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Anesthesia and Neuroinflammation Cause Postoperative Cognitive Impairment

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Introduction

Cognitive impairment after surgery is a common problem that primarily affects the elderly and is classified as postoperative delirium or postoperative cognitive dysfunction. Both phenomena are accompanied by neuroinflammation; however, the precise molecular mechanisms underlying cognitive impairment following anaesthesia are still unknown. Because anaesthetic drugs can have a long-term effect on protein transcription, epigenetics is a possible mechanism that influences cognitive function. Epigenetic mechanisms could be responsible for long-term effects and could lead to novel therapeutic approaches. It is clear that anaesthetics alter the expression of DNA and histone modifying enzymes [1-3], which in turn affect epigenetic markers such as methylation, histone acetylation and histone methylation on inflammatory genes (e.g., TNF-alpha, IL-6, or IL1 beta) and neuronal development genes (such as brain-derived neurotrophic factor). Following anaesthesia, neuroinflammation increases and neuronal growth decreases. All of these changes have the potential to impair cognitive function. Inhibiting histone deacetylase, in particular, alleviates cognitive impairment following surgery and may be a novel therapeutic option for treatment. However, because the majority of the findings come from animal models, more research with human subjects is required.

Description

More than six decades ago, the first reports of long-term cognitive impairment in elderly patients following surgery were published. Despite decades of intensive research, little is known about effectively preventing perioperative stress in the ageing brain. In the context of postoperative cognitive impairment, two major disorders are described: postoperative delirium (POD) and postoperative cognitive dysfunction (POCD). Delirium, also known as acute confusional syndrome, is defined by impaired orientation, decreased attention and abnormal perception. It is defined as a syndrome characterised by acute fluctuating cognitive impairment that resolves within days of surgery.

The pathogenesis is multifactorial and it is common in elderly patients admitted to general hospitals. In this context, we must also distinguish between mild cognitive impairment and dementia, which are not the same thing despite the fact that both are characterised by objective evidence of cognitive impairment. The main difference is that dementia always involves more than one cognitive domain and causes significant interference with daily life. Furthermore, dementia is an irreversible state, which distinguishes it from the phenomena described here. The relationship between POD and POCD is not fully understood and both overlap and divergence are being debated. Delirium can cause POCD or long-term cognitive impairment for a variety of reasons. One is that delirium patients are frequently incapable of maintaining hydration and nourishment, even with good nursing care; additionally, sleep deprivation may occur and cause neuronal damage [4,5].

Furthermore, drug toxicity may occur; for example, the use of benzodiazepines has been linked to cognitive impairment. POD is defined by the Diagnostic and Statistical Manual of Mental Disorders as an acute brain failure that occurs directly after surgery in 15-53% of older patients and is assessed by the Confusion Assessment Method for Intensive Care Unit or the Nursing Delirium Screening Scale. It is linked to a longer hospital stay, prolonged ventilation time, increased nursing effort and increased mortality. From hours to months, postoperative delirium recovers with high intraindividual differences and can manifest as either a more common hypoactive subtype or a hyperactive or mixed form, accompanied by apathy, confusion, agitation, or delusions, as well as signs of sympathetic arousal.

Conclusion

The pathophysiology of the syndromes is still unknown, but it is assumed that delirium is caused by an acute change in neurotransmitter levels, whereas long-term cognitive impairment is caused by chronic neurodegeneration and is triggered by neurotoxicity. Despite the fact that neither disease's pathophysiology is fully understood, there appear to be similarities, as neuroinflammation plays a key role in both. Other risk factors for cognitive impairment, however, must be mentioned. They include pre-operative general and cognitive health, medical comorbidities such as hypotension and hypoxia, hospitalisation for any reason, general anaesthesia exposure and psychoactive drug exposure.

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Conflict of Interest

There are no conflicts of interest by author.

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