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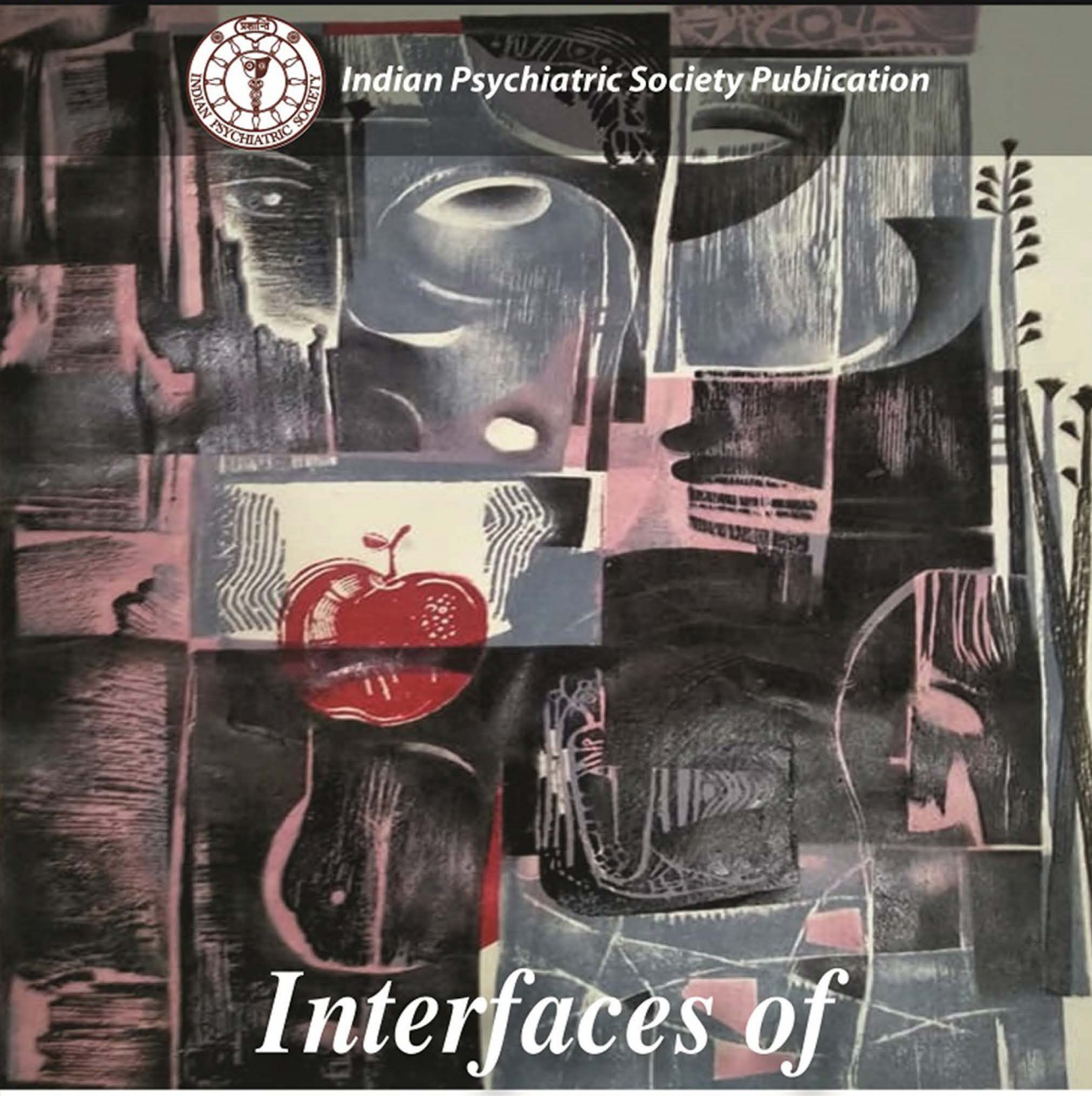

Interfaces of PSYCHIATRY

Editors
Vinay Kumar
Sandeep Grover

JAYPEE

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CHAPTER 3

Interface with Neurology

Adarsh Tripathi, Erika Pahuja, Sujita Kumar Kar

ABSTRACT

Neuropsychiatry as a branch comprised both neurology and psychiatry till around 20th century after which brain/mind dichotomy arose and neurology and psychiatry parted their ways. Recent advances in neuroscience make it almost impossible at this time to precisely draw the line between neurological and psychiatric disorders. Because of the vast increase in knowledge in recent years, most of the psychiatric disorders have been found to have neuropathological basis. This chapter focuses on the areas of cross fertilization between the two branches. Clearly, keeping in view the inseparability of these branches, it is also imperative to liaison between the branches.

INTRODUCTION

Ramsay in 1979 rightly quoted "Neurology and psychiatry are a pair of Siamese twins who cannot be separated".[1] Till 20th century, Neurology and Psychiatry were considered a single branch of medicine, "Neuropsychiatry". Brain and mind were considered one entity.[2]

The founding father of neuropsychiatry, Wilhelm Griesinger (1817–1868), professor of neurology and psychiatry stated, "Psychische Krankheiten sind Erkrankungen des Gehirns," or "Mental illnesses are diseases of the brain".[3] In his revolutionary textbook of psychiatry, Pathologie und Therapie der Psychischen Krankheiten (1845), Griesinger emphasized on a change of attitude toward the psychiatric patient from that of sinner who should be punished and isolated, to sufferer of a biological disease who requires curing.[3] Charcot, Freud, Jackson, Bleuler, Meynert, Liepmann, Pick, Oppenheim, Korsakoff, von Monakow, Babinski, Janet, Kraepelin, Bonhoeffer, and Alzheimer thought in terms of unified study of brain and mind. They were considered Neuropsychiatrists and worked in both the specialties.[4] These neuropsychiatrists were influenced by Griesinger's work and hoped to discover organic causes of mental illnesses.[3]

During 20th century, with the development of psychodynamics, brain/mind dichotomy arose and neurology and psychiatry parted their ways.[4] Neurology laid claim to those disorders of the nervous system with cognitive and behavioral abnormalities with elicitable physical signs, established etiologies and demonstrable anatomic pathology. Psychiatry claimed those disorders of mentation, mood and thought for which there was no or minor elicitable physical signs and no visible pathology, i.e., idiopathic functional illnesses.[4] Hence psychiatry remained more dependent on symptoms whereas neurology became dependent on signs and laboratory tests/imaging. This led to the emergence of "organic versus functional" dichotomy.[4]

After World War II, the division between neurology and psychiatry became explicit

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with the separation of the journal "The Archives of Neurology and Psychiatry" into two journals,[5] foundation of The American Academy of Neurology in 1948 to deal with "pure" neurological issues, and establishment of separate neurology departments throughout the United States, deletion of compulsory psychiatry training for neurologists by the Residency Review Committee for Psychiatry and Neurology in 1965.[4]

But even till date there are certain disorders, gray zones, which pose a difficulty drawing a line between neurology and psychiatry. This is because however, far these specialties drift away, they have "the common roots: Brain".

NEUROPSYCHIATRY

Neuropsychiatry has been defined in a variety of ways. International Neuropsychiatric Association defines the flavor of Neuropsychiatry in the most plausible way as "a field of scientific medicine that concerns itself with the complex relationship between human behavior and brain function, and endeavors to understand abnormal behavior and behavioral disorders on the basis of an interaction of neurobiological and psychological—social factors".[6] As stated by Arzy et al. in their work The Science of Neuropsychiatry: Past, Present, and Future (2014), the suggested definitions of "Neuropsychiatry" may be divided into two main categories—minimal and maximal. The minimal approach puts neuropsychiatry in the borderland between neurology and psychiatry, and sees its subject matter as "disorders that cross the boundary between the two disciplines", as well as neurological aspects of psychiatric disorders and psychiatric aspects of neurological disorders. The maximal approach includes the full range of central nervous system (CNS) diseases (occurring "above the foramen magnum"), as well as most of the psychiatric disorders, within the scope of neuropsychiatry.[3]

Describing the disorders in maximal approach is beyond the scope of this chapter. The disorders in minimal approach are described herewith.

DISORDERS THAT CROSS THE BOUNDARY BETWEEN TWO DISCIPLINES

Delirium

Delirium in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) is defined as acute decline in both the level of awareness and cognition with particular impairment in attention.[7] Delirium often involves perceptual disturbances, abnormal psychomotor activity, and sleep cycle impairment and can be life-threatening. Term delirium is derived from a Latin word *Delirio* which means *to be crazy*. It is actually decompensation of cerebral functions. The core features include altered consciousness, global disturbance of cognition, perceptual disturbance in the background of a physical abnormality with a rapid onset and fluctuating course.[8] Delirium tremens (DT) is alcohol withdrawal delirium characterized by core features of delirium and tremors, usually has an onset 3–4 days after the last drink. A number of short-term and long-term negative consequences follow delirium. Short-term consequences include stress to the patient and family members, prolonged hospital stay, delay in postoperative mobilization and rehabilitation and increased mortality. Long-term consequences include functional and cognitive decline which may progress to dementia.[9]

Epidemiology

Data suggest an incidence rate of delirium is 3–42% in hospitalized patients and prevalence to vary from 5 to 44% among the hospitalized patients. Indian studies reveal that prevalence

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of delirium in intensive care units (ICUs) varies from 26.2 to 68.2% and the incidence rates vary from 9.27 to 59.6%.[9]

Clinical Features

Delirium is characterized by acute onset of symptoms, fluctuating course, and resolution between days to weeks.[10] Prolonged delirium lasting >6 months has been observed in studies by Levkoff et al.[10,11] and Mori and Yamadori.[12] Delirium is of three types: (1) hyperactive, (2) hypoactive, and (3) mixed (fluctuating between hyperactive and hypoactive). Hyperactive is a more common subtype and hypoactive is associated with prolonged hospital stay and higher mortality rates.[10,13]

Clinical features of delirium have been categorized in a number of ways. They are categorized as cognitive, noncognitive, and motoric symptoms.[9] Taylor and Lewis divided delirium symptoms into six subheadings: (1) impairment of consciousness, (2) thinking, (3) memory, (4) psychomotor disturbances, (5) perceptual, and (6) emotional disturbances.[10,14] Impaired attention, fluctuating orientation, reduced or excessive awareness to environmental stimuli, hallucinations predominantly visual, fluctuating delusions, altered sleep wake cycle, sundowning (worsening symptoms after sunset) are characteristic features of delirium.[8,10]

Risk Factors[8,10]

Risk factors for delirium are described in **Table 1**.

Pathophysiology[8]

Multiple theories have been proposed to understand pathophysiology of delirium:[8]

TABLE 1: Risk factors for delirium.

Predisposing factors	*Precipitating factors*
Elderly	Severe acute illness
Male sex	Poor nutritional status/anemia
Sensory deficits	*Metabolic abnormalities:* Hyponatremia, hypokalemia
Dementia	Hypoxemia
Past history of delirium	Shock
Depression	Bladder catheter use and urinary tract infections
Immobility	Infections
History of falls	Intensive care unit admissions
Underlying physical illness: DM, renal, hepatic, endocrine disease, CAD, etc.	Dehydration
Substance use: Alcohol, BZPs	Fever/hypothermia
Compromised brain	Use of physical restraints
Dehydration and malnutrition	Pain
Use of psychotropics (particularly medications with anticholinergic properties)	• Cardiac and noncardiac surgeries • Orthopedics surgery • *Neurological disorders:* Stroke, meningitis • *Use of drugs:* BZPs and alcohol withdrawal, polypharmacy, anticholinergic drugs

(BZPs: benzodiazepines; CAD: coronary artery disease; DM: diabetes mellitus)

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- Disturbance in brain oxygen supply versus demand
- Generalized disruption in higher cortical function
- Neurochemical disturbances affecting cholinergic, dopaminergic, glutamatergic, and gamma-aminobutyric acid (GABA) neurotransmission
- Inflammation

Differential Diagnosis

Differential diagnosis of delirium includes dementia, depression, and psychosis **(Table 2)**. Catatonia is a differential diagnosis for hypoactive delirium.[8,9]

TABLE 2: Differential diagnosis of delirium.

	Delirium	*Dementia*
Onset	Acute	Insidious
Consciousness	Altered	Intact
Course	Fluctuating/ reversible	Progressive/ downhill
	Delirium	*Depression*
Mood	Mood lability	Persistent sadness
Diurnal variation	Sundowning/ evening worsening of symptoms	Morning worsening of symptoms
	Delirium	*Psychosis*
Onset	Sudden	Acute to insidious
Course	Fluctuating, sundowning	Stable
Consciousness	Reduced	Clear
Attention	Impaired	May be impaired
Cognition	Globally impaired	Selectively impaired
Orientation	Fluctuating/ impaired	Usually intact
Delusions	Fleeting/ poorly formed	Well formed
Hallucinations	Usually visual	Usually auditory
Cause	Organic	Organicity usually absent

Assessment[9]

Screening instruments: Delirium Observation Scale, Clinical Assessment of Confusion—A and B, Confusion Assessment Method (CAM).

Diagnostic instrument: CAM-ICU (Confusion Assessment Method for ICU)[9]

Management

Following key points need to considered during management:[9]

- Evaluate for the underlying cause, predisposing, precipitating, maintaining factors, and correct the cause
- Stop unnecessary medications
- Maintain hydration and correct biochemical abnormalities
- Monitor vitals, input/output, symptoms regularly
- Maintain liaison with the primary treating team (treating the underlying cause)
- Prevent secondary complications

Pharmacological management includes use of low-dose antipsychotics (haloperidol, olanzapine, risperidone, quetiapine). The dictum "start low and go slow" needs to be followed. Cholinesterase inhibitors also show improvement in some patients. DT requires adequate benzodiazepine replacement.[9]

Nonpharmacological methods include repeated reorientation and support, unambiguous environment, ensure proper sleep, correct sensory impairments by providing vision glasses, hearing aids, encourage mobilization.[9]

Psychoeducation of the family members about the course and prognosis.

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Dementia

Dementia is defined as a disease process marked by progressive cognitive impairment in clear consciousness. It is cognitive impairment in one or more of the domains of complex attention, executive functions, learning and memory, language, perceptual motor ability, and social cognition.[8]

According to the World Health Organization (WHO), around 50 million people have dementia worldwide and every year around 10 million new cases are diagnosed.[15]

Describing dementia and its types in detail is beyond the scope of the chapter, hence only a brief introduction is given.

Dementia has multiple etiologies and each type differs in some features from each other. Some are reversible causes such as endocrine and nutritional causes whereas others are irreversible such as neurodegenerative, vascular, and neurological causes.[8] Dementia may be subcortical or cortical. Alzheimer's disease is the most common dementia globally **(Table 3)**.[8]

Scales for Assessment[8]

- *Mini-Mental State Examination (MMSE):* Screening tool. MMSE score <23 points toward dementia. Most commonly used tool for screening.
- Mini Cog
- Addenbrooke's Cognitive Assessment–revised
- Montreal Cognitive Assessment (MoCA)

Neuroimaging is advised to rule out neurological causes that can cause or contribute to dementia severity.

Treatment

Both pharmacological and nonpharmacological treatment have equal importance.[8]

TABLE 3: Etiology of dementia.

Neurodegenerative	• Alzheimer's disease • Dementia with Lewy body • Frontotemporal dementia • Parkinson's disease • Huntington's disease • Corticobasal syndrome • Progressive supranuclear palsy • Prion-related: Creutzfeldt–Jakob disease, variant Creutzfeldt–Jakob disease, Gerstmann–Straussler–Scheinker disease, fatal familial insomnia
Vascular	• Infarction • Binswanger's disease • Hemodynamic insufficiency • Small vessel disease • Watershed area hypoperfusion
Neurological	• Multiple sclerosis • Normal pressure hydrocephalus • Brain tumor • Nonconvulsive status epilepticus • Acute intermittent porphyria
Endocrine	• Hypothyroidism • Hypoparathyroidism • Hyperparathyroidism • Cushing's syndrome • Adrenal insufficiency • Hypoglycemia
Nutritional	• Vitamin B_{12} deficiency • Thiamine deficiency • Niacin deficiency • Folate deficiency
Infectious	• Human immunodeficiency virus (HIV) • Neurosyphilis • *Cryptococcus* • Whipple's disease

Contd...

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Contd...

	• Progressive multifocal leukoencephalopathy • Tuberculosis • Sarcoidosis
Metabolic	• Hepatic insufficiency • Renal insufficiency • Wilson's disease • Metachromatic leukodystrophy • Neuroacanthosis
Brain injuries	• Subdural hematoma • Dementia pugilistica • Post anoxic • Post encephalitic
Exposure	• Alcohol • Wernicke–Korsakoff syndrome • Marchiafava–Bignami disease • Heavy metals • Irradiation • Anticholinergic medications • Carbon monoxide • Industrial/environmental toxins (fertilizers, pesticides)
Vasculitides	• Lupus erythematosus • Sjögren's syndrome

Pharmacological treatment involves use of cholinesterase inhibitors, N-methyl-D-aspartic acid (NMDA) receptor antagonist.[8]

Nonpharmacological interventions include psychoeducation of patient and family members, cognitive retraining, stimulation-oriented therapies, safety awareness and management, and caregiver support.[8]

Behavioral and Psychological Symptoms of Dementia

Behavioral and psychological symptoms of dementia (BPSD) include mood changes (depression—most common), anxiety, personality changes, psychosis, sleep disturbances, agitation, impulsivity, disinhibition, wandering, and suspiciousness. These symptoms are treated accordingly.[8]

NEUROLOGICAL DISORDERS WITH PSYCHIATRIC MANIFESTATIONS

Multiple Sclerosis

Multiple sclerosis (MS) is the most common chronic disabling CNS disease in young adults.[16] It affects 1 in 1,000 people in Western countries.[16] It is an autoimmune, demyelinating disorder with symptoms of muscle weakness, vision loss, spasticity, sexual dysfunction, bladder dysfunction, and fatigue. It is characterized by immune system attacking the myelin sheath, oligodendrocytes, and nerve itself. MS is diagnosed by McDonald criteria (revised in 2017).[17]

Neuropsychiatric manifestations of MS: Neuropsychiatric symptoms (NPSs) are found in up to 60% of patients of MS. These symptoms contribute to morbidity and mortality associated with MS and have significant impact on quality of life of people with MS.[18,19] Generally, neurological symptoms precede NPS, but in around 2–3% concomitant neurological and psychiatric symptoms; and in around 0.2–2% psychiatric manifestations precede the neurological symptoms.[20,21] Most common psychiatric disorders observed with MS are described in **Table 4**.

Major Depressive Disorder

Major depressive disorder (MDD) is the most common psychiatric disorder seen in patients of MS.[22] Fatigue, poor concentration, anorexia, memory deficits, and sleep issues are shared symptoms between MS and MDD and pose a difficulty in diagnosis of comorbid MDD in MS.

The 7-item Beck Fast Screen for medically ill patients, the Hospital Anxiety and Depression

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TABLE 4: Most common psychiatric disorders inmultiple sclerosis.

Psychiatric disorder	*Prevalence*
MDD	• 15% annual prevalence[22] • 50% of MS patients experience it in lifetime[23,24]
BPAD	• 0.3–2.4%[18] • Twice as that of general population[25]
Psychosis	• 2–4% • Thrice as that of general population[26]
Anxiety disorders	• 36% • GAD—19%; panic disorder—10%; OCD—9%; social anxiety disorder—8%[27]
Alcohol—harmful use/dependence	13.6%[19,28]
Pseudobulbar affect	10%[19]

(BPAD: bipolar affective disorder; GAD: generalized anxiety disorder; MDD: major depressive disorder; MS: multiple sclerosis; OCD: obsessive-compulsive disorder)

Scale, and the Beck Depression Inventory have been used in MS patients with MDD but should be interpreted cautiously due to common symptoms between the two disorders. Beck Fast Screen for medically ill patients excludes the physical symptoms that would normally confound other screening tools.[19]

One of the important causes of mortality in patients with MS is suicide with rates as high as 3% (completed suicide).[19,29] This is approximately 7.5 times of the general population.[19]

Etiological factors implicated in development of MDD in patients of MS include dysfunctional hypothalamic pituitary adrenal axis; psychosocial factors such as lower socioeconomic status, inadequate coping, unpredictable disease course, limited social support, loss of recreational activities, severe physical disability, perceived physical incapacity, hopelessness together with uncertainty about prognosis.[19,30] Treatment with disease-modifying agents interferon beta-1a and 1b has been linked with development of depression in MS patients though no conclusive evidences are available till date.[19,31]

Neuroimaging studies show that around 40% of patients with comorbid MDD and MS have lesions in medial inferior prefrontal cortex and the anterior temporal lobe.[32] Abnormalities in white matter connectivity and regional integration in frontal lobes and limbic regions; including the hippocampus and amygdala and subcortical regions have been observed.[19] These findings are suggestive of a biological correlation between demyelination of nerve fibers and psychiatric signs and symptoms.

There is a crunch in randomized controlled trials (RCTs) evaluating treatment options for MDD in MS. Available options include use of antidepressants such as selective serotonin reuptake inhibitor (SSRI)—sertraline, paroxetine; tricyclic antidepressant (TCA) like desipramine; cognitive behavioral therapy (CBT).[19] SSRIs can potentially reduce demyelination by reducing inflammation, reducing axonal degeneration by induction of glycogenolysis.[33] Studies on animal models show that lithium abolishes experimental autoimmune encephalomyelitis (EAE) and reduces demyelination. So, lithium may be used with caution in cases of treatment-resistant MDD.[34] Electroconvulsive therapy (ECT) has also been seen effective in treatment-resistant cases.

Bipolar Affective Disorder

Incidence of mania is almost twice as compared to general population, and develops later in the course of illness.[25] Etiological factors contributing are use of steroids,[19,35]

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antidepressant-induced manic switch, other agents including baclofen, dantrolene, tizanidine.[19,36] Genetic contributions with reports of familial clustering of both disorders and genetic loci 6q21-22 in close approximation with HLA are being studies.[37]

Treatment options include use of mood stabilizers such as valproate, lithium (additional disease-modifying benefits; used with caution due to risk of polyuria), atypical antipsychotics such as olanzapine.[19]

Psychosis

Affective as well as nonaffective psychosis with reports showing predominance of positive symptoms have been observed in patients with MS. Etiological factors include inflammatory process, psychosocial circumstances, medicinal use of cannabis, and use of steroids.[16]

Magnetic resonance imaging (MRI) findings show demyelination in periventricular white matter, temporal, and frontotemporal regions.[26]

Treatment options include use of low doses of second-generation antipsychotics with fewer propensities for extrapyramidal symptoms (EPS). Risperidone[38] and quetiapine[39] have demonstrated disease-modifying benefits in EAE mice models.

Anxiety Disorder

There is a dearth of research in relation anxiety disorders in MS. Risk factors for anxiety disorders include newly diagnosed MS, increased MS disease activity, experiencing pain, fatigue or sleep disturbance, female gender, social isolation, a past history of suicide and MDD, and comorbid alcohol or psychoactive substance misuse.[16] Development of self-injectable disease-modifying treatments (DMTs) has led to "self-injection anxiety" in around 50% of the patients.[40]

Treatment includes use of SSRI (first line), venlafaxine, buspirone, pregabalin, gabapentin, and beta-blockers. Psychotherapy may be considered.[19]

Psychoactive substance use: High rates of use of cannabis and alcohol have been observed in MS. Cannabis use alleviates certain neurological symptoms such as spasticity, tremors, pain, insomnia, and bladder dysfunction. Certain synthetic cannabinoid compounds are licensed for treatment of spasticity and neuropathic pain in MS in some European countries.[19,41]

Pseudobulbar Affect and Euphoria

Pseudobulbar affect (PBA), also known as pathological laughing and crying, emotional incontinence, involuntary emotional expression disorder, occurs in chronic sufferers and in progressive stages; with uncontrollable crying more common than laughing.

PBA is possibly due to lesions in cerebropontocerebellar pathways.[42] Monoaminergic system involvement has also been implicated.[19]

Treatment options include use of combination of dextromethorphan and quinidine, SSRI, norepinephrine reuptake inhibitor (SNRI), mirtazapine, levodopa, and amantadine.[19,43,44]

Epilepsy

According to the International League Against Epilepsy, 2014; a person is considered to have epilepsy if they meet any of the following conditions:[45]

- At least two unprovoked (or reflex) seizures occurring >24 hours apart
- One unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years
- Diagnosis of an epilepsy syndrome

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Epilepsy is considered to be resolved for individuals who had an age-dependent epilepsy syndrome but are now past the applicable age or those who have remained seizure-free for the last 10 years, with no seizure medicines for the last 5 years.[45]

According to WHO, approximately 50 million people currently have epilepsy worldwide. The current prevalence of active epilepsy (i.e., continuing seizures or with the need for treatment) at a given time is between 4 and 10 per 1,000 people. However, some studies suggest a higher prevalence between 7 and 14 per 1,000 people in the low- and middle-income countries (LMICs). Around 70% of people with epilepsy respond to antiepileptics and can live apparently normal lives. Measured in disability-adjusted life years (DALYs), epilepsy forms 0.5% of the global burden of disease, with 80% of that burden corresponding to the developing countries.[46] Epilepsy is associated with a number of neuropsychiatric manifestations; which also reflect the severity of the illness. There are a number of associations between epilepsy and psychiatric disorders:

- Associated social limitations, discrimination and stigmatization
- Neurobiological involvement
- Seizures increase the vulnerability for psychiatric disorder and vice versa as both are caused by abnormality of brain.
- Psychotropics (clozapine, clomipramine, bupropion) decrease seizure threshold. Antiepileptics such as levetiracetam are associated with behavioral problems.

Prevalence of various psychiatric disorders in epilepsy are described in **Table 5**.

TABLE 5: Prevalence of various psychiatric disorders in epilepsy.

Psychiatric disorder	*Prevalence*
Any mental health disorder	35.5%[47]
Mood disorder	24.4%[47]
Anxiety disorder	22.8%[47]
Suicidal ideations	25%[47]
Psychosis	7–8%[48,49]
Personality disorder	18%[50]

Psychiatric disorders are also commonly found comorbid with pediatric epilepsy. Studies have found that attention-deficit hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) are frequently associated with pediatric epilepsy.[51] Other commonly associated disorders include anxiety disorders, depression, conduct disorder, and developmental delay (National Survey of Children's Health).[51,52]

Psychiatric problems associated with epilepsy are divided between ictal, peri-ictal, and interictal.[48]

Ictal Features

Epileptic aura may be characterized by mood changes, anxiety, depersonalization, derealization, hallucinations (visual, auditory, gustatory, olfactory), forced thinking, and pleasurable auras.

Peri-ictal

Prodromal symptoms occurring before a seizure episode are irritability, depression, headache, and confusion. Postictal symptoms include postictal psychosis (PIP) (60% of all psychotic experiences associated with epilepsy).[47] PIPs usually occur in patients with temporal lobe epilepsy (TLE), and extratemporal structural lesions.[47,53] PIP is characterized by lucid interval of 24–72 hours,[48] florid psychomotor excitation, mystic delusions, mood swings, paranoia. It lasts for days to week,[47] shows increased slowing in electroencephalogram (EEG), usually resolve spontaneously but may require low dose of antipsychotics in some patients to reduce mortality and morbidity.

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Interictal

Mood disorder: Depression is the most common psychiatric comorbidity in epilepsy.[48] Most common mood state is dysthymia or interictal dysphoric disorder of epilepsy.[48,54] Involvement of the mesiotemporal structures (hippocampus and amygdala) and the modulation of major neurotransmitter pathways (serotonin) by the epilepsy accounts for comorbid mood disorders.[47] Treatment involves use of antidepressants with low potential to reduce seizure threshold.

Psychosis: It occurs in chronic/treatment refractory TLE.[47,48] A fraction of patients have psychotic symptoms with worsening seizure frequency or antiepileptic withdrawal and other fraction exhibits Landolt's phenomenon/forced normalization/paradoxical normalization/alternating psychosis, i.e., improving seizure frequency and EEG normalization with worsening psychosis.[47,48,55,56] Patients with early onset of seizures, long interval of poorly controlled partial complex seizure with secondary generalization, left temporal focus, mediobasal temporal lesions, recently diminished seizure frequency are predisposing factors for development of interictal psychosis (IIP).[48] Treatment involves adequate control of seizures with antiepileptics. Antipsychotics may be required in some patients.

Personality disorder: Most common personality disorder associated with epilepsy is borderline personality disorder (BPD). Gastaut–Geschwind syndrome occurs in a subset of patients with TLE. It is characterized by intensified mental life, viscosity of speech, hypergraphia, hyper-religiosity, hyposