimer's disease [1]. Iatrogenic Alzheimer's disease (AD) is a rare and concerning consequence of medical treatments or procedures that inadvertently lead to the development of Alzheimer's-like symptoms in patients. One historical source of iatrogenic AD is the administration of cadaveric pituitary-derived growth hormone (c-hGH) used to treat growth hormone deficiencies [2]. This practice, prevalent before the advent of recombinant human growth hormone (rhGH) in the mid-1980s, posed significant risks due to potential contamination with prions or misfolded proteins that can induce neurodegenerative diseases. During the period when c-hGH was widely used, thousands of individuals, primarily children, were treated for conditions such as growth hormone deficiency. In the late 1980s, cases of Creutzfeldt-Jakob disease (CJD) were reported among recipients of c-hGH, raising concerns about the safety of these treatments. Subsequent investigations suggested that the contaminated growth hormone might also increase the risk of developing Alzheimer's disease [3]. This case study explores the mechanisms, clinical manifestations, and preventive measures related to iatrogenic AD in individuals who received c-hGH, providing valuable insights into the implications of medical treatments derived from biological sources.

Background

The use of c-hGH began in the 1960s and continued until the mid-1980s. During this period, thousands of children and adults received treatments derived from human cadaveric pituitaries. In the late 1980s, reports began to surface linking c-hGH treatments to cases of CJD, a rare but fatal prion disease. Subsequent studies have suggested a potential link between c-hGH treatment and an increased risk of developing AD [4].

Pathogenesis

Alzheimer's disease is characterized by the accumulation of amyloid-beta plaques and tau protein tangles in the brain, leading to progressive neurodegeneration. Prions, the infectious agents in CJD, are similarly characterized by the abnormal folding of proteins [5]. The hypothesis connecting c-hGH to AD posits that the extracted hormones could be contaminated with amyloid-beta or tau proteins, or with prions capable of inducing misfolding in endogenous proteins, thus initiating or accelerating neurodegenerative processes akin to those in AD.

Clinical features

Patients who developed AD after c-hGH treatment present with the classical symptoms of Alzheimer's, including memory loss, confusion, difficulty in completing familiar tasks, and changes in mood and behavior. However, the onset may be earlier than sporadic AD, reflecting the iatrogenic nature of the disease. Diagnosis involves a combination of clinical assessment, imaging studies, and sometimes post-mortem neuropathological examination revealing amyloid plaques and tau tangles [6].

Case study analysis

Patient profile

Age: 45

Sex: Female

Treatment history: Received c-hGH treatment between ages 10-15 for growth hormone deficiency.

Symptoms: Began experiencing memory loss and cognitive decline at age 43.

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Clinical course: Initial assessment revealed mild cognitive impairment, which progressed over two years to a diagnosis of early-onset Alzheimer's disease.

MRI and PET scans showed patterns consistent with AD, including hippocampal atrophy and amyloid-beta accumulation.

Diagnosis: A thorough medical history and exclusion of other causes of dementia supported the diagnosis of iatrogenic AD.

Genetic testing ruled out familial AD, strengthening the link to prior c-hGH treatment.

Discussion

The patient's early-onset AD, following a history of c-hGH treatment, highlights the potential long-term risks associated with cadaver-derived treatments. The case underscores the need for vigilant long-term monitoring of individuals exposed to such treatments and advances our understanding of the mechanisms through which iatrogenic factors can contribute to neurodegenerative diseases.

Preventive measures and current practices: The discovery of prion disease transmission via c-hGH led to the discontinuation of cadaver-derived hormone treatments and the development of recombinant DNA technology to produce synthetic human growth hormone (rhGH), which is free from the risk of prion contamination. Current practices emphasize strict screening and processing protocols for all biological products, rigorous post-market surveillance, and robust patient education regarding the potential long-term risks of their treatments [7-10].

Conclusion

Iatrogenic AD in recipients of c-hGH highlights a tragic chapter in medical history, illustrating the unintended consequences of early biomedical treatments. It serves as a reminder of the importance of ongoing vigilance, rigorous safety standards, and the need for continued research into the long-term effects of medical therapies. Moving forward, the lessons learned from c-hGH-associated neurodegenerative diseases will inform safer therapeutic practices and enhance our ability to protect patients from similar risks.

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Conflict of Interest

None

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