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Volume 1

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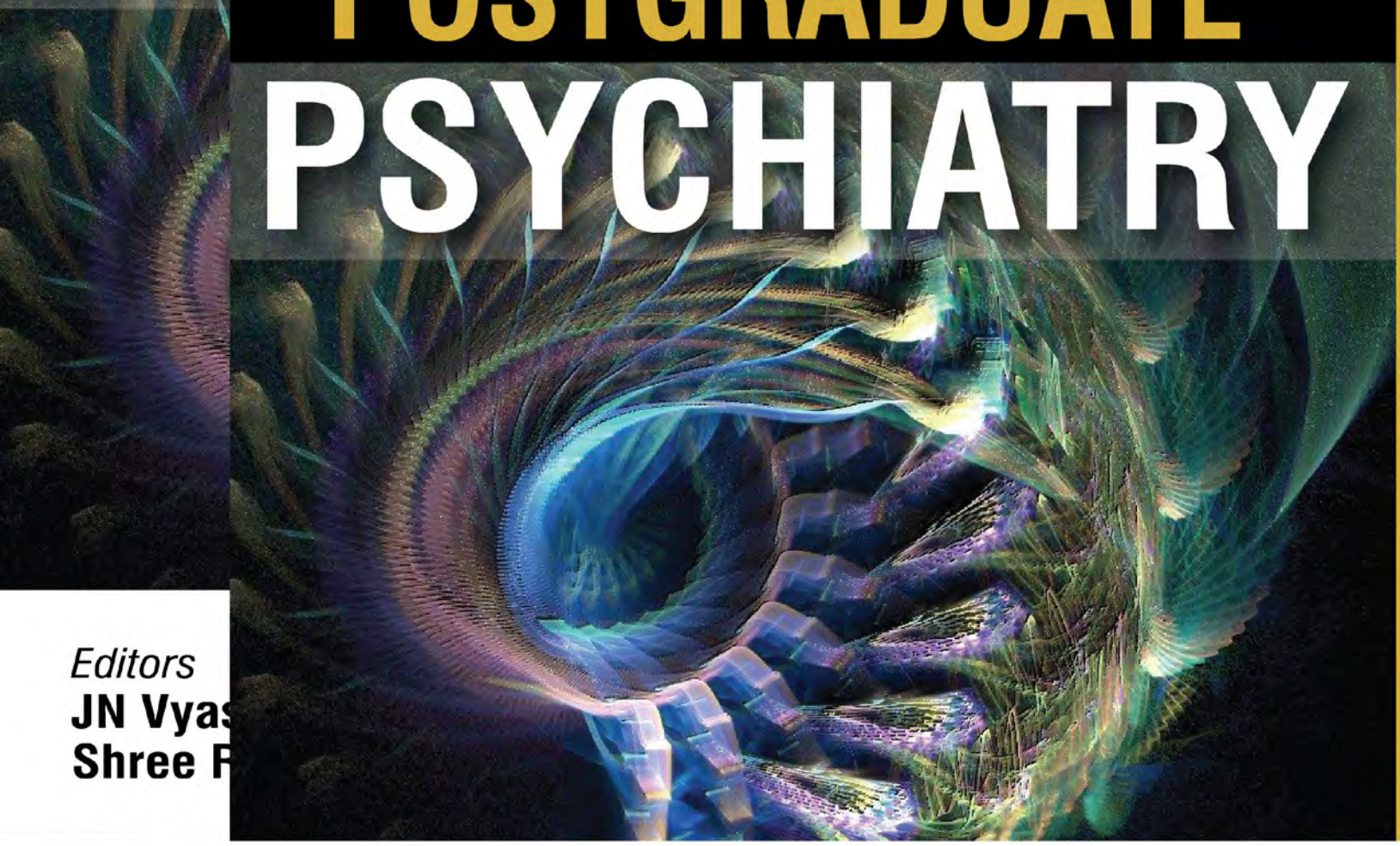
Textbook of POSTGRADUATE PSYCHIATRY



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CHAPTER

4 Organic Mental Disorders

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INTRODUCTION

Organic mental disorders are group of conditions caused directly by abnormalities of the brain structures or by alterations of brain neurochemistry or neurophysiology. They are traditionally called as organic mental disorders or organic brain disorders to distinguish them from the functional disorders such as schizophrenia or depression. This terminology "functional" and "organic" has been removed from most of the classificatory systems since neurodiagnostics and neurobiological evidences have found that every psychiatric condition had a biological component. With the advent of newer technologies and new evidences; newer etiologies are discovered and newer medications are manufactured. In a country like India, where the population is increasing and there are significant improvements in economic and health care, the quantum of dementia and other cognitive disorders are increasing. Global prevalence of dementia in 2010 was estimated to be 35.6 million and in India it is 3.7 million. It is projected to double every 20 years, to 65.7 million in 2030 and 115.4 million in 2050 and has been attributed to increase in the numbers of people with dementia in LIMC. Hence, it is important to identify these disorders and consider early management to prevent further complications and disability.

HISTORY

In the 5th century BC, the father of medicine Hippocrates described for the first time the conditions of "phrenitis" and "paraphrenitis" which in the present day is related to delirium. He referred to a term called as lethargus to describe inertia and a dulling of the senses and suggested that lethargus can turn into phrenitis and back again into lethargus. The term "delirium" was coined by Aulus Cornelius Celsus (greatest Roman medical writers) to describe mental disorders. "Delirare" translated from Latin means "to be off the track" or "to go wrong" and was used to define general mental illness. Organic mental disorders were divided into acute and chronic type by Aretaeus of Cappadocia. Later Galen introduced the concept of primary and secondary brain diseases. Also the concept of hyper- and hypoactive forms of delirium have been described by Galen. Karl Bonhoeffer (1909) made a significant contribution to the concept of organic mental disorders. He stated that the brain reacts to exogenic irritants in a nonspecific manner and that the number of such reactions is limited and depends on the constitution of a person. Based on this, authors have suggested that completely different etiological phenomena may evoke similar psychotic symptoms. Bonhoeffer had suggested five acute brain reactions which include delirium, amentia, a twilight state (or epileptic type), excitement and acute hallucinosis. In 1913, a German, psychiatrist and philosopher Karl Jaspers gave more clarity to mental illness and was the first who described consciousness disorder symptoms. Eugen Bleuler gave a detailed definition of organic brain syndrome and Manfred Bleuler described symptoms of organic brain disorder. The modern concept of delirium was provided by an American psychiatrist Zbigniew J Lipowski in 1980. He sub-classified delirium into increased, reduced, or mixed motor activity during delirium. Philippe Pinel was credited to give detailed description of dementia. There has been a varied course in the evolution of the concept of organic mental disorders. With the enormous development of neuroimaging, genetic studies, biomarkers and understanding of these disorders, the conceptualization of organic mental disorders may change.

DEFINITIONS

Confusion

It refers to clinical signs and symptoms where patient is unable to think with clarity and coherence. It can manifest in both psychiatric and organic conditions. Cognitive impairment is preferred than confusion and is one of the earliest symptom of insult to the brain. In acute conditions the confusion is due to impairment of consciousness, however in chronic condition it is due to disruption of thought processes due to

structural brain damage. Sometimes confusion can present without any structural damage in psychiatric conditions such as heightened emotional reactions and early stages of psychotic conditions.

Clouding of Consciousness

Clouding of consciousness is also known as brain fog or mental fog. There is mild global impairment of cognitive functions in addition to decreased awareness of environment. It is described as an abnormality in the "regulation" of the "overall level of consciousness that is mild and less severe than a delirium. It is the mildest form of impairment of consciousness. The sufferer experiences a subjective sensation of mental clouding described as feeling "foggy". This may affect performance on virtually any cognitive task. According to Jasper, they may have additional phenomenon such as vivid affects, hallucinations and fantasies.

Minimally Conscious State

The criterion for minimally conscious state (MCS) was proposed by Aspen group to categorize patients who are not in a vegetative state but are unable to communicate consistently. It is a condition where the patients show limited but clear evidence of awareness of themself or their environment, on a reproducible or sustained basis, by at least one of the following behaviors: following simple commands, gestural or verbal yes/no response (regardless of accuracy), intelligible speech, purposeful behavior (including movements or affective behavior that take place in relation to stimuli in the environment and are not due to reflexive activity). According to[1] emergence from the minimally conscious state is defined by the ability to communicate or use objects functionally and they further improvement occurs better than in vegetative states.[2,3]

Locked-in Syndrome

Locked-in syndrome is defined by sustained eye opening, awareness of the environment, aphonia or hypophonia, quadriplegia or quadriparesis, and vertical or lateral eye movement or blinking of the upper eyelid to signal yes/no responses. The main method of communication is by eyes or eyelid movements. The term was introduced by Plum and Posner to describe the quadriplegia and anarthria resulting from the disruption of corticospinal and corticobulbar pathways, respectively.[4,5]

Vegetative State

The patients in this state are awake but are unaware of themselves or their environment. The term vegetative is defined as "an organic body capable of growth and development but devoid of sensation and thought". There are two terms related to vegetative states. "Persistent vegetative state" which is a reversible state and defined as a vegetative state remaining 1 month after acute traumatic or non-traumatic brain damage and "permanent vegetative state" which is irreversible and present if the vegetative state is prolonged for 3 months after nontraumatic brain damage or 12 months after traumatic injury.[6-9]

Twilight States

It is one of the forms of exogenous reaction to pathogenic factors according to Bonhoeffer's. It is a condition where there is an interruption in the continuity of consciousness and it has an abrupt onset and termination. There is clouding and narrowing of consciousness, but patients may be able to perform certain activities like driving, walking, etc. and they have amnesia for the event. It usually occurs in patients with epilepsy, head injury, alcoholism, dissociative disorders, etc. It may lead to violent behaviors and such patients who are vulnerable to this phenomenon may have pre-existing organic brain pathology. The duration may last from few hours to weeks. Oneiroid state is a similar clinical phenomenon where there is narrowing of consciousness with multiple scenic hallucinations but in this condition they remember the events compared to twilight states. This condition is perceived as strange and dream-like and has been found in patients who are very dependent on others and schizophrenic patients. It is used to describe hysterical manifestations in addition to acute organic reactions.

Coma

It is a state of unresponsiveness characterized by the absence of arousal and consciousness. In this condition the patient lies with the eyes closed, cannot be aroused, and has no awareness of self and surroundings. They will not produce spontaneous periods of wakefulness or eye-opening with significant stimulation. In order to distinguish it from syncope, concussion, or other states of transient unconsciousness, coma must persist for at least one hour. Patient who survive the coma gradually improve within 2 to 4 weeks but the recovery may progress to vegetative or minimally conscious state before a complete consciousness state is achieved.[4]

Delirium

It is a neuropsychiatric syndrome characterized by disturbances in the baseline cognitions such as consciousness, orientation, memory, perception, thought and behavior; not explained by pre-existing or evolving neurocognitive disorder. It is a reversible condition and has an acute onset, fluctuating course within the day and worsening in the evening or night due to decreased stimuli. The etiology could

be substance withdrawal, medication use, exposure to toxins or abnormalities of metabolic parameters. It is associated with high morbidity and mortality. They manifest with features of irritability, anger, euphoria, aggression, sleep wake disturbances, screaming, moaning and sometimes they may be apathetic and silent.

Dementia

Dementia is a syndrome due to disease of the brain—usually of a chronic or progressive nature—in which there is disturbance of multiple higher cortical functions, including memory, thinking, orientation, comprehension, calculation, learning capacity, language, and judgement. Consciousness is not clouded. The impairments of cognitive function are commonly accompanied, and occasionally preceded, by deterioration in emotional control, social behavior, or motivation. This syndrome occurs in a large number of conditions primarily or secondarily affecting the brain. The four most common subtypes in order of frequency are Alzheimer's disease, vascular dementia (VaD), dementia with Lewy bodies (DLB), and frontotemporal dementia (FTD).[10,11]

CLASSIFICATION

In DSM-4, the term "organic mental disorder" was removed for the first time. The disorders were divided into three groups: (1) delirium, dementia, amnestic disorders, and other cognitive disorder (2) mental disorders due to general medical conditions and (3) substance-related disorders. Previous classifications such as organic mood or organic anxiety disorders were subsumed under second category as mentioned above.[12]

In the DSM-5,[13] dementia and amnestic disorders are subsumed under neurocognitive disorders and delirium is retained. The delirium has further specifiers such as acute or persistent course and type of delirium (hyperactive, hypoactive and mixed). The neurocognitive disorders (NCD) include group of disorders in which the primary clinical deficit is in cognitive functions, which are acquired rather than developmental. Even though the psychiatric disorders also have cognitive dysfunctions, only those disorders whose core features are cognitive are included in this category. The NCD is further divided into major and minor. The criteria for the major NCD is given below and has specifiers of the etiological factors and further specifiers such as severity and association of behavioral disturbances. The difference between major and minor NCD are the time course, severity of cognitive impairment, characteristic domains affected and associated symptoms. In DSM-5, new separate criterias are presented for frontotemporal, Lewy body, traumatic brain injury, substance/medication, HIV infection, prion disease, Parkinson's disease, Huntington's disease and other medical conditions.

