

Postoperative Cognitive Impairment is caused by Anesthesia and Neuroinflammation

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Introduction

The condition of cognitive impairment following surgery, also known as postoperative delirium or postoperative cognitive dysfunction, is a frequent issue that mostly impacts the elderly. Neuroinflammation is present in both occurrences, but the precise molecular pathways leading cognitive impairment after anaesthesia are still understood. Epigenetics is a potential mechanism that affects cognitive function since anaesthetic medications have the potential to have a long-term impact on protein transcription. Long-term consequences may be caused by epigenetic pathways, which may also inspire new therapeutic modalities.

It is evident that anaesthetics change the expression of DNA and histone modifying enzymes [1-3], which then affects epigenetic markers like methylation, histone acetylation, and histone methylation on inflammatory genes (such as TNF-alpha, IL-6, or IL1 beta) and genes involved in neural development (such as brain-derived neurotrophic factor). Neuroinflammation rises and neuronal development falls after anaesthesia. Each of these alterations has the potential to deteriorate cognitive ability. Particularly, inhibiting histone deacetylase reduces cognitive damage after surgery and could be an unique therapeutic approach. More study including human participants is necessary, though, as the majority of the findings originate from animal models.

Description

The earliest reports of long-term cognitive impairment in elderly people after surgery were published more than 60 years ago. Little is understood about efficiently preventing perioperative stress in the ageing brain despite decades of rigorous research. Two important illnesses are discussed in relation to postoperative cognitive impairment: postoperative delirium (POD) and postoperative cognitive dysfunction (POCD). Delirium, commonly referred to as acute confusional syndrome, is characterised by aberrant perception, diminished attention, and impaired direction. Acute fluctuating cognitive impairment that clears up within days of surgery is what is known as this syndrome.

Elderly people admitted to general hospitals frequently have the multifactorial aetiology. In this context, it is important to recognise that dementia and mild cognitive impairment are two distinct conditions, despite the fact that both are characterised by objective signs of cognitive impairment. The fundamental distinction is that dementia always affects many cognitive domains and significantly interferes with daily functioning. Dementia differs from the events discussed here in that it is an irreversible state. The overlap and divergence of POD and POCD are being discussed because their link is

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not fully known. For a number of causes, delirium can result in POCD or long-term cognitive damage. One is that, even with excellent nursing care, delirium patients typically cannot maintain hydration and nutrition; in addition, sleep deprivation may develop and result in neuronal damage [4,5].

Additionally, drug toxicity can happen; for instance, benzodiazepine use has been associated with cognitive impairment. The Confusion Assessment Method for Intensive Care Unit or the Nursing Delirium Screening Scale are used to diagnose POD, which is described by the Diagnostic and Statistical Manual of Mental Disorders as an acute brain failure that happens right after surgery in 15 to 33% of older patients. Longer hospital stays, more time spent ventilating patients, more work for nurses, and higher mortality are all associated with it. The recovery of postoperative delirium varies greatly among individuals and might take the form of the more prevalent hypoactive subtype, a hyperactive or mixed form, or both. It is frequently accompanied by apathy, confusion, agitation, or delusions as well as symptoms of sympathetic arousal.

Conclusion

Long-term cognitive impairment is thought to be caused by chronic neurodegeneration and is brought on by neurotoxicity, whereas delirium is thought to be caused by an abrupt change in neurotransmitter levels. Although the pathogenesis of neither disease is completely known, there appear to be similarities because neuroinflammation is a critical factor in both. However, it is necessary to note additional risk factors for cognitive impairment. They consist of general and cognitive health before to surgery, medical comorbidities such hypotension and hypoxia, admission to the hospital for any cause, exposure to general anaesthesia, and use of psychoactive drugs.

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Conflict of Interest

There are no conflicts of interest by author.

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