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Review on Delirium Disorder

Miss. Nandini Pravinrao Kalmore, Dr. Avinash S. Jiddewar
Miss. Ishwari Bhauji Gate, Miss.Urvashi Sunil Jadhav
NSPM College of Pharmacy, Darwha, Yavatmal

Abstract: *Delirium, a condition characterized by an acute change in attention, awareness and cognition, is caused by a medical condition that cannot be better explained by a pre-existing neurocognitive disorder. Multiple predisposing factors (for example, pre-existing cognitive impairment) and precipitating factors (for example, urinary tract infection) for delirium have been described, with most patients having both types. Because multiple factors are implicated in the aetiology of delirium, there are likely several neurobiological processes contributing to delirium pathogenesis, including neuroinflammation, brain vascular dysfunction, altered brain metabolism, neurotransmitter imbalance and impaired neuronal network connectivity. The Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) is the most commonly used diagnostic system upon which a reference standard diagnosis is made, although many other delirium screening tools have been developed owing to the impracticality of using DSM-5 in many settings. Pharmacological treatments for delirium (such as antipsychotic drugs) are not effective, reflecting substantial gaps in our understanding of pathophysiology. Currently, the best management strategies are multidomain interventions that focus on treating precipitating conditions, medication review, managing distress, mitigating complications, and maintaining engagement to environmental issues. Effective implementation of delirium detection, treatment and prevention strategies remains a major challenge for healthcare organizations.*

Keywords: acute confusion,disorientation,altered mental status,hallucination,agitation,inattention , disorganized thinking ,memory problems and sleep-wake cycle disruption

I. INTRODUCTION

The term `delirium' is derived from the latin word delirare,meaning `to go out of the furrow',that is, to deviate from straight line , to deranged delirium is sever neuropsychatric syndrome that characterized by acute onset of deficits in defattention and other aspects of congition .patient also often have altered arousal , from reduced responsiveness at the level of near-coma to hypervigilance . and sever agitation .they may also experienced highly distressing symptoms of pyschosis ,including delusion and hallucination -delirium is a clinical syndrome thet usually develops in the elderly.it is characterized by an alteration of attention,consciousness and congition with a reduced ability to focus ,sustain or shift attention -the clinical presentation can vary,usually with psychometer behavioral disturbance such as hyperactivity or hypoactivity with sleep duration and architecture impairment -defination of delirium is caused by an underlying medical condition and is not better explained by another preexisting,evolving or established neurocognitive disorder .the underlying cause of delirium can vary widely and involve anything that stresses the baseline homeostasis of a vulnerable patient.example include substance intoxication or widrawl medication side effect,infection,surgery ,metabolic dearrangments ,pain,or even simple condition -A Variety of term has been used in the literature to describe delirium,including ‘'acute brain syndrome,'' ‘'acute cerebral insufficiency,.however ,delirium is now the prefferd term and has been suggested that acute confussional state should be only accepted synonym for this syndrome -delirium is an acute medical emergency with psychiatric menifastations ,which is seen across different treatment settings ,with higher prevalence in intensive care unit[ICUs]and palliative care settting -despite a exponential rise in delirium research,understanding of pathophysiological process underlying remain delirium low regardless of cause,delirium presents as a reasonably common syndrome ,particularly when it result from direct or indirect brain injury ,but progresss is being hindered by a by categorical organization.Robust description which adequately reflects the severity spectrum of the syndrome ,outcomes of ranging management strategies is required .classifying all delirium presentation

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under one umbrella term may therefore be hindering proper advancement. -Although it is seen across all the medical-surgical settings,it is often underrecognized and undertreated .some of the developed countries have recognize the importance of early identification and prevention of delirium in various treatment settings.many authors have developed bedside assessment instruments to detect delirium at the earliest and have also developed countries have also developed various intervention packages ,suchwawa as the hospital Elder life program (HELP) and assess,prevent,and manage pain ,Both Spontaneous Awaking trial (SAT) and Spontaneos brathing trial (SBT) : choice of analgesia and sedation;and the delirium;assess, prevent ,and Manage Early mobility and Exercise ,and family engagement and empowerment bundle for early detection,prevention,and management of delirium -The terminology overlap of delirium and acute encephalopathy has recently been under scrutiny . Acute encephalopathy describes a pathobiological brain precess , presenting clinically as subsyndromal delirium,delirium,or coma .The term 'Delirium' facilitates patient-centred focus allowing screening,preventative measures, and psychological maintenance

EPIDERMIOLOGY

PREVALENCE ESTIMATES

Epidermiological studies of delirium provide accurate estimates when standard diagnostics criteria or validated detection tools are used and when the study sampleis representative of the population being studied. Here, we present data from quality studies or systematic reviews that adhere to established standards of reporting,such as the prefferedreporting items for systematic reviews and Meta-Analyses (PRISMA) checklist.

Delirium is the most common psychiatric syndrome observed in hospitalized patients.the incidence of general medical wards ranges from 11% to 42% ,and it is as high as 87% among critically ill patients . A preexisting diagnosis of dementia increases the risk for delirium fivefold. Other risk factors include sever medical illness age,sensory impairment ,and male gender .