- Evidence of significant cognitive decline from a previous level of performance in one or more area of cognitive domains (complex attention, executive function, learning and memory, language, perceptual-motor or social cognition) based on:
  - Concern of the individual, a knowledgeable informant or the clinician that there has been a significant decline in cognitive function
  - Substantial impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.
- The cognitive deficits interfere with independence in everyday activities.
- The cognitive deficits do not occur exclusively in the context of a delirium.
- The cognitive deficits are not better explained by another mental disorder (e.g. major depressive disorder, schizophrenia).

In India, the International Classification of Diseases (ICD)[14] system of classification is used commonly. It has similar flaws such as the preliminary editions of DSM and retains the psychotic and nonpsychotic dichotomy. In the ICD-10 version, the term "organic mental disorder" continues to find its place and has introduced a new term called "mild cognitive disorder" which has been associated with acquired immunodeficiency syndrome (AIDS).

With the advent of newer evidences, newer classificatory systems would evolve and lead to better understanding of the psychiatric conditions. However, we should not forget the value of classical teaching of phenomenology because even with advanced neuroimaging diagnostic abilities of these newer methods are only complimentary and not confirmatory.

DELIRIUM

Delirium is one of the most common and serious medical illness. Many terminologies have been used to describe it such as "acute confusional state", acute brain syndrome or toxic metabolic syndrome; however, the "acute confusional state" is accepted as the synonym for delirium. Delirium has been derived from the word *delirare* which means to become "crazy or to rave". This term was first used by Celsus in 1st AD. It has been documented and described in the literature for more than 2000 years. Hippocrates described the subtypes of delirium hyperactive as phrenitis and hypoactive as lethargus. The outcome of delirium is usually adverse due to poor diagnosis and fluctuating nature of the syndrome.[15]

Epidemiology

The prevalence of delirium in the community is lower and is estimated to be 1 to 2%. However, the rates are higher in

hospitalized patients, it has varied from 10 to 31% during the time of admission and an incidence of 3 to 29% during the hospitalization. There have been significantly increased prevalence rates in different settings in the hospitals. In the intensive care units a prevalence of 80%, in palliative care units the rates are 85% and in surgical settings an incidence of 10 to 70% after surgery.[16]Also, there has been higher rates are noticed in especially in patients undergoing cardiothoracic surgery, emergency orthopedic procedures (repair of a hip fracture), vascular surgery, or cataract removal.[17] In India, the prevalence of delirium has varied from 3 to 27% in hospitalized patients (>65 years) and an incidence of 4.3% in postcataractomy patients in a year. Grover et al. found a prevalence of 30 to 38% for all ages and 48 % in patients more than 60 years.[18,19]

Clinical Types

Three clinical forms of delirium have been described, they include hyperactive, hypoactive, and mixed, based on psychomotor behavior. Even though ICD-10 or DSM-IV do not recognize this classification, several studies have confirmed the existence. The *hyperactive subtype* is most easily identified and manifests as increased psychomotor activity, hypervigilance, restlessness, agitation, aggression, mood lability, disruptive behavior hallucinations and delusions.[20] However, the *hypoactive form* is the most common type in the elderly and manifests as withdrawn behavior, drowsiness, lethargy, apathy, confusion and decreased psychomotor. They have reduced spontaneity and answer slowly to questions and without. In a study by Khurana et al, they found a high prevalence of 65% of hypoactive delirium elderly delirious patients who were hospitalized. This subtype is usually difficult to recognize and can be easily missed.[18] *Mixed subtype* is the most common subtype and consists of features of both the types mentioned above. *Persistent delirium* defined as delirium present on admission and at the time of discharge or beyond.[15] *Excited delirium syndrome* is a syndrome with uncertain, likely multiple, etiologies. It is characterized by delirium, agitation, acidosis, and hyperadrenergic autonomic dysfunction, typically in the setting of acute-on-chronic drug abuse or serious mental illness.[21]

Subsyndromal Delirium

Recently a new concept of subsyndromal deliruim has emerged. It is defined as the presence of one or more core diagnostic symptoms (inattention, altered level of consciousness, disorientation and perceptual disturbances) that do not meet the full criteria for delirium and may not progress to full blown delirium.[22] In intensive care units, the prevalence rates of 30–50% have been reported and the incidence rate was 5.2 per 100 person-weeks of follow-up.[23] The risk factors for this type are also similar to the delirium. This variety needs detailed attention and early identification and treatment. Subsyndromal delirium occurs in 21 to 76% of hospitalized elderly people in long-term care elderly residents, with dementia, the occurrence was 48.4 or 50.3%, depending on the criteria used. A recent cohort study has found that 68 of the 104 residents had incident subsyndromal delirium during 6 months of observation. The risk factors for subsyndromal delirium are similar to those for classical overt *delirium*.[24]

Clinical Features

Delirium has been defined as "the rapid onset of symptoms that tend to fluctuate even during the same day with an altered level of consciousness, global disturbance of cognition or perceptual abnormalities and evidence of physical cause, substance intoxication or withdrawal, or multiple etiologies" according to APA.[25] The diagnosis is based on detailed history, physical examination, mental state examination and laboratory investigations. The central features of delirium are sudden or acute onset, fluctuating course and inattention. The symptom fluctuations are unpredictable, intermittent, and often worse at night.

Disturbance in consciousness: It is one of the earliest symptoms and fluctuates frequently. The disturbances may be from mild drowsiness to coma in advanced cases. However, in hyperactive delirium as such in alcohol or sedative withdrawal, hypervigilance and hyperactivity may be noticed. The disturbance is seen mainly in the evening when environmental stimulation is at its lowest. Patients also present with disorientation to time first and then to place. In terms of memory, they have deficits in short-term memory and retrieval of information.

Disturbance in psychomotor activity: It is also another important clinical feature of delirium it may be increased or decreased.

Disturbances in thought also present with disorganized thinking, which can present as incoherent speech, irrelevant conversation or illogical flow of ideas. They have difficult in taking appropriate decisions or do simple tasks. The patients can have poor judgment and insight. They can also present with delusions in 30% of patients and they are predominantly paranoid or persecutory in nature.

Disturbances in perception also present with perceptual disturbances. They may include illusions, misinterpretations and hallucinations (visual). The content of the hallucinations may range from simple colors, lines, or shapes to dangerous animals or bizarre images. They also present with emotional disturbances, such as anxiety, fear, irritability, anger, depression, and euphoria.

Other symptoms found delirium include sleep-wake cycle disturbance, characterized by an excessive daytime sleepiness with insomnia at night, sleep fragmentation, reduction of sleep or complete sleep-cycle reversal and disturbance of circadian rhythm. Sundowning syndrome seen in delirium and is characterized by worsening of disruptive behavior in the late afternoon or evening. This aspect has been attributed to sleep fragmentation, disturbance of circadian rhythm, fatigue and reduced sensory input toward the evening.

