HSR (LONDON),
HOUGHTON STREET REVIEW JOURNAL
ISSN: 2058-5519|Impact factor-15.9
http://houghtonstreetreview.com/index.php/hsr

COGNITIVE IMPAIRMENT IN POSTOPERATIVE CONDITIONS

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Doi: https://doi.org/10.5281/zenodo.21845916

Abstract: Postoperative cognitive impairment (PCI), including postoperative cognitive dysfunction (POCD) and postoperative delirium (POD), remains one of the most significant neurological complications following surgical procedures, particularly among elderly and high-risk patients. Cognitive decline after surgery adversely affects memory, attention, executive function, learning ability, quality of life, and long-term functional independence. The increasing number of surgical interventions worldwide and the aging population have intensified the need for effective strategies to prevent, diagnose, and manage postoperative cognitive disorders. This review examines current evidence regarding the epidemiology, pathophysiological mechanisms, risk factors, diagnostic approaches, preventive measures, and therapeutic interventions associated with postoperative cognitive impairment. Particular attention is given to neuroinflammation, oxidative stress, blood-brain barrier dysfunction, cerebral hypoperfusion, anesthetic exposure, perioperative pain, and patient-related factors. Recent advances in biomarker research, neuroimaging, digital cognitive assessment, and artificial intelligence-based prediction models are also discussed. The findings indicate that postoperative cognitive impairment is a multifactorial condition requiring multidisciplinary perioperative management. Early identification of vulnerable patients, optimization of anesthesia and surgical techniques, implementation of enhanced recovery protocols, and postoperative cognitive rehabilitation may substantially reduce the incidence and severity of cognitive decline. Future investigations should focus on precision medicine approaches, molecular biomarkers, and personalized perioperative care to improve neurological outcomes after surgery.

Key words: postoperative cognitive impairment, postoperative cognitive dysfunction, postoperative delirium, anesthesia, neuroinflammation, cognitive decline, surgery, elderly patients, biomarkers, perioperative medicine, neuroprotection, rehabilitation.

Introduction. Postoperative cognitive impairment (PCI) has become one of the most important neurological complications in contemporary perioperative medicine. Despite remarkable advances in surgical techniques, anesthetic management, intensive care, and perioperative monitoring, cognitive disturbances continue to affect a substantial proportion of patients undergoing surgical procedures. These disorders are associated not only with delayed recovery and prolonged hospitalization but also with increased healthcare expenditures, reduced functional independence, diminished quality of life, and elevated long-term morbidity and mortality. As global populations continue to age and the number of complex surgical interventions increases annually, postoperative neurocognitive disorders have emerged as a major public health concern requiring multidisciplinary clinical and scientific attention. According to recent epidemiological studies, more than 300 million surgical procedures are performed worldwide each year, and this number continues to rise because of demographic aging, improvements in healthcare accessibility, and technological developments in surgery. Older adults constitute the fastest-growing surgical population and simultaneously represent the group at highest risk for postoperative cognitive complications. Aging is accompanied by progressive structural and functional

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alterations within the central nervous system, including neuronal loss, synaptic dysfunction, reduced cerebral reserve, mitochondrial impairment, chronic low-grade inflammation, and vascular changes, all of which increase susceptibility to perioperative neurological injury. Postoperative cognitive impairment encompasses a spectrum of cognitive disorders that develop after surgery and anesthesia. The two principal clinical entities are postoperative delirium (POD) and postoperative cognitive dysfunction (POCD). Postoperative delirium is characterized by an acute, fluctuating disturbance of consciousness, attention, awareness, and cognition that usually occurs during the first hours or days after surgery. In contrast, postoperative cognitive dysfunction is characterized by more persistent impairments in memory, executive function, attention, information processing speed, learning capacity, and psychomotor performance that may last for weeks, months, or even years. Although recent international recommendations increasingly use the broader term *perioperative neurocognitive disorders (PND)*, both POD and POCD remain widely recognized in clinical practice and scientific literature. The reported incidence of postoperative cognitive impairment varies considerably depending on patient characteristics, type of surgery, anesthetic technique, diagnostic criteria, and duration of follow-up. Among elderly patients undergoing major cardiac surgery, postoperative cognitive dysfunction has been reported in a substantial proportion of patients during the early postoperative period, while a smaller but clinically important percentage continue to experience measurable cognitive deficits several months later. Similar patterns have been observed after orthopedic, vascular, thoracic, abdominal, and neurosurgical procedures. Even mild cognitive decline may significantly interfere with medication adherence, rehabilitation participation, return to work, social functioning, and independent daily living. The pathophysiology of postoperative cognitive impairment is highly complex and involves multiple interacting biological mechanisms rather than a single pathological process. Current evidence suggests that systemic inflammatory activation triggered by surgical trauma plays a central role in disease development. Tissue injury induces the release of pro-inflammatory cytokines, including interleukin-1β (IL-1β), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and high-mobility group box 1 protein (HMGB1). These mediators can alter blood-brain barrier integrity, promote migration of peripheral immune cells into the central nervous system, activate microglia and astrocytes, and ultimately initiate neuroinflammatory cascades responsible for neuronal dysfunction and synaptic damage. Neuroinflammation is closely associated with oxidative stress, another major contributor to postoperative cognitive decline. Excessive production of reactive oxygen species (ROS) during surgical stress leads to mitochondrial dysfunction, lipid peroxidation, DNA damage, protein oxidation, and impaired neuronal metabolism. Oxidative injury particularly affects hippocampal neurons, which play a critical role in learning and memory formation. Simultaneously, cerebral hypoperfusion caused by intraoperative hypotension, anemia, hypoxia, or microvascular dysfunction may exacerbate ischemic injury and reduce neuronal resilience. Growing attention has also been directed toward the influence of anesthetic agents on postoperative cognitive outcomes. Experimental studies have suggested that certain inhalational and intravenous anesthetics may influence neurotransmitter balance, calcium homeostasis, mitochondrial activity, amyloid-beta metabolism, tau phosphorylation, synaptic plasticity, and apoptotic signaling pathways. Nevertheless, current clinical evidence indicates that anesthesia alone is unlikely to be the primary cause of postoperative cognitive impairment. Instead, cognitive decline appears to result from a complex interaction between surgical stress, patient vulnerability, inflammatory responses, genetic predisposition, perioperative complications, and pre-existing neurological conditions. Numerous patient-related risk factors have been identified through prospective cohort studies and systematic reviews. Advanced age remains the strongest independent predictor of postoperative cognitive impairment because of reduced neuroplasticity and diminished cognitive reserve. Additional

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predisposing factors include pre-existing mild cognitive impairment or dementia, low educational attainment, frailty syndrome, diabetes mellitus, hypertension, atrial fibrillation, chronic kidney disease, chronic obstructive pulmonary disease, depression, anxiety, malnutrition, sensory impairment, alcohol misuse, sleep disorders, and previous cerebrovascular events. Genetic susceptibility, particularly the presence of the apolipoprotein E ε4 (APOE ε4) allele, has also been associated with increased vulnerability to postoperative cognitive decline in several investigations. Surgical and perioperative factors further influence the development of postoperative neurocognitive disorders. Longer operative duration, extensive tissue trauma, cardiopulmonary bypass, significant blood loss, perioperative transfusion, prolonged mechanical ventilation, severe postoperative pain, infections, electrolyte imbalance, prolonged intensive care unit stay, and postoperative complications all contribute to increased neurological risk. These findings emphasize the importance of comprehensive perioperative optimization rather than focusing exclusively on anesthetic management. Accurate diagnosis of postoperative cognitive impairment remains a significant clinical challenge. Standardized neuropsychological assessment tools—including the Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), Confusion Assessment Method (CAM), Trail Making Test, Digit Span Test, and computerized neuropsychological batteries—are widely used to evaluate cognitive domains before and after surgery. However, variations in testing protocols, timing of assessment, learning effects, educational background, and cultural differences continue to complicate comparisons among studies. Recent advances in biomarker research have substantially improved understanding of postoperative neurocognitive disorders. Blood and cerebrospinal fluid biomarkers such as neurofilament light chain (NfL), glial fibrillary acidic protein (GFAP), S100β protein, total tau, phosphorylated tau, amyloid-beta peptides, C-reactive protein (CRP), and inflammatory cytokines have demonstrated promising diagnostic and prognostic value. Simultaneously, magnetic resonance imaging (MRI), diffusion tensor imaging (DTI), functional MRI (fMRI), positron emission tomography (PET), and electroencephalography (EEG) have provided important insights into structural and functional alterations associated with postoperative cognitive decline. Modern perioperative medicine increasingly emphasizes preventive strategies instead of treatment after neurological injury has already occurred. Enhanced Recovery After Surgery (ERAS) programs integrate evidence-based interventions such as preoperative optimization, individualized anesthesia, multimodal analgesia, minimally invasive surgical techniques, maintenance of physiological homeostasis, early mobilization, nutritional support, delirium prevention protocols, sleep optimization, and cognitive rehabilitation. Numerous clinical trials have demonstrated that these multimodal approaches significantly reduce postoperative complications while improving neurological recovery. Digital health technologies are transforming perioperative cognitive care. Artificial intelligence and machine learning algorithms can analyze large clinical datasets to identify patients at increased risk for postoperative cognitive impairment before surgery. Wearable monitoring devices, remote cognitive assessment platforms, mobile health applications, and telemedicine-based rehabilitation programs enable continuous postoperative surveillance and individualized follow-up. Precision medicine approaches combining clinical characteristics, genomic information, imaging findings, laboratory biomarkers, and intraoperative physiological monitoring may further improve risk prediction and personalized intervention strategies. Despite considerable scientific progress, multiple challenges remain unresolved. Universal diagnostic criteria, standardized neuropsychological testing protocols, validated molecular biomarkers, long-term outcome assessment, and evidence-based therapeutic interventions require further investigation through multicenter prospective clinical studies. Furthermore, greater integration between anesthesiology, surgery, neurology, geriatrics, rehabilitation medicine, neuropsychology, and biomedical engineering will be essential for advancing research and

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improving patient outcomes. Therefore, the present study aims to comprehensively evaluate the current scientific evidence regarding the epidemiology, pathophysiological mechanisms, clinical manifestations, diagnostic approaches, preventive strategies, and management of postoperative cognitive impairment. In addition, this review explores emerging technologies, biomarker research, and precision medicine approaches that may contribute to earlier diagnosis, individualized perioperative care, and improved neurological outcomes for surgical patients in the future.

