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Letter to the Editor

Delirium-a letter update

Sir,

Delirium is essentially acute brain failure that results from stressors that surpass the brain's homeostatic reserve.[1] Delirium is caused by a combination of risk factors that are both predisposing and precipitating. Advanced age, frailty, medication exposure or withdrawal, sedation depth, and sepsis are all known risks. Stressors most likely have coordinated rather than independent effects, and the systems they affect are linked rather than separate. Changes in the blood-brain barrier and the central nervous system's de novo synthesis or elaboration of inflammatory mediators account for the pathophysiology of delirium. It also seems that neuro-inflammatory activity contributes to the dysregulation of neurotransmitters.

The intensive care units of today have saved the lives of an increasing number of patients. Survival is made possible by interdisciplinary teams that specialize in and concentrate on certain organs. The organ whose primary function is to guarantee one's survival is the brain. Because the brain is unable to operate properly, acute brain failure, also known as delirium, increases the likelihood of death.[2] There is a bidirectional association between systemic illness and brain dysfunction. A wide range of physiological and behavioural impacts, such as the activation of the hypothalamic-pituitary-adrenal axis, changes in the functioning of neurotransmitter systems, and immunological function, can result from changes in this environment.[3]

In the real world, mind-body dualism approach negatively impacts patient care. ‘Mind’ is a philosophical idea revived in the seventeenth century by Rene Descartes and is being pursued today by modern medicine practitioners despite the lack of supporting evidence from science and the rejection of its applicability by the majority of philosophers.[4] The clinicians overlook the reality that every acute or chronic systemic illness involves altered brain functioning. Sickness behaviour is caused by systemic ailments. The typical physiological adaptive response to systemic inflammation is behaviour, which is typified by lethargy, decreased activity, and decreased hunger. This behaviour is intended to conserve energy and reduce exposure to other stressors. Brain dysfunctions are frequently diagnosed as "mind problems" and are not addressed with currently available, efficient medication treatments from a psychiatrist. In everyday medical practice, this kind of dualism approach has detrimental effects on patient care.

Attention must be paid to improving the categorization of delirium subtypes or phenotypes (i.e., psychomotor classifications). Recent progress in the association of clinical phenotypes with clinical outcomes broadens our understanding and identifies potentially modifiable targets. Several delirium biomarkers have been studied in critical care, with disrupted functional connectivity being the most accurate in detecting delirium. Recent advances emphasize the importance of mechanistic pathways such as cholinergic activity and glucose metabolism in delirium as an acute and partially modifiable brain dysfunction. Pharmacologic agents have been evaluated in randomized controlled prevention and treatment trials, with dismal results. Antipsychotics are still widely used after 'negative' trials, but they may play a role in certain subtypes. Antipsychotics, on the other hand, do not appear to improve clinical outcomes.[5]

Alpha-2 agonists may have greater current and future application potential. The role of thiamine appears promising, but more research is needed. In the future, clinicians should prioritize mitigating predisposing and precipitating risk factors as much as possible. Further research is needed to identify modifiable targets within individual delirium psychomotor subtypes and clinical phenotypes that have the potential to improve not only delirium duration and severity, but also long-term outcomes such as cognitive impairment.[5]

A precise approach to reducing delirium in critical illness requires a broad understanding of its complexity due to its multifactorial nature, different clinical phenotypes, and potential neurobiological causes. We implore medical professionals to treat patients with a perspective that the brain is the organ that allows for survival, and to recognize and treat any brain malfunction as quickly as possible using cutting-edge medication preferably in liaison with a psychiatrist. This probably could help reduce the incidence of delirium in patients with comorbid medical and psychiatric disorder.

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