In children and adolescents, the most common symptoms included sleep-wake cycle disturbance and impaired orientation. The other symptoms which they presented were impaired attention (89.5%), impaired short-term memory (84.2%), agitation (68.4%), and lability of affect (60.5%). However, delusions and hallucinations were only in few. The pathology detected in them includes infection of various types, followed by neoplasms.[18]

Pathophysiology

The exact pathophysiology of delirium is not clear. However various theories have been proposed. Based on the current understanding and evidence neurotransmission disruption has been proposed. In the neurotransmitter hypothesis, cholinergic deficits and dopaminergic excess has been implicated in the development of delirium.[26] There has been a strong evidence for the role of cholinergic system in the pathophysiology of delirium based on the evidence that the cholinergic system has an important role in cognition and attention and anticholinergic properties may precipitate delirium in vulnerable individuals based on this hypothesis a surrogate marker called "serum anticholinergic activity" (SAA) has been developed to detect anticholinergic activity and there is a relationship between SAA levels and development of delirium.[27]

Excessive dopamine has also been implicated in the pathophysiology of delirium. It has been found that the dopamine plays a significant role in the motor activity and cognitive functions and based on the evidence that dopaminergic drugs cause delirium in Parkinson's disease. Also, excess dopamine has been associated with emergence of psychotic symptoms. All these evidences reinforce the fact that excessive dopamine could have a role in delirium.[28]

Researchers have found evidence for increased production of cytokines after trauma, infection or postoperative which may induce delirium in vulnerable patients. A recent review of older adults suggested that increase in cytokines lead to development of cognitive dysfunction which is observed in delirium.[29] These evidences suggest an inflammatory hypothesis for the pathophysiology of delirium. Other hypothesis proposed are hypercortisolemia, medication induced (valproate) and lower plasma cholinesterases.[30,31]

Risk Factors

The identification of risk factors are important for the prevention and early management of delirium. The etiology of delirium could be a single factor (as in alcohol withdrawal) or multifactorial. Researchers have categorized the risk factors into predisposing (vulnerability to develop delirium) and precipitating (acute factors triggering delirium). The predisposing factors have been found to contribute more to the development of delirium than precipitating factors. The predisposing factors are advanced age, pre-existing dementia (second most common), male gender, pre-existing depression, visual and hearing impairment, functional dependence, dehydration and malnutrition, poly pharmacy (mainly psychoactive drugs), alcohol abuse and coexistence of multiple, and severe medical conditions. The most common precipitating factors are intercurrent illnesses (e.g. infections), iatrogenic complications, metabolic derangements, primary neurological conditions (e.g. acute stroke), surgery, drugs (benzodiazepines, narcotic analgesics, drugs with anticholinergic effects and uncontrolled pain have also been associated with the development of delirium.[17]

Prognosis

Delirium is usually reversible within 10 to 12 days but can vary in some cases and persist for more than 2 months. In a study, they found that one-third of patients continued to remain in delirium for almost 6 months. Delirium has been associated with increased hospital stay, cognitive decline, functional decline, institutionalization and mortality.[32] The incident delirium was found to accelerates the trajectory of cognitive decline in hospitalized elderly patients with Alzheimer's disease.

Diagnosis

The diagnosis of delirium is purely clinical and there are no specific diagnostic tests. It is made based on clinical history, observation and comprehensive physical and a detailed mental state examination. History obtained from family members, nursing staff and care givers may be very valuable. Even thogh there are around 11 scales to identify delirium the confusion assessment method (CAM) has the most evidence for its use as a bedside tool. However, the minimental status examination (MMSE) was found to have the least useful test for identifying delirium.[33] An attempt should be made by the physician to identify the underlying cause for the delirium. Some of the important investigations to be considered include complete blood count, blood urea, creatinine levels, electrolytes, blood sugar, C-reactive protein, liver function, thyroid function and plasma ammonia. EEG and imaging may be warranted in cases where the physicians are suspecting a intracranial pathology.

DSM-5 Diagnostic Criteria for Delirium

a. A disturbance in attention (i.e. reduced ability to direct, focus, sustain, and shift attention) and awareness (reduced orientation to the environment).
b. The disturbance develops over a short period of time (usually hours to a few days), represents a change from baseline attention and awareness, and tends to fluctuate in severity during the course of a day.
c. An additional disturbance in cognition (e.g. memory deficit, disorientation, language, visuospatial ability, or perception).
d. The disturbances in criteria a and c are not better explained by another pre-existing, established, or evolving neurocognitive disorder and do not occur in the context of a severely reduced level of arousal, such as coma.
e. There is evidence from the history, physical examination, or laboratory findings that the disturbance is a direct physiological consequence of another medical condition, substance intoxication or withdrawal (i.e. due to a drug of abuse or to a medication), or exposure to a toxin, or is due to multiple etiologies.

Specify whether

Substance intoxication delirium: This diagnosis should be made instead of substance intoxication when the symptoms in criteria a and c predominate in the clinical picture and when they are sufficiently severe to warrant clinical attention.

PREVENTION

Studies report that one-third of delirium episodes could be prevented and prevention as a cost-effective strategy.[34,35] In the Yale Delirium Prevention Trial, the intervention was focussed towards minimizing 6 risk factors in elderly patients (≥70 years of age) who were at risk for developing delirium. Some of the interventions used included orientation activities for the cognitively impaired, early mobilization, preventing sleep deprivation, minimizing the use of psychoactive drugs, use of eyeglasses and hearing aids, and treating volume depletion. In the intervention group, the delirium incidence had reduced to 9.9% compared with 15% in the usual care. In another study, patients with post hip fractures who were randomized to either standard care versus the addition of a geriatrics consultation preoperatively or immediately after hip repair, providing recommendations based on a structured protocol. They found that the incidence of delirium reduced to 32% in the geriatrics consultation group versus 50% in the standard care group.[36,37]

Regarding the role of medication in the prevention of delirium, a Cochrane review found 6 RCT in hospitalized surgical patients and found that low-dose haloperidol prophylaxis was effective in reducing the severity, duration of delirium and reduced the length of hospital stay in hip surgery patients, but it did not prevent delirium occurrence. Another review by Campbell et al. evaluated 9 studies and found that use of a single-dose risperidone after cardiac surgery decreased delirium incidence compared to placebo.[38-40]

Treatment

Delirium is a medical emergency and an attempt should be made to identify the underlying cause. The first line of treatment is nonpharmacological intervention and this involves providing supportive care, preventing complications and treating behavioral problems. Supportive care involves close monitoring and care by nursing staff. They need to take care of the patient's airway, adequate nutrition, correction and prevention of dehydration vital sign monitoring, attention to oral intake, prevention of aspiration, encourage of mobility, and ensuring a good sleep pattern. It is also important to involve the family and caregivers. They can assist the health professionals in facilitating effective communication, reorientating the patient, calm, protect, and support older people.[34,35] Physical restraint has to use mainly to control violent behavior or to prevent the removal of important devices, such as endotracheal tubes.[34]

Environmental manipulation such as ensuring that there is a big clock and a calendar in the room; verbal reminders of the time, day, and place frequently; avoiding change in paramedical/nursing staff changes; transferring the patient to an isolated room, if possible; obtaining familiar possessions from home (e.g. family picture); avoiding sensory deprivation (e.g. windowless room) or sensory overload (e.g. too much noise); reducing sensory impairment (including vision and hearing loss) by the use of corrective devices.