Literature Review. Postoperative cognitive impairment has become one of the most extensively investigated topics in perioperative medicine during the past three decades. Increasing life expectancy, advances in surgical technology, and the growing number of elderly patients undergoing major operations have stimulated substantial scientific interest in postoperative neurocognitive disorders. Contemporary literature demonstrates that postoperative cognitive impairment is not a single disease entity but rather a heterogeneous syndrome influenced by multiple biological, surgical, anesthetic, and patient-related factors. The concept of postoperative cognitive dysfunction (POCD) first gained widespread recognition during the International Study of Postoperative Cognitive Dysfunction (ISPOCD), one of the largest multicenter investigations in perioperative neuroscience. ISPOCD studies demonstrated that cognitive decline may occur after both cardiac and non-cardiac surgery, particularly among elderly patients. These investigations showed that deficits in memory, executive function, attention, and psychomotor speed could persist for several weeks or months after surgery, highlighting the need for systematic perioperative cognitive assessment. More recently, the International Perioperative Cognition Nomenclature Working Group recommended the umbrella term perioperative neurocognitive disorders (PND) to unify postoperative delirium, delayed neurocognitive recovery, and postoperative neurocognitive disorder within a standardized diagnostic framework. This updated terminology has facilitated greater consistency among clinical studies and has improved communication between anesthesiologists, neurologists, geriatricians, surgeons, and neuropsychologists. One of the most consistently reported mechanisms underlying postoperative cognitive impairment is neuroinflammation. Numerous experimental and clinical investigations have demonstrated that surgical trauma initiates a systemic inflammatory response characterized by increased circulating concentrations of interleukin-1β (IL-1β), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), high-mobility group box-1 protein (HMGB1), and C-reactive protein (CRP). These inflammatory mediators alter blood-brain barrier permeability, allowing peripheral immune cells and cytokines to enter the central nervous system. Activation of microglia and astrocytes subsequently induces prolonged neuroinflammation, synaptic dysfunction, impaired neurogenesis, and neuronal apoptosis, particularly within the hippocampus, a brain region essential for learning and memory. Animal studies further support the neuroinflammatory hypothesis. Rodent models have shown that surgical procedures induce activation of hippocampal microglia, increased cytokine production, oxidative injury, and behavioral deficits that closely resemble postoperative cognitive dysfunction observed in humans. Experimental administration of anti-inflammatory agents, including selective cyclooxygenase inhibitors, corticosteroids, and cytokine-targeted therapies, has demonstrated partial neuroprotective effects, although translation into routine clinical practice remains limited. Several studies have investigated the influence of surgical factors on postoperative neurocognitive outcomes. Cardiac surgery involving cardiopulmonary bypass has traditionally been associated with a relatively high incidence of cognitive decline because of cerebral microembolization, inflammatory activation, and alterations in cerebral blood flow. Nevertheless, recent evidence demonstrates that major orthopedic, thoracic, vascular, gastrointestinal, transplant, oncological, and neurosurgical procedures may also produce clinically significant postoperative cognitive impairment through similar inflammatory and physiological mechanisms. Pain management has emerged as another important

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determinant of postoperative neurological recovery. Severe postoperative pain activates neuroendocrine stress pathways, increases sympathetic activity, and stimulates systemic inflammatory responses, thereby potentially worsening cognitive dysfunction. Contemporary studies consistently support multimodal analgesia, opioid-sparing techniques, regional anesthesia, and individualized pain management as effective strategies for reducing postoperative neurocognitive complications. Sleep quality represents another growing area of investigation. Hospitalization, environmental noise, pain, intensive care admission, and circadian rhythm disruption frequently impair postoperative sleep architecture. Clinical studies indicate that sleep deprivation promotes neuroinflammation, oxidative stress, impaired glymphatic clearance, and cognitive dysfunction. Consequently, interventions aimed at improving perioperative sleep have gained increasing recognition within Enhanced Recovery After Surgery (ERAS) protocols. Significant advances have also occurred in diagnostic methodology. Traditional neuropsychological assessments, including the Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), Confusion Assessment Method (CAM), Trail Making Test, Stroop Test, Rey Auditory Verbal Learning Test, and Digit Span Test remain widely used. However, computerized cognitive batteries now enable more sensitive evaluation of multiple cognitive domains while minimizing examiner variability and improving longitudinal assessment. Biomarker research has expanded considerably over the last decade. Blood biomarkers such as neurofilament light chain (NfL), glial fibrillary acidic protein (GFAP), S100β, neuron-specific enolase (NSE), total tau, phosphorylated tau, amyloid-beta peptides, and inflammatory cytokines have demonstrated promising diagnostic and prognostic potential. Neuroimaging modalities—including magnetic resonance imaging (MRI), diffusion tensor imaging (DTI), resting-state functional MRI (rs-fMRI), positron emission tomography (PET), and electroencephalography (EEG)—have further improved understanding of structural and functional alterations associated with postoperative cognitive disorders. Despite substantial scientific progress, several important knowledge gaps remain. Considerable heterogeneity persists regarding diagnostic criteria, neuropsychological testing protocols, follow-up duration, biomarker validation, and definitions of postoperative cognitive impairment. Furthermore, relatively limited evidence is available from low- and middle-income countries, where demographic characteristics, healthcare resources, perioperative practices, and population genetics may differ substantially from those reported in high-income settings. Future multicenter prospective studies employing standardized diagnostic criteria, harmonized biomarker assessment, advanced neuroimaging, and long-term follow-up are therefore essential to establish evidence-based strategies for prevention, early diagnosis, and personalized management of postoperative cognitive impairment. Overall, the available scientific literature consistently demonstrates that postoperative cognitive impairment represents a multifactorial neurological syndrome arising from complex interactions among systemic inflammation, oxidative stress, blood-brain barrier disruption, patient vulnerability, surgical trauma, anesthetic exposure, and perioperative physiological disturbances. Comprehensive multidisciplinary perioperative care, combined with precision medicine approaches and emerging digital technologies, offers the greatest potential for improving cognitive outcomes in surgical patients.

Results and Discussion. The present study synthesized current scientific evidence regarding the incidence, mechanisms, risk factors, diagnosis, prevention, and management of postoperative cognitive impairment (PCI). Analysis of recent clinical studies, systematic reviews, meta-analyses, and international clinical guidelines indicates that postoperative cognitive impairment is a multifactorial neurological complication resulting from complex interactions between patient characteristics, surgical trauma, anesthetic exposure, inflammatory responses, and perioperative management. The reviewed evidence consistently demonstrates that elderly patients represent the population at greatest risk for postoperative cognitive disorders. Age-related reductions in cognitive

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reserve, neuroplasticity, mitochondrial function, and cerebrovascular integrity substantially increase susceptibility to perioperative neurological injury. Patients with pre-existing cognitive impairment, cardiovascular disease, diabetes mellitus, frailty syndrome, chronic kidney disease, and previous cerebrovascular events also exhibit significantly higher rates of postoperative neurocognitive complications.

Major Risk Factors Identified. Comprehensive evaluation of the literature identified several independent predictors associated with postoperative cognitive impairment.

Table 1. Major Risk Factors Associated with Postoperative Cognitive Impairment

Risk Factor	Mechanism	Clinical Impact
Advanced age	Reduced cognitive reserve and neuronal plasticity	Very high
Pre-existing cognitive impairment	Limited compensatory capacity	Very high
Major surgical procedures	Increased inflammatory response	High
Prolonged anesthesia	Greater physiological stress	Moderate–High
Diabetes mellitus	Microvascular dysfunction	High
Hypertension	Cerebral vascular injury	Moderate
Postoperative pain	Neuroendocrine activation	Moderate
Sleep disturbance	Impaired neuronal recovery	Moderate
Postoperative infection	Enhanced systemic inflammation	High
Intensive care admission	Delirium-related complications	High

These findings indicate that postoperative cognitive decline is rarely attributable to a single factor. Instead, cumulative exposure to multiple perioperative stressors determines the overall neurological outcome. Current evidence demonstrates that neuroinflammation remains the central biological mechanism responsible for postoperative cognitive impairment. Surgical tissue injury stimulates systemic release of inflammatory mediators, including IL-1β, IL-6, TNF-α, and C-reactive protein. These mediators increase blood-brain barrier permeability and activate microglial cells, leading to neuronal dysfunction within the hippocampus and prefrontal cortex. Oxidative stress represents another major contributor. Increased production of reactive oxygen species causes mitochondrial dysfunction, impaired synaptic transmission, lipid peroxidation, and neuronal apoptosis. The interaction between oxidative stress and inflammation amplifies neurological injury and delays cognitive recovery. Furthermore, disturbances in cerebral perfusion, endothelial dysfunction, metabolic imbalance, and postoperative hypoxia contribute to impaired neuronal function. The combination of these mechanisms explains why cognitive impairment frequently persists beyond the immediate postoperative period.

Diagnostic Assessment. The reviewed studies consistently support standardized cognitive assessment before and after surgery.

Table 2. Common Diagnostic Tools Used in Clinical Practice

Assessment Tool	Primary Purpose	Clinical Advantages
Mini-Mental State Examination (MMSE)	Global cognitive screening	Simple and widely available
Montreal Cognitive Assessment (MoCA)	Detection of mild cognitive impairment	High sensitivity
Confusion Assessment Method (CAM)	Diagnosis of postoperative delirium	Rapid bedside evaluation

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Trail Making Test	Executive function assessment	Sensitive to attention deficits
Digit Span Test	Working memory evaluation	Easy administration
Computerized cognitive batteries	Comprehensive neuropsychological testing	High reproducibility

Emerging biomarkers—including neurofilament light chain (NfL), glial fibrillary acidic protein (GFAP), S100β protein, total tau, phosphorylated tau, amyloid-beta peptides, and inflammatory cytokines—demonstrate promising diagnostic value. Neuroimaging techniques such as magnetic resonance imaging (MRI), diffusion tensor imaging (DTI), functional MRI (fMRI), and positron emission tomography (PET) further improve understanding of postoperative neurological alterations.

Preventive Strategies. Analysis of contemporary clinical evidence identified several interventions associated with improved cognitive outcomes.

Table 3. Evidence-Based Preventive Measures

Intervention	**Expected Benefit**
Preoperative cognitive screening	Early identification of high-risk patients
Individualized anesthetic management	Reduced neurological stress
Multimodal analgesia	Improved pain control with reduced opioid exposure
Maintenance of normothermia and oxygenation	Prevention of cerebral hypoxia
Early mobilization	Enhanced neurological recovery
Nutritional optimization	Improved metabolic resilience
Delirium prevention protocols	Lower incidence of postoperative delirium
Cognitive rehabilitation	Faster restoration of cognitive function

Implementation of Enhanced Recovery After Surgery (ERAS) protocols consistently demonstrated reductions in postoperative complications, shorter hospital stay, earlier functional recovery, and improved postoperative cognitive performance. One of the most important findings emerging from recent literature is the increasing role of artificial intelligence (AI) in perioperative medicine. Machine learning algorithms integrating demographic variables, laboratory data, imaging findings, intraoperative physiological parameters, and electronic health records can accurately identify patients at elevated risk before surgery. Digital cognitive monitoring, wearable sensors, remote assessment platforms, and predictive analytics enable continuous postoperative surveillance and individualized rehabilitation strategies. Precision medicine approaches combining genomic biomarkers with perioperative clinical data are expected to improve personalized neuroprotective interventions.

Discussion**.** The findings of this review strongly support the contemporary understanding that postoperative cognitive impairment is a complex and multifactorial neurological disorder rather than an isolated consequence of anesthesia or surgery. The observed association between advanced age, systemic inflammation, oxidative stress, vascular dysfunction, and impaired cognitive recovery is consistent with evidence reported in major international cohort studies and systematic reviews. The central role of neuroinflammation identified in this review agrees with current experimental investigations demonstrating activation of microglia and astrocytes following surgical trauma. Persistent inflammatory activation contributes to neuronal dysfunction, synaptic loss, and impaired neurogenesis, thereby explaining prolonged cognitive deficits observed in vulnerable patients. These mechanisms further emphasize the importance of perioperative anti-inflammatory strategies and optimization of surgical techniques to minimize tissue injury. The literature also indicates that oxidative stress and mitochondrial dysfunction play complementary roles in postoperative neurodegeneration. Preservation of mitochondrial integrity through optimized oxygen delivery, maintenance of hemodynamic stability, adequate pain management, and reduction of perioperative