Delirium prevalence varies considerabely by patient group and setting. Delirium is common in hospitalized older adults in general medical settings,with a 2020 meta-analysis of 33 studies of medical patients finding an overall delirium prevalence of 23% .the prevalence of delirium after surgery ranges from low single-figure percentages in medically well patients undergoing minor elective surgery to 20% in high-risk patients undergoing major surgery,especially under emergency condition . for example , in a systematic review of delirium risk in patients undergoing in coronary artery nypass grafting (CABG),delirium prevelance was 24% in studies in which a diagnostic instrument was used The prevalence of delirium in infants ,children and adolescence is less well understood . studies from Europe and the USA report prevalence estimates delirium in children and adolescents ranges from 4%to almost 50% in critically ill children and adolescents in one study from the USA more than hslf of infants under 2years of age experienced delirium while critically ill.

The prevelance of subsyndromal delirium is highly variable and depends on the population being studied and which delirium definition is used .however ,in one study form north America,almost two- third of adults over 65 years of age who were admitted to an inpatient medical or geriatric service had subsyndromal delirium

RISK FACTOR

The risk of delirium is determined by predisposing risk factors (that is,the background characteristics of patients)the precipitatingrisk factors (that is acute insults,injury or drugs). Predisposing risk factors for delirium include increased age , cognitive impairment (such as dementia or developmetal delay), frailty .comorbidities (including cardiovascular renal disease),depression or other psychiatric illness ,Alcohol use, poor nutritional status and visual and hearing impairment .total risk depend on the number of risk factor in each individual and ,where applicable ,their severity ; for example , frailty men,which typically enocompasses a number of risk factor ,is strongly associated with delirium risk ,and degree of cognitive shows a strong linear association with delirium risk .Precipitating factor for delirium span wide range of different kinds of insults ,including amongst other,acute medical illness (such as sepsis,hypoglycemia,stroke and liver failure)trauma(such as fractures or head injury)surgery ,dehydration and psychological stress.typically ,more than one risk factor present in patient ,in addition drug used and widrawl, and medical changes are associated with delirium .

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Precipitating factors for delirium span a wide range of different kinds of insults, including, amongst others, acute medical illness (such as sepsis, hypoglycaemia, stroke and liver failure), trauma (such as fractures or head injury), surgery, dehydration and psychological stress. Typically, ore than one risk factor is present in patients50,51. In addition,drug use and withdrawal, and medication changes are associated with delirium. Of note, benzodiazepines, dihydropyridines (L-type calcium channel blockers typically used in the treatment of hypertension), antihistamines and opioids may convey the highest risk of delirium, although insufficiently managed pain may itself be a risk factor52,53; however, the exact relationship between pain medication, pain management and delirium risk remains unclear. In addition to common premorbid factors, specific healthcare setting-related factors,such as mechanical ventilation.

DIAGNOSIS

There are multiple challenges to establishing the diagnosis of delirium in a timely fashion ,including the fluctuating course of symptoms ,difficulty conducting cognitive testing during the extremes of psychomotor disturbance ,overlooking the hypoactive phenotype and the need to ascertain base line cognitive functioning .

Multiple validated delirium screening tools with high sensitivity and specifying have been developed ,including the confusion assessment method and the 4AT rapid clinical test for deliriym ,which improve the detection of delirium by a variety of health care proffessinals.

DIFFERENTIAL DIAGNOSIS

A careful medication history can clarify the risk of serotonin syndrome ,particularly in patient on standing serotonergic medications who then require additional serotonergic medications who then require additional seroternigic agent addresss acute medical issues . A broader differential diagnosis can be considered in the context of the patient presentation at the time of consultation.

Delirium is medically ill patient is often multifactorial,and while attention is importantly given to broad surveillance and monitoring of contributing variables , the roke of psychiatric consultant often involves focusing specifically on potential neuropsychatric process.substance withdrawl mat require a careful medication history to identify and clarify use pattern, even of prescribed benzodiazepines and opoids ;this may also inform seizure risk assessment .

The diagnostic process involves two fundamental steps . first, a bedside clinical assessment of the patient is performed to ascertain the level of attention arousal and the presence of other cognitive deficits ,psychotic features or other mental status abnormalities .second,evidence of acute change from baseline attention and awareness ,which may fluctuate in presence and severity is sought from caregivers or staff who know the patient, fpor medical record or ,less commonly ,from from the clinician's own knowledge of the patient.

Whie scope of practice may very with training experience ,it would be reasonable to recommend a neurological consultation when there are focal neurological finding ,including any new asymmetry on physical examination,movement abnormalities ,sustained poor mental status (concerning for status epilepticus),and abrupt deterioration in mental status after a period of delirium previously marked primarily by inattention and disorientation.

IMPACT OF DELIRIUM

A meta analysis of delirium in the elderly showed that even after controlling for confounding factors, including age,sex,dementia,comorbid illness,and illness severity ,delirium is independently associated with a two fold increase in risk of death ,a 2.4-fold increase in risk of instituationalization ,and a 12.5- fold increase in risk of dementia.

Delirium has also been strongly associated with sustained decline in physical function, with the average loss of one activity of daily living per delirious episode, sustained at 6-month foolow up. In patient with and without dementia,multiple symptoms of delirium have been shown to shown to persist for 12month after the onset of delirium. In addition to significant patient mortality and negative functional outcomes , delirium is also associated with high health care costs. On average, hospital stays are 5-10 days longer for patient who develop for delirium than for patient without delirium.

1. cognitive and physiological impact

Short term

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-severe confusion and disorientation
-patient may not know where they are or what time it is
-impaired attention and concentration
-memory disturbance
-hallucination or delusions
-disorganized thinking
long term
-increased risk of developing persistant cognitive imperment
-higher likehood of later dementia,especially in older adults
2. functional impact
-loss of independence in activities like walking,eating,or self-care
-increased risk of falls,injuries,and accidental removal of medical devices
-s;ower recovery from surgery or illness

PATHOPHYSIOLOGY

In clinical studies,disentangling the contribution of multiple triggers and process ,such as hypoxia,inflammation and sedation,is possible only to a limited extent .thus studies in animals relatively 'PURE' aetiologies are important to allow strong predictions to be made about which biological factors have a causative role in exceeding key threshold and triggering delirium.

As severable different factors are impactable in the aetiology of delirium,including sepsis,fractures,surgery medication changes, hypoglycemia and leaver failure ,it follow that distinct neurobilogical mechanism or combination of them are involved in delirium pathogenesis.

Inflammation,hypoxia,and oxidative stress all contribute to increased brain exposures to toxin and a hypocholinergic-hyperdopaminergic state.inflammation creates a vulnerable physiological state with impaired brain function and increased permability of the blood brain barrier.suspectibility to circulating deliriogenic medications,endogenious toxins,and proinflammatory cytokines may cause or sustain delirium

That multiple aetiological factors may contribute to delirium in a patient, several neurobiological mechanism may also interact to produce the observed syndrome and it remains difficult to clarify delirium either on distinct aetiologies or distinct neurobiological mechanism.nonetheless , delirium with a single aetiologies (that is,involve single causative factor)has been demonstrated in human studies of hypoxia , hypoglycemia and cholinergic antagonism .

The reticular activating system is primarily involved in regulating alertness and attention are a fundamental substrate for all domains of cognition,the cognitive deficits seen in delirium are diffuse and sonspecific .thus,any cognitive deficits ecilited on bedside testing during a delirious state should be interpreted with caution.other neurotransmitter derangments implicated in delirium include melatonin deficiency ,resulting in sleep wake cycle disruption,and excess norepinephrine and glutamate.

As several different factors are implicated in the aetiology of delirium, including sepsis, fractures, surgery, medication changes, hypoglycaemia and liver failure, it follows that distinct neurobiological mechanisms or combinations of them are involved in delirium pathogenesis. Unravelling these mechanisms in different patient populations and clinical settings is necessary. However, given that multiple aetiological factors may contribute to delirium in a patient, several neurobiological mechanisms may also interact to produce the observed syndrome and it remains difficult to classify delirium based either on distinct aetiologies or on distinct neurobiological mechanisms. Nonetheless, delirium with a single aetiology (that is, involving a single causative factor) has been demonstrated in human studies of hypoxia, hypoglycaemia79 and cholinergic antagonism80. As the different aetiologies lead to a shared, albeit heterogeneous, core syndrome, some researchers have proposed that a common pathogenetic pathway underpins delirium; however, evidence is currently lacking for such a single pathway. Although adopting the term 'delirium' has been valuable in highlighting its importance for patient outcomes, 'lumping' together all types of delirium when researching the

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underlying neurobiology of the disorder might be less useful than 'splitting' the syndrome into different subtypes based on aetiological contributors (that is, the underlying acute pathophysiological disruption or precipitants leading to delirium)10

DEGERATION BRAIN VULUERABILITY

In higher-risk individuals,delirium is a fsilure of the vulunerable brain to show resielence in response to an acute strassor .this vulunerability can be caused by a multitude of process are not mutually exclusive .key process include changes in brain connectivity ,neuroinflammatoey and glial cell alteration and vascular changes.first,brainnetworkvconnectivity is impaired by ageing and neurodegeneration and cholinergic and noradrenergic neuronal populations degenerate with increasing age and dementia,both of which have consequence for network and cognitive function in response to acute stressors.

The interaction between underlying a predisposition and a superimposed acute stressor is key to delirium pathophysiology .although major acute streassor (such as head injury stroke and septic shock) can trigger delirium ,even in relisient individuals,predisposing factors,such asold age,frailty and existing cognitive impairment ,also substaintially increased delirium risk .prior cognitive impairment is a progressive risk fractor as a baseline cognitive decrease ,delirium risk is increase in a linear fashion and,therefore,in both rodent and humans,less-severe acute stressors.