The pharmacological interventions should be considered mainly in the management of behavioral disturbances. Haloperidol or olanzapine, lower doses and for a short period are recommended, however, it is better avoided in Parkinson's disease or DLB patients.[35] In alcohol and benzodiazepine withdrawal, or neuroleptic malignant syndrome, benzodiazepines have been recommended. Regarding the use of cholinesterase inhibitors no specific evidence from controlled trials that donepezil or rivastigmine are effective in the treatment of this medical condition.[41]

DEGENERATIVE DEMENTIAS

DEMENTIA

Aging is a worldwide phenomenon. Health facilities in the past few years have increased the life expectancy and are providing better health care. However, it has increased the burden of noncommunicable disorders such as dementia and psychiatric illnesses. The word dementia is derived from the Latin word "dementatus" meaning out of one's mind. Dementia is not part of normal aging but a syndrome due to disease of the brain, usually of a chronic or progressive nature, in which there is an intact consciousness but there is disturbance of multiple higher cortical functions, including memory, thinking, orientation, comprehension, calculation, learning capacity, language, and judgment.[42]

Epidemiology

There have been improvements in the data related to dementia and Alzheimer's Disease International (ADI) in 2005, commissioned a panel of experts to review all available epidemiological data and reach a consensus estimate of prevalence in each of 14 world regions.[43] They estimated that the prevalence of dementia in people aged 60 years and above was 24.3 million in 2001, 60% were living in low middle income countries (LMIC). The global prevalence of dementia in 2010 was estimated to be 35.6 million people worldwide were living. It is projected to double every 20 years, to 65.7 million in 2030 and 115.4 million in 2050 and has been attributed to increase in the numbers of people with dementia in LIMC.[44]

Late Onset Dementia

Among the global burden of disease (GBD) study 2010, Western Europe had highest prevalence of dementia (7.0 million), followed by East Asia (5.5 million), South Asia (4.5 million) and North America (4.4 million). In terms of countries with a prevalence of one million or more, China had 5.4 million, USA (3.9 million), India (3.7 million), Japan (2.5 million), Germany (1.5 million), Russia (1.2 million), France (1.1 million), Italy (1.1 million) and Brazil (1.0 million).

Indian Scenario

In Indian, the reporting of prevalence rates of dementia has improved significantly. Based on the quality of data and the methodology, using survey diagnosis or clinical diagnosis of DSM 4 or ICD 10 the estimated prevalence of dementia in people above 60 years ranged from 0.6% to 3.5% in rural areas and 0.9–4.8% in urban areas. It was estimated that 3.7 million Indian people aged over 60 have dementia (2.1 million women and 1.5 million men) in 2010. However, these rates are higher than previously reported estimates by Delphi consensus.[43] In the Indian states, Trivandrum (Kerala) and Thirupur (Tamil Nadu) had highest rates, where as Ballabgarh and Vellore have the lowest based on an study involving 42,000 older people studied in eight centres (5 urban and 4 rural areas).[45-49]

Young Onset Dementia

It is defined as typically as onset of dementia before the age of 65 years. So far only four major studies have attempted to estimate the prevalence in young onset dementia (YoD), they include European Collaboration on Dementia group (EUROCODE), two registry-based studies from the United Kingdom and The Rotterdam study (1995); but the expert did not attempt a meta-analysis in view of scarcity of data and variability of the estimates. Using the Delphi consensus, estimates were attempted with the data of two United Kingdom studies, one carried out in Cambridgeshire and four London boroughs. They found prevalence of persons aged 45–64 of about 120/100 000 (London) and 101/100 000 (Cambridgeshire) for males and 77/100 000 (London) and 61/100 000 (Cambridgeshire) for females. Also the expert's consensus was that the prevalence increased exponentially with increasing age, roughly doubling every five years from 9/100 000 at age 30 to 156/100 000 at age 60–64 years. They also reported that 68% of all YoD were aged 55 and above and males predominated over females (gender ratio of 1.7 to 1) in India, prevalence of YOD were as low as those seen in high-income population-based surveys: 328/100 000 (60–64 years) in Kerala *(24)*, 249/100 000 in Ballabgarh (55–64 years), and 63/100 000 (50–59 years) and 280/100 000 (60–64 years) in Mumbai.[50-52]

Clinical Features

Cognitive Impairment

The memory is significantly affected in persons with dementia (PwD). In early stages of dementia the short-term memory is impaired. It is manifested by forgetting recent interactions, events and conversations, repetitive questions related to recent events (even they forget that they have eaten their meals and demand to be given food), they forget important dates and appointments, they misplace commonly used objects and, lose money or costly items. They have difficulty learning new information or new skills. In later aspect of the disease long-term memory starts becoming impaired and they will have difficulty recall their past memories and will forget previously highly learned material. However, they are able to preserve the procedural memory for longer time.

Apraxia

Apraxia is the inability to carry out motor activities, even if the motor functions are normal. The apraxia presents as difficulty in using familiar objects (i.e. cooking, washing) or skills which have been previously acquired (i.e. using machinery, cycling). As the disease progresses they have difficulties in dressing, bathing, or feeding which affect their activities of daily living.

Agnosia

Agnosia is defined as the failure to recognize or identify objects in spite of having normal perception and sensory functions. It presents as difficulty in recognize familiar objects, familiar people and in end stages one's face may not be recognized. Visuospatial dysfunction manifests as difficulty navigating around their familiar surroundings, they may be confused and lose their way and sometimes wander away. There is also significant executive dysfunction manifested by problems in planning, organizing, sequencing, and abstracting. They

have difficulty performing complex tasks or activities such as managing money, medications and hence lead to poor judgment.

Behavioral Disturbance

In most of the dementias, behavioral disturbance is common and manifests as disinhibition, agitation, aggressive behavior, uncooperative behavior, and wandering. There is significant agitation which is worse in the evenings or night as the sensory stimuli reduces.

Personality Changes

There have been different types of personality changes manifestations in dementia. It is very common in Frontotemporal dementia. They often presents with disinhibition and impulsivity. The PwD present as inappropriate jokes, overfamiliarity with strangers, social norms are violated, sexual disinhibition or behaviors and impulsive buying. Also, depending on the dementia and involvement of brain structures apathy syndrome with a motivation and withdrawal Alzheimer's disease and vascular dementia. Exaggerated of pre-existing personality traits may occur in some cases. Also, personality change was particularly associated with severity of cognitive impairment, longer duration of illness, and neurological signs. The findings emphasize the biological basis of personality changes in dementia. Also associated with it, PwD have disturbances of self-awareness.[53]

Psychiatric Manifestations

About 10% of the PwD may have mood symptoms and depression is the common presentation (10–25%) especially early phase.[54] Depressive symptoms are associated with greater temporal lobe and cingulate gyrus hypometabolism. Suicidal attempts may also occur in them, especially in those with milder cognitive impairment and those who have insight about the disease. Anxiety disorders are also very common and reported to be more than 50%. It manifests as fear of being alone, have "catastrophic reactions" when cognitively challenged and they would expect the family members to be with them always. Manic is less common and the estimated prevalence is less than 5%.[55] Psychotic symptoms are also common and occur in the middle phase of dementia. They have delusions that their belongings have been stolen and accuse neighbors or relatives. These paranoia may be associated with their primary cognitive dysfunctions. In the latter aspects, hallucinations become were prominent and are associated with delusions. In fact, visual type are the most common compared to auditory or olfactory or tactile. They see small animals or people and they are complex in nature. They also have misidentification syndromes, commonly associated with their family members.

Normal Aging vs Dementia

There are no clear cut criteria to distinguish the both. Some authors suggest that it is a continuum. Normal aging (NA) occurs due to physiological processes over a person's lifetime. Some persons can have successful aging without cognitive dysfunction while others may develop mild dysfunction which may not be pathological. The "threshold hypothesis" of normal aging, suggests that the cognitive reserve slowly diminishes and a critical level may be reached and vice versa someone may start with a low reserve and more easily reach the threshold for the clinical manifestation of dementia as they age. In normal aging, they complain of memory loss often and provide considerable details on how they forgot but PwD may complain of memory problems only if specifically asked for and not able recall instances. In normal aging, they are more concerned about alleged forgetfulness than are close family members but in PwD the close family members much more concerned. The recent memory for important events, affairs, conversations not impaired in NA whereas in PwD have significant cognitive decline on testing. NA have occasional word-finding difficulties, but PwD have frequent word-finding pauses and substitutions. NA are able to operate common appliances even if not willing to learn how to operate new devices but PwD are unable to operate common appliances; unable to learn to operate even simple new appliances. PwD may get lost easily due to cognitive dysfunction but NA may not. PwD may exhibits loss of interest in social activities and inappropriate behaviors but NA may not.[56]

In view of the above, many terms were used such as "benign senescent forgetfulness" "age associated mental impairment" (AAMI) and "mild cognitive impairment (MCI)". The criteria for AAMI, which includes 50 years of age, complaints of memory loss in everyday life, memory performance on standardized tests at least one standard deviation below the average for young adults, and the absence of dementia. But this is not used widely. The MCI is widely used on early cognitive deficits indicating an illness that leads to dementia and have been adopted to indicate alternative interpretations of cognitive decline with increasing age. They performance in memory tasks is 1.5 standard deviations below age-matched controls that cover the spectrum between normal aging and dementia. 4–12% of MCI patients are expected to develop AD each year.[57,58]

Etiology

There are multiple etiological factors involved in dementia. They can be classified into neurodegenerative, vascular, intracranial space occupying lesions, metabolic, endocrine,

traumatic brain injury, epilepsy, infections, toxic, hypoxia, vitamin deficiency and rare causes. The dementias can also be classified into primary and secondary based on the etiological. The types are listed in Table 1. Neurodegenerative causes are the most common globally and in that Alzheimer's disease is the most common and prototypical of all dementias. Other common dementias include vascular dementia (VaD), dementia with Lewy bodies (DLB), and frontotemporal dementia (FTD).[59] The presentations and clinical features are different in most for the dementia. Based on the current diagnostic guideline, we can make a probable diagnosis and only postmortem studies are confirmative.

Protective Factors in Dementia

The psychosocial protective factors in dementia include higher education, rich social network, mentally stimulating activity, active social engagement and regular physical exercise and vascular factors in late life include use of antihypertensive, use of NASID's and light to moderate alcohol use. Higher education was associated with reduced incidence of dementia. This was supported by two meta-analysis which found 60% increased risk of dementia with lowest education. This evidence supports the "cognitive reserve" hypothesis which proposes that higher education improves the cognitive reserve and which provides compensatory mechanisms to cope with the degenerative pathology and therefore delay

Table 1: Primary and secondary dementias

Primary dementias

Definition: Primary degenerative disease are defined as those dementias in which there is selective and progressive loss of specific populations of neurons for reasons that in most cases remain unknown.

- Cortical
  - Alzheimer's disease
  - Vascular dementias
  - Prion disorders (including Creutzfeldt–Jakob disease (CJD)
  - Dementia with Lewy bodies (DLB)
  - Frontotemporal dementias (including Pick's disease)
  - Behavioral variant of FTD (bvFTD)
  - Semantic dementia (SD)
  - Progressive nonfluent aphasia (PNFA)
- Subcortical
  - Parkinson's disease
  - Huntington's disease
  - Progressive supranuclear palsy
  - Idiopathic basal ganglia calcification (Fahr's disease)
  - Spinocerebellar degeneration
  - Thalamic degeneration
  - Progressive subcortical gliosis

Secondary dementias

Dementias in which there is no degenerative process and for which there are causative agents or related to medical illness.

Endocrine and metabolic disorders

- Thyroid disease
- Parathyroid disease
- Adrenal disease
- Panhypopituitarism
- Prolonged hypoglycemia
- Pancreatitis
- Chronic obstructive pulmonary disease
- Congestive heart failure
- Hypoxia
- Hepatic encephalopathy

Contd...

- Hyponatremia
- Hypernatremia
- Uremia
- Dialysis

Nutritional disorder

- Folate, niacin, thiamine and vitamin B_{12} deficiency

Toxin and drug induced disorders

- Alcohol related syndromes
- Drugs and toxins

Infection related disorders

- Chronic meningtidis (fungal, tuberculosis, viral, bacterial)
- Neurosyphilis
- Neurobrucellosis
- Lyme neuroborreliosis
- Brain abscess
- Human immunodeficiency virus
- Arbovirus encephalitis
- Progressive rubella panencephalitis
- Progressive multifocal leukoencephalopathy
- Subacute sclerosing panencephalitis
- Creutzfeldt-Jakob disease
- Gerstmann-Straussler disease
- Postviral encephalitic syndromes
- Kuru
- Alpers' disease
- Whipple's disease
- Behçet's syndrome

Collagen vascular disorders

- Systemic lupus erythematosus
- Temporal arteritis
- Rheumatoid vasculitis
- Sarcoidosis
- Thrombotic thrombocytopenic purpura
- Granulomatous angiitis
- Idiopathic hypereosinophilic syndrome

Miscellaneous disorders and conditions

- Multiple sclerosis
- Brain tumor
- Radiation-induced
- Limbic encephalitis
- Subdural hematoma
- Hydrocephalus
- Sequelae of open and closed head injury
- Atrial myxoma

the dementia.[60,61] Systematic reviews have shown that poor social network or engagement in late life is associated with increased risk of dementia.[62] Rich social network or high level of social engagement has been hypothesized to improve social networks and resources which provides intellectual stimulations that affect cognitive and other health outcomes. Mentally stimulating activities, especially complex work with data or people have been associated with reduced risk of dementia. Again this supports the cognitive reserve hypothesis. A neuroimaging study also found that higher levels of complex activity was correlated with reduced rate of hippocampal atrophy.[63]

A higher level of physical activity was found to reduce the risk of dementia by 30–45%.[64] Regular physical exercise may promote vascular and circulatory health by reducing the blood pressure, serum lipids, obesity and blood glucose levels. Using antihypertensives especially angiotensin receptor blockers for a long-term has been found have beneficial effect in preventing dementia. Also, fewer neuritic plaques and neurofibrillary tangles in medicated hypertensive than in nonhypertensives.[65] Initial studies on dietary factors showed promising effects of antioxidants, vitamin B_{12} and folic acid in reducing the risk of dementia, later studies were did not support. Independent of vascular risk factor and physical activity, the Mediterranean diet (higher intake of fish, fruits and vegetables rich in antioxidants) has been found to reduce the risk of dementia.[66,67] Heavy alcohol use has been associated with three fold increase in AD and dementia especially in those with APOE epsilon 4 allele. However, two meta-analysis found that light to moderate alcohol use (1–3 drinks per day) was protective against dementia and cognitive decline (30–40% reduced in risk).[68]

Diet

Diets high in fish, fruit and vegetables are high in antioxidants and polyunsaturated fatty acids (PUFAs). In some observational population-based studies, people who had a high intake of vitamins E and C (both antioxidants) were less likely to show cognitive decline and had a lower AD risk than individuals with a low intake of these vitamins. By contrast, other large prospective studies of the effects of vitamins on AD risk found no such associations, and investigations examining the effect of dietary PUFAs on the risk of cognitive dysfunction proved inconclusive. Indeed, while several studies showed that the consumption of PUFAs led to reductions in the risks of dementia and AD, MCI and age-related cognitive decline, other studies found no association between dietary PUFAs and cognitive impairment. Scarmeas et al. reported that consumption of a Mediterranean-type diet (MeDi)—a diet characterized by a high intake of plant foods and fish (with olive oil as the primary source of monounsaturated fat), a moderate intake of wine and a low intake of red meat and poultry—reduced the incidence of AD and showed a trend towards reducing the risk of MCI. These effects were independent of levels of physical activity and vascular comorbidity. In a subsequent cohort study in France, MeDi was found not to alter performance on the Isaacs Set Test, the Benton Visual Retention Test or the Free and Cued Selective Reminding Test, but was associated with high MMSE scores, providing some support for the findings from the initial studies of this diet. In a meta-analysis of 15 prospective studies exploring the effect of alcohol on dementia risk, light to moderate alcohol consumption was associated with a reduction in the risk of AD and dementia.

Most RCTs examining the effects of antioxidant supplementation have found no association with cognitive performance. To date, prospective clinical trial data for dietary supplementation with omega-3 PUFAs have shown no overall effect on cognition in patients with MCI or AD, but have suggested that docosahexaenoic acid supplementation has a beneficial effect on cognitive function in people harboring the APOE ε4 allele and in the earliest stages of AD.

Reactive oxygen species are clearly associated with neuronal damage in AD; however, whether the presence of these molecules reflects a primary or secondary event in the neurotoxic process remains unclear. Deposition of Aβ, which is an early event in AD, leads to a decrease in cerebral iron and copper concentrations, resulting in oxidative stress and neuronal damage. Evidence from *in vitro* studies indicates that vitamin E reduces the extent of Aβ-induced lipid peroxidation and cell death. In addition, carotenes and vitamin C protect against lipid peroxidation. Furthermore, vitamin C reduces the formation of nitrosamines and may affect catecholamine synthesis. Evidence also exists that antioxidant intake reduces AD risk through a reduction in the risk of cerebrovascular disease. Besides reducing oxidative stress, PUFAs have favorable effects on neuronal and vascular functions and inflammatory processes.

Physical Activity

Epidemiological and experimental data suggest that physical exercise may promote brain health. Conflicting results have, however, emerged from cross-sectional and longitudinal observational studies that examined the relationship between exercise levels and cognitive decline or dementia: while some studies indicated that physical activity has a beneficial effect on brain health, others showed no association between these variables.

Physical activity could affect cognition via multiple mechanisms. An improvement in aerobic fitness increases cerebral blood flow, oxygen extraction and glucose utilization, and activates growth factors that promote structural brain changes, such as an increase in capillary density. In addition, rodent studies suggest that physical activity decreases the rate of amyloid plaque formation. RCTs exploring the effects of exercise on cognitive function in

healthy elderly individuals have yielded conflicting results. A recent meta-analysis that included 11 RCTs involving cognitively healthy people aged >55 years suggested that undertaking of aerobic physical activities improves selective cognitive functions, including cognitive speed, as well as auditory and visual attention.

Intellectual Activity

Following initial reports that elderly people with higher levels of education had a lower incidence of dementia than individuals with no education, cognitive activity was suggested to decrease the risk of cognitive decline by increasing cognitive reserve. Several prospective studies subsequently found that both young and old people who engage in cognitively stimulating activities, such as learning, reading or playing games, were less likely to develop dementia than individuals who did not engage in these activities.

RCTs have shown a beneficial effect of intellectual interventions on cognitive function in elderly, dementia-free individuals. The benefits of cognitive training seem to be domain specific, however. Several trials found that while cognitive training can improve memory, reasoning and mental processing speed in older adults, cognitive training did not have an effect on all cognitive domains, and did not affect day-to-day functioning. In addition, one study found that among elderly individuals, those with memory impairment showed less improvement in cognition through memory training than those without such impairment. Consequently, in elderly people, the effect of cognitive training on the risk of dementia is unclear, but several trials are underway.[69]

Diagnosis

Dementia is a syndrome like delirium. Hence careful attention has to be given and a detailed history from the patient and an informant, in addition to mental state examination and cognitive testing is recommended. In the history, look for the type of onset of cognitive impairment (insidious or sudden), course (gradual or stepwise, progressive or episodic, or fluctuating), duration of impairment, any accompanying symptoms (like mood, psychotic symptoms or personality changes). Efforts should be made to look for evidence of treatable causes in the history, so the focus of physical examinations and investigations can be planned. Past and family history will give leads into the genetic etiology. Also, past history of psychiatric or neurological disorders may be helpful in further management. Also, an attempt should be made to look for any abuse during the physical examination.

The mental state examination is a very valuable and very important to diagnose and categorize the type of dementia. It may be painstaking and time consuming to do a MSE with elderly patients but the amount of time invested will give fruitful evidence for further management. In the general appearance, patient's attire, cleanliness and rapport will help in suggesting the severity of dementia. Cognitive tests are the key to diagnosis but PwD may not co-operate for the full assessment and hence the interview sessions have to be split and time should be given for them to complete tasks. It is important to remember the age of the patient, disabilities due to aging (such as vision and hearing deficits) intellectual capacity, socioeconomic background, educational background, rural or urban settings and premorbid personality. The tests as general fund of information, similarities and differences and tests of abstraction also have to be accommodative of all these factors to appropriately diagnose the deficits. Extended cognitive tests may be required if there are any confusion or it is historically evident but not picked up by bedside cognitive tests. There are lots of scales to evaluate and have their own limitations. The DSM-IV diagnosis of dementia and amnestic disorder are subsumed under newely named entity major neurocognitive disorder (NCD) in DSM-5.

DSM-5 Criteria for Major Neurocognitive Disorder

a. Evidence of significant cognitive decline from a previous level of performance in one or more cognitive domains (complex attention, executive function, learning and memory, language, perceptual-motor, or social cognition) based on:
   1. Concern of the individual, a knowledgeable informant, or the clinician that there has been a significant decline in cognitive function; and
   2. A substantial impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.

b. The cognitive deficits do not interfere with independence in everyday activities (i.e., at a minimum, requiring assistance with complex instrumental activities of daily living such as paying bills or managing medications).

c. The cognitive deficits do not occur exclusively in the context of a delirium.

d. The cognitive deficits are not better explained by another mental disorder (e.g. major depressive disorder, schizophrenia).

Specify whether due to:

• Alzheimer's disease	Frontotemporal lobar degeneration
• Lewy-body disease	Vascular disease
• Traumatic brain injury	
• Substance/medication use	HIV infection
• Prion disease	Parkinson's disease
• Huntington's disease	Another medical condition
• Multiple etiologies	Unspecified

Coding note: Code based on medical or substance etiology. In some cases, there is need for an additional code for the etiological medical condition, which must immediately precede the diagnostic code for major neurocognitive disorder, as shown in Table on pp. 302-304.

Contd...

Contd...

Specify:

Without behavioral disturbance: If the cognitive disturbance is not accompanied by any clinically significant behavioral disturbance

With behavioral disturbance (*specify disturbance*): If the cognitive disturbance is accompanied by a clinically significant behavioral disturbance (e.g. psychotic symptoms, mood disturbance, agitation, apathy, or other behavioral symptoms).

Specify current severity:

Mild: Difficulties with instrumental activities of daily living (e.g. housework, managing money).

Moderate: Difficulties with basic activities of daily living (e.g. feeding, dressing).

Severe: Fully dependent.

DSM-5 Criteria for Mild Neurocognitive Disorder

a. Evidence of modest cognitive decline from a previous level of performance in one or more cognitive domains (complex attention, executive function, learning and memory, language, perceptual-motor, or social cognition) based on:
   1. Concern of the individual, a knowledgeable informant, or the clinician that there has been a mild decline in cognitive function.
   2. A modest impairment in cognitive performance, preferably documented by standardized neuropsychological testing or, in its absence, another quantified clinical assessment.
b. The cognitive deficits do not interfere with capacity for independence in everyday activities (i.e. complex instrumental activities of daily living such as paying bills or managing medications are preserved, but greater effort, compensatory strategies, or accommodation may be required).
c. The cognitive deficits do not occur exclusively in the context of a delirium.
d. The cognitive deficits are not better explained by another mental disorder (e.g. major depressive disorder, schizophrenia).

Specify whether due to:

- Alzheimer's disease
- Frontotemporal lobar degeneration
- Lewy body disease
- Vascular disease
- Traumatic brain injury
- Substance/medication use
- HIV infection
- Prion disease
- Parkinson's disease
- Huntington's disease
- Another medical condition
- Multiple etiologies
- Unspecified

Coding note: For mild neurocognitive disorder due to any of the medical etiologies listed above, code 331.83 (G31.84). Do not use additional codes for the presumed etiological medical conditions. For substance/medication-induced mild neurocognitive disorder, code based on type of substance; see "Substance/Medication-Induced Major or Mild Neurocognitive Disorder." For unspecified mild neurocognitive disorder, code 799.59 (R41.9).

Contd...

Contd...

Specify: Without behavioral disturbance: If the cognitive disturbance is not accompanied by any clinically significant behavioral disturbance.

With behavioral disturbance (specify disturbance): If the cognitive disturbance is accompanied by a clinically significant behavioral disturbance (e.g. psychotic symptoms, mood disturbance, agitation, apathy, or other behavioral symptoms).

Laboratory and Investigations

Once a detailed history is collected and physical examination has been completed. Laboratory investigations need to be considered to corroborate the history and to identify the etiology of some treatable dementias and to look into comorbid physical disorders. In this aspect the investigations can be classified into three aspects:

To Corroborate the Diagnosis

Neuroimaging is very importance to rule out infarcts, mass lesions, tumors, and hydrocephalus. It is also very vital to identify the pattern of neuronal damage and also may provide clues to the diagnosis. Computed tomography without contrast or magnetic resonance imaging may be useful in the essential work-up of PwD.

Rule Out Treatable or Contributory Causes of Dementia

Complete blood count, serum electrolytes, renal and hepatic function, blood glucose, albumin and protein, vitamin B_{12} and folate, rapid plasma reagin or VDRL (syphilis), thyroid-stimulating hormone, urinalysis are important investigations to be considered to rule out treatable causes of dementia.The dementias can be classified into reversible and irreversible types based on the reversible causes such as infections, vitamin deficiencies, etc. The list of types of reversible and irreversible dementias are given in Table 2.

However, sensitive tests such as VDRL, HIV and HBsAG may be required in suspected cases of exposure to STD and patients with personality changes associated with dementia due to high-risk behavior.

The CSF analysis also may be important when suspecting neurosyphilis, cysticercosis, viral, fungal infections and CSF pressure analysis in normal pressure hydrocephalus.

Specialized tests such as drug screen, toxins level, electrolytes are very important especially when the history is not clear or there is delirium superimposed on dementia. It should be based on clinical suspicion and need to consider the cost effectiveness of the tests especially in a country like India.

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