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metabolic stress may therefore represent important therapeutic targets. The present findings suggest that anesthesia should not be considered the sole determinant of postoperative cognitive decline. Rather, cognitive impairment reflects interactions among surgical stress, patient vulnerability, inflammatory responses, perioperative complications, and pre-existing neurological conditions. Consequently, future preventive strategies should focus on comprehensive perioperative management rather than isolated anesthetic modifications. Another significant observation concerns the growing importance of standardized perioperative cognitive assessment. Routine application of validated instruments such as MMSE, MoCA, and CAM before and after surgery enables early recognition of cognitive deterioration and facilitates timely intervention. Integration of objective biomarkers with conventional neuropsychological testing may further improve diagnostic accuracy and risk stratification. Recent advances in molecular medicine provide additional opportunities for individualized care. Biomarkers including neurofilament light chain, GFAP, tau protein, inflammatory cytokines, and advanced neuroimaging findings appear capable of identifying subclinical neuronal injury before overt cognitive symptoms develop. Nevertheless, additional multicenter validation studies remain necessary before routine implementation in clinical practice. The rapid development of artificial intelligence represents another promising direction. Machine learning models capable of analyzing thousands of perioperative variables may substantially improve prediction of postoperative cognitive impairment and support personalized decision-making. Integration of AI with electronic health records, continuous physiological monitoring, and wearable technologies may transform perioperative neurological care during the coming decade. Despite considerable progress, important limitations remain within the current literature. Significant heterogeneity exists regarding diagnostic criteria, cognitive testing methodology, follow-up duration, biomarker selection, and study populations. Most available evidence originates from high-income countries, while data from low- and middle-income healthcare systems remain comparatively limited. Future international multicenter prospective studies employing standardized diagnostic protocols, harmonized biomarker assessment, and long-term follow-up are essential for establishing universally applicable clinical recommendations. Overall, the evidence reviewed in this study demonstrates that postoperative cognitive impairment represents a major challenge in modern perioperative medicine. Successful prevention requires multidisciplinary collaboration among anesthesiologists, surgeons, neurologists, geriatricians, rehabilitation specialists, psychologists, intensive care physicians, and nursing professionals. Comprehensive perioperative assessment, individualized anesthetic care, evidence-based ERAS implementation, digital monitoring technologies, biomarker-guided diagnosis, and precision medicine approaches collectively provide the greatest opportunity for reducing postoperative cognitive complications and improving long-term neurological outcomes.

Conclusion. Postoperative cognitive impairment (PCI) remains one of the most significant neurological complications encountered in modern perioperative medicine. The present review demonstrates that postoperative cognitive decline is a complex, multifactorial condition resulting from the interaction of patient-related vulnerability, surgical stress, systemic inflammation, oxidative stress, blood-brain barrier disruption, cerebral hypoperfusion, metabolic alterations, and perioperative clinical management. Rather than being attributable to a single etiological factor, postoperative neurocognitive disorders arise from multiple interconnected biological pathways that collectively influence neuronal survival and cognitive recovery. The analysis of current scientific evidence confirms that advanced age, pre-existing cognitive impairment, frailty, cardiovascular and metabolic diseases, prolonged surgery, postoperative pain, infections, and intensive care admission are among the most important predictors of postoperative cognitive dysfunction and delirium. These findings emphasize the necessity of comprehensive preoperative risk assessment to identify vulnerable patients before surgery

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ISSN: 2058-5519|Impact factor-15.9
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and to implement individualized preventive strategies. The review further highlights neuroinflammation as the principal pathophysiological mechanism underlying postoperative cognitive impairment. Activation of systemic inflammatory pathways following surgical trauma initiates complex neuroimmune responses that impair synaptic plasticity, neuronal communication, and hippocampal function. Oxidative stress, mitochondrial dysfunction, endothelial injury, and cerebral microvascular alterations further aggravate neurological damage, explaining the persistence of cognitive deficits in susceptible individuals. In conclusion, postoperative cognitive impairment should be regarded not merely as a postoperative complication but as an important component of comprehensive perioperative patient care. Early identification of high-risk individuals, implementation of evidence-based preventive strategies, and adoption of innovative diagnostic technologies will be essential for improving long-term cognitive outcomes and reducing the global burden of postoperative neurocognitive disorders.

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