Furthermore ,astrocytes are metabolically impaired by lossing interaction with healthy neurons during neurodegeneration also trigger in the brain vascular reactivity ,disruption of the transport of important plasma proteins in the brain and leakiness of the blood brain barrier (BBB),potentially making the brain more vulnerable to disruption of energy or oxygen supply and the effect of circulatory inflammatory molecules .

This list is not exhaustive,and the potential for acute stress to distrupt function in the vulnursble brain can be further potentiated by poor nutrition and hydration and by ageing associated renal and impairment,causing slower metabolic and clearance of potentially neurotoxic drugs and metabolites. Furthermore,impaired BBB function may increase permability systemically administered medications that can have a psychoactive and deleterious effect on brain function.therefore,acute derangments occurring during acute illness or trauma have greater effect on a vulnerable brain than on a young healthy brain.

BRAIN ENERGY METABOLISM

The brain requires a large amount of energy, and as either oxygen or glucose deficiency can markedly constrain brain function, acute illness might impair brain metabolism in multiple ways. The long- standing 'cerebral metabolic insufficiency hypothesis proposes that delirium is caused by the failure to meet the energy requirements of the brain, and although this hypothesis is supported by data from various sources, as we discuss below, substantial gaps in our understanding persist owing to the paucity of studies directly testing this hypothesis in animals or patients.

Effective blood oxygenation is essential to avoid brain sequelae. As several clinical scenarios can lead to hypoxia, this is a highly plausible driver of delirium. For example, impaired lung function during respiratory distress causes hypoxemia, haemodynamic shock can impair blood flow to the brain and, even after blood pressure normalization,microcirculatory impairment can still result in decreased brain perfusion and tissue hypoxia. In small studies in patients, delirium was associated with decreased cerebral blood flow (30–50%) that resolved at the time of recovery92 and with impaired autoregulation in patients with sepsis93. Consistent with a possible role for hypoxia, cerebrospinal fluid (CSF) lactate levels were significantly elevated in hip fracture and general medical patients with delirium94,95. Brain tissue hypoxia can be assessed in patients using near infra-red spectroscopy (NIRS), and a systematic review found evidence for an association between low regional cerebral oxygenation and delirium, although there are still too few studies for a meta-analysis.

In addition to gross changes in blood flow and blood oxygenation, there is evidence for impaired neurovascular coupling in ageing and dementia. Thus, even when brain perfusion is normal, dynamic increases in regional brain activity may not be adequately supported by functional hyperaemia, meaning that activity-dependent increases in oxygen and glucosecannot be achieved in specific brain regions when required, with likely consequencesfor brain

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functions requiring this dynamically increased flow. Finally, even in normoxia, mitochondrial dysfunction, as occurs in dementia99, might impair utilization of pyruvate and other substrates for ATP generation.

PREVENTION

In provided detail guideline about prevention of delirium it summarized it follow;

-Avoid polparmacy:carriew out a medication review:both type and number of medictions

-use anticholinergic ,opioid and sedative carefully

-maintain hydration status

-train caregivers and nursing staff to recognize delirium

-assessment especially in elderly

-avoid moving people within and between wards or rooms unless absolutely necessary

In a clinical seetings with high rates of delirium,such as critical care and postoperstive units,it is helpful to predict risk of delirium inform prognosis and the and the risk-benefit analysis of elective surgery .the PRE-DELIRIC(PREdiction of DELIRIum in intensive care patient) is a delirium prediction model for intensive care patients based on nine clinical and demographic factors at the time of admission,which have been validated in seven countries.A similar delirium prediction store (Delphi;delirium prediction based on hospital information) developed for general surgery.

Delirium prevention focuses on identifying at risk individual reducing modifiable triggers through comprehensive ,non-pharmacologic intervention.key strategies include refular orientation ,ensuring access to hearing ads and glasses, promoting healthy sleep-wake cycles,early mobilization,adequate hydration and nutrition ,effective pain control and minimize high-risk medication such as benzodiazepines and anticholinergics .preventing sensory deprivation ,trating underlying medical conditions promptly(e.g., infection,hypoxia,dehydration) and involoving family members also significantly reduce risk.

Additional measures to prevent delirium focus on optimizing the patient environment and reducing physiological stressors that can triggers cognitive decline .ensuring adequate lighting during the day reduced stimulation at night help maintain cardiac rhythm,regular cognitive stimulation such as conversation ,simple activities and involvement in daily routines-can reduced disorientation .avoiding unneccesary transfers between rooms or unit reduces confusion.while early identification and

treatment constipation and urinary retention,hypoxia and pain prevent medical complication that may precipitate delirium.reducing the use of invansive devices,such as urinary catheters and physical restraints ,further lowers the risk encouraging family involvement providing emotional reassurance

,and maintaining a calm ,predictable environment all contribute significantly to preventing the onset of delirium in vulnerable patient.

NONPHARMACOLOGICAL STRATEGIES

Prevention is the most effective strategy for reducing the morbidity, mortality, and health care costs associated with delirium. Since the cause of delirium is typically multifac-torial, delirium prevention approaches that target multiplerisk factors tend to be the most effective. The Yale Delirium Prevention Trial, a randomized controlled trial, demon-strated that a multimodal nonpharmacologic strategy is feasi-ble (87% adherence rate) and can decrease the incidence ofdelirium on a general teaching medical unit from 15% to 9%(32). The delirium prevention protocol in the study targeted six risk factors by focusing on orientation, early mobilization,medication reconciliation, sleep-wake cycle preservation,sensory impairment, and dehydration. This protocol has beenshown to be adaptable to and effective in, various othersettings, including surgical units and nursing homes (33, 34).Environmental strategies that promote sleep consolidation,such as minimizing night time noise and light exposure, alsocontribute to delirium preventionNonpharmacologic StrategiesPrevention is the most effective strategy for reducing themorbidity, mortality, and health care costs associated with delirium. Since the cause of delirium is typically multifac-torial, delirium prevention approaches that target multiplerisk factors tend to be the most effective. The Yale DeliriumPrevention Trial, a randomized controlled trial, demon-strated that a multimodal nonpharmacologic strategy is feasi-ble (87% adherence rate) and can decrease the incidence of delirium on a general teaching medical unit from 15% to 9%. The delirium prevention protocol in the study targete six risk factors by

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There is good evidence that multicomponent interventions can reduce the risk of incident delirium in at-risk hospital inpatients, and such interventions are recommended in the Scottish Intercollegiate Guidelines Network (SIGN) guidelines243. Intervention packages vary across studies but include components such as physiotherapy, reorientation, cognitive stimulation, early mobilization, non-pharmacological promotion of sleep, correction of sensory impairments, identification and treatment of underlying causes or postoperative complications, pain management, avoidance of constipation, hydration, nutrition and oxygen delivery (Box 2).

The ICU is a particularly deliriogenic environment, with patients are exposed to more than ten delirium risk factors (on average) during their critical illness264, many of which areamenable to

modification. Although many baseline risk factors, such as advanced age, are not modifiable, others can be addressed18. Vision and hearing impairment27, for example,can be improved with eyeglasses and hearing aids and the risk of delirium due to baseline cognitive impairment may be mitigated by

frequent reorientation. In addition, many acute risk factors can be avoided or reduced. Immobility can be avoided by minimizing the use of physical restraints and employing early mobility protocols; hypovolaemia, electrolyte abnormalities and infection are amenable to acute interventions; and the risk attributable to deliriogenic sedatives, such as benzodiazepines, can be avoiding by minimizing sedation in general and by using alternative medications265.

In the critical care setting, despite associated limitations, studies have shown that early physical and occupational therapy during daily breaks in sedation, in comparison to the control group of daily interruption of sedation with therapy as ordered by the primary care team (usual care), in mechanically ventilated medical ICU patients led to a shorter delirium duration in the ICU in the (median 2.0 days, interquartile range 0.0–6.0 for the intervention group versus 4.0 days, 2.0–8.0 for the control group; P = 0.02)266. Similarly,early mobilization of surgical patients in the ICU also led to improved functional status and fewer days spent in the ICU with delirium compared with the control group (standard of care) (P = 0•0161)267. These preventive strategies have been bundled into a process of care that promotes awake and spontaneously breathing mobile patients, such as the ABCDEF bundle268. Compliance with the ABCDEF bundle is associated with reduced risk of delirium on the following day (adjusted odds ratio (OR) 0.60; 95% CI 0.49–0.72) and multiple other important outcomes in critically ill patients269 (Figure 6). Further work quantifying the effectiveness and magnitude of possible benefits of such bundles needs to be confirmed in proper randomized prospective studies270.

PHARMACOLOGICAL STRATEGIES

The use of antipsychotics for the prevention of delirium remains controversial, with both positive and negative studies in various postoperative populations, critical care populations, and general hospital settings . Interpreting the positive and negative studies is challenging, however, because of their heterogeneous populations, differing measures of delirium, and varied anti-psychotic selection and dosing. In the postoperative setting, however, there have been three meta-analyses, all of which support the use of antipsychotics for reducing the incidence of delirium.

Pharmacological management is required is delirium only if it is due to drug withdrawal (alcohol/ benzodiazepines)or when there is physical aggression causing harm to patient or others. Mainly antipsychotics ad benzodiazepines are used, where benzodiazepines are used for alcohol/ benzodiazepine withdrawal delirium.

In contrast to the very small number of studies examining multidomain approaches for treatment of delirium, multiple studies have examined drugs as a standalone treatment for delirium. The delirium syndrome, rather than particular features of delirium, such as agitation or psychosis, has been the main eligibility criterion for inclusion in these studies; that is, delirium has been considered a single entity in randomized controlled trials of drug treatments. The rationale is that particular

neurochemical abnormalities, such as excessive dopaminergic transmission or reduced cholinergic transmission, are assumed to be present in delirium and thus the syndrome is responsive to drugs that act on these abnormalities.

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However, this theoretical basis is questionable given the likely involvement of multiple neurobiological mechanisms in delirium pathogenesis, as described earlier.

Dexmedetomidine, a selective a2-adrenergic receptor agonist, has been shown to reduce the incidence of delirium and ventilator-associated events while increasing ventilator-free hours. Dexmedetomidine has both analgesic and sedative properties, which allows for a reduction in the amount of deliriogenic medication exposures, including opioids and benzodiazepines. Its use can be limited by the potential for hypotension and bradycardia as well as cost.

Finally, there is a small but emerging literature to support the use of melatonin and melatonin receptor agonists (e.g.,ramelteon) for the prevention of delirium in medical, surgical, and intensive care settings. Melatonin is a hormone produced by the pineal gland that helps maintain circadian rhythms and regulate sleep, the disruption of whichi as known risk factor for delirium. Studies of serum melatonin levels demonstrate that the circadian secretion of melatonin is disrupted in patients who develop delirium.

TREATMENT

NONPHARMACOLOGICAL APPROACHES

There is only limited high-quality evidence in the literature addressing nonpharmacologic treatment of delirium, highlighted in a recent systematic overview in older patients.Both single and multicomponent protocols have undergone trials, from bright lights, earplugs, and music therapy to more comprehensive team-based approaches that can extend to family engagement (55). The evidence for multicomponent nonpharmacological interventions preventing delirium is currently much stronger than that for the treatment of already established delirium. The primary treatment of delirium is identification and management of the underlying medical etiologies,which may be highly variable within and across treatment populations, and multimodal treatment strategies to minimize the severity and duration of delirium are thus essentia.

Once delirium has developed, nonpharmacologic approaches are integral to limiting overall morbidity and mortality, including risk of long-term cognitive impairment. There is significant overlap between the nonpharmacologic strategies used in prevention and those used in treatment. These strategies target sleep-wake regulation, orientation, early mobilization, vision and hearing optimization, and nutrition and hydration. In the critical care setting, the scope of intervention expands to include daily trials of sedation reduction and spontaneous ventilation, as delineated in the ABCDEF bundle), an evidence-based guide for optimizing ICU patient recovery. The ABCDEF bundle's components include assessing, preventing, and managing pain; both spontaneous awakeningand spontaneous breathing trials; choice of analgesia and sedation; delirium assessment, management, and prevention;early mobility; and family engagement. A prospective study comparing complete ABCDEF bundle performance to proportional performance demonstrated that complete performance was associatedwith decreased risk of hospital death within 7 days.

ANTISYCHOTICS

Atypical Antipsychotics can also be used, however, evidence is still not robust and agents like Clozapine may even cause delirium. Among atypical maximum evidence is for Risperidone, Olanzapine, with emerging evidence on Quetiapine.Second-generation antipsychotics, such as Risperidone, Clozapine, Olanzapine, Quetiapine,Ziprasidone, and Aripiprazole, may be considered. But these agents are associated with increased mortality in patient of dementia.

While there are no medications approved by the U.S. Food and Drug Administration (FDA) for the treatment of delirium, antipsychotics are commonly used as a first-line pharmacologic approach to manage symptoms that threaten safety or impede care when nonpharmacologic approaches are insufficient. The efficacy of antipsychotic medications for the treatment of delirium is controversial. Although some studies suggest that the benefits of using antipsychotics outweigh the risks when used to manage specific target symptoms (e.g., agitation, paranoia, psychosis) , a recent meta-analysis found that antipsychotics demonstrated no significant effect on delirium incidence,duration, severity, length of stay, or mortality. While the current evidence regarding the effect of antipsychotics on duration of delirium is unclear, we do not yet have studies demonstrating the impact of antipsychotics on other meaningful patient measures often seen in delirium, such as emotional distress, ability to participate in care, and long-term functional outcomes.

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In the absence of conclusive data, it is recommended that antipsychotic use be limited to judicious, time-limited trials for the management of high-risk and high-distress symptoms of delirium, including agitation, paranoia, and hallucinations,which pose a safety risk to the patient or staff or impede the provision of medical care (Table 1). Antipsychotics can be helpful for treating clear psychotic symptoms associated with delirium, such as hallucinations, delusions, and paranoia. The sedative effects of antipsychotics can also be helpful for the acute management of agitation. It should be noted, however, that there is no clear evidence that antipsychotics have an impact on the core attentional or cognitive symptoms of delirium. Furthermore, it is critical that antipsychotic trials include careful monitoring for both treatment response and side effects. The selection of the antipsychotic agent may be guided by the agent's pharmacodynamic and side effect profile to maximize benefit for the unique clinical presentation. For example, patients with profound circadian disturbances and perceptual disturbances may benefit from sedating antipsychotics, such as quetiapine, dosed primarily at nighttime. Patients with hyperactive delirium characterized by rapidly escalating agitation may benefit from haloperidol, which is available in intravenous and intramuscular formulations and can be administered to patients who cannot safely receive oral medication of delirium.

TABLE 1. Antipsychotics and other medication of delirium

Medicatio n	Startin g regime n	route	Half life	Maximum daily dose	Dosing adjustment	Monitor and discontinue
haloperid ol	0.5mg to 1mg	p.o,i.v,i.m	14-30 hour s	Upper limit has been not be establishe d	No renal or hepatic adjustment required	QTc prolomgation extrapyramidal symptoms,rising liver function test values
quetiapin e	12.5mg to 25mg	p.o	6-7 hour s	800mg	No renal adjustments required;titra re slowly in hepatic impairment	QTc prolongation,extrapyrami dal symptoms
risperidon e	0.5mg	p.o,ODT	20-30 hour	8mg	Avoid in renal impairment	QTc prolongation extrapyramidal symptoms
olanzapin e	2.5 mg	p.o,ODT,i m	30 hour	20mg	No renal or hepatic adjustment required	Avoid a patient receiving parentral benzodiazepines
Ziprasidon e	10mg	p.o,im	7hou r	160mg	No renal or hepatic adjustment required	Avoid in patient with prolonged QTc and those receiving other QTc-prolonging medivations

In terms of dosing, as-needed daytime doses can be initiated, as well as either an as-needed or a standing bedtime dose,depending on symptom severity. Frequent use of as-needed doses should prompt initiation of standing doses at the lowest effective dose and frequency. If the patient demonstrates only partial response, doses may be gradually titrated upward, as long as daily maximum limits are not exceeded. As the patient begins to improve, standing daytime doses should be transitioned back to as-needed doses, reserving the standing bed time dose as the last to be transitioned back to as-needed. The three main risks associated with antipsychotic use include QTc prolongation (which increases the risk of sudden death by torsade de pointes), extrapyramidal symptoms, and increased all-cause mortality in elderly patients with dementia. While QTc prolongation is commonly observed with antipsychotic medications, the absolute increases are moest. A study performed for the FDA by Pfizer comparing the QTc interval before and after exposure to the

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maximum recommended daily doses of commonly used antipsychotic medications demonstrated QTc prolongation ranging from 4.7 ms (with haloperidol) to a maximum of 20.3 ms (with ziprasidone).

Patients receiving antipsychotics must also be monitored for extrapyramidal symptoms, as akathisia, rigidity, and dystonias may exacerbate the underlying restlessness and disorientation seen in delirium. Akathisia is most commonly observed in patients receiving high doses of first-generation antipsychotics, although the risk of developing akathisia ap-pears to be attenuated in patients receiving 4.5 mg/day or less of haloperidol, as well as those receiving second-generation antipsychotics . Patients experiencing rigidity must be monitored for the development of neuroleptic malignant syndrome, an uncommon but life-threatening condition following exposure to antipsychotic medications that is characterized by lead-pipe rigidity, elevated creatine kinase levels, fever, mental status changes, and autonomic instability.The differential diagnosis for neuroleptic malignant syndrome includes malignant catatonia and serotonin syndrome. The development of such symptoms should prompt immediate discontinuation of antipsychotics, as well as escalation of care to an ICU setting for close monitoring and supportive treatment, including aggressive volume resuscitation, electrolyte correction, and temperature regulation. In severe cases, additional treatment options would include benzodiazepines, dopaminergic agents, dantrolene, or electroconvul- sive therapy (6–10 bilateral treatments).

NON-ANTIPSYCHOTICS

Antiepelitics

Antiepileptics.Valproic acid has shown promise in case series and retrospective cohort studies for the treatment of delirium. Postulatedmechanisms of actionincludemodulation of a range of

neurotransmitters (GABA, dopamine, glutamate,acetylcholine) and increasing melatonin levels. Valproic acid may be administered orally or intravenously, and it may provide secondary benefits for delirious patients with comorbid alcohol withdrawal, history of traumatic brain injury, or mood disorder. Loading doses are often, but not uniformly, utilized. In a recent retrospective study, the me- dian dosage was 23mg/kg per dayin divided doses .While serum levels are useful to identify toxicity, to achieve effect for delirium, serum levels need not reach the range of 50–125 mg/mL

recommended for management of mood instability. Valproic acid should be avoided in patients with significant hepatic or pancreatic dysfunction, patients with active bleeding or a low platelet count, and pregnant patients. Blood counts and liver enzyme levels should be obtained beforeinitiating valproic acid and then monitored. Ammonia levels should be monitored, as hyperammonemia can contribute to hepatic encephalopathy, confusing the presentation of delirium. Once agitation has remitted, a taper schedule should be established, decreasing by 250–500 mg daily until discontinued

Melatonin.As discussed above, thereis an emergingliterature to support the role of melatonin and melatonin receptor agonists for the prevention of delirium in a variety of hospital settings, yet little is known about the efficacy of melatonin for the treatment of delirium once it has developed. Case studiesand retrospective studies indicate that ramelteon is helpful for treating delirium, particularly the hyperactive subtype. However, generalization of these results is limited by small sample size, lack of randomization, and lack of a control group. Ramelteon was remarkably well tolerated in all these studies, with no significant adverse effects. A double-blind randomized placebo-controlled trial comparing a nightly dose of 3 mg of melatonin to placebo in 56 patients who developed delirium in the setting of organophosphorus compound poisoning showed that the duration of delirium was significantly reduced in the intervention group (6 compared with 3 days; p50.001). In light of the favorable tolerability and safety profile of melatonin in a medically vulnerable population, the role of melatonin and melatonin receptor agonists for the treatment of delirium warrants additional research. In the in- terim, a low threshold is recommended for an empirical trial of melatonin or a melatonin agonist for sleep consolidation and preservation of the sleep-wake cycle in delirious patient.

Thiamine. Nutritional deficiencies, particularly of the B vitamin B1) deficiency can lead to a spectrum of mental status changes, including Wernicke's encephalopathy (triad onystagmus, ophthalmoplegia, and mental status changes), Korsakoff's syndrome (irreversible memory impairment,usually as a consequence of untreated Wernicke's encephalopathy), and delirium. Although the most common cause of thiamine deficiency is alcoholism, a variety of conditions that result in malnutrition, including conditions that result in poor feeding, such as anorexia nervosa and orofacial cancers; conditions that limit absorption, such as gastric bypass surgery, gastric cancer, and colon cancer; and

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hyperemesis gravidarum, can cause thiamine deficiency (76). If thiamine deficiency is suspected, patients should be treated with 250 mg/day of thiamine intravenously for 3 to 5 days.

DISCUSSION

This systematic review suggests that there is a limited research on delirium from India. However, a good thing is that in recent times, delirium has received more researchattention, compared with about a decade back. This review further suggests that although psychiatrists have performed the majority of research in the area of delirium, in the recent years, other specialists too, especially, anesthetists have alsoshown interest in delirium and some of the studies havefocused on incidence/prevalence of delirium in ICUs, posoperative delirium,32,33 and emergent delirium Another important aspect that is evident from the available literture is that most of the studies have been based on standard evaluation instruments.

Studies that have evaluated the factor structure of symptoms of delirium suggest that symptoms cluster on to two tothree factors. In the majority of these studies, various conitive symptoms load onto the same factor, the motoric and psychotic symptoms load together, and the third factor consists of language and thought process abnormalities. In geeral, data suggest that the motoric and psychotic symptomsmore consistently load on to the same factor across different studies. These findings are also supported by the existing literature from other parts of the world.

suggest that most of the ICU clinicians do not assess patients for delirium on regular basis and consider the prevalence rates of delirium, especiallyamong those patients on mechanical ventilation to be lowerthan what is reported in the literature Data also suggest that there is a low concordance rate between the diagnosis made by the psychiatrist and other clinician and most of the ICU clinicians are aware of the importance of early mobilization of ICU patients but are not able to practice the same due to lack of support staff and safety concerns. A study which focused on improving the knowledge of the nursesshowed that it was associated with significant improvementin the knowledge and practice of nurses toward deliriumTaken together, it can be said that the incidence and prevlence of delirium in various treatment settings in India arehigh and comparable to the rest of the world. However, many times, clinicians do not focus on this entity due to lack ofawareness and do not screen patients for delirium regularly.Accordingly, it can be said that there is a need to improve theknowledge base, awareness, and change in clinical practice toidentify delirium and manage the same in the Indian contextto reduce the negative impact of the same on the patientsand their caregivers. In India, ICU facilities and ventilators arealso a scarcity. Accordingly, prevention and early identifiction of delirium can reduce the duration of ICU stay and the number of days on ventilators. This would help more patientsto utilize these scarce resources. Mental health professionalsneed to play an important role in improving the awareness of other clinicians about delirium. Mental health profession can also train the clinicians and nurses to identify the patients who are at higher risk of delirium, screen patients, if not allinpatients, then patients at high risk for delirium.

II. CONCLUSION

To conclude, this systematic review suggests that although there is limited research on delirium from India, in recent years there is an increase in research output concerning delirium. Availabledata suggest that most of the studies have focused on the incdence, prevalence, and symptom profile of patients of delirium.

Delirium is an acute confusional state which affects the global outcome and prognosis of severely ill patients.It substantially surges health-care utilization and costs; therefore, prevention, early recognition and effective treatment of delirium are indispensable in this condition. Swift screening and proactive management is the cornerstone of reducing the incident and prevalence. Along with pharmacological, non-pharmacological interventions are imperative.

Given the high morbidity and mortality associated withdelirium, ongoing efforts to develop and apply proactive interventions to prevent or reduce the severity and duration of delirium are essential. It is crucial to remember that the primary treatment of delirium is identification and management of the underlying medical etiologies, which may be highly variable within and across treatment populations. This adds complexity to research and application of findings across subgroups of populations, even among the geriatric populations who have been the focus of much of the delirium research to date. Safety and symptom relief, including management of the attendant risk of agitation with minimal use of restraints, are important treatment goals. Further research on additional pharmacologic and nonpharmacologic

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interventions is urgently needed. Meanwhile, we can provide our patients with careful application of the tools currently available to optimize outcomes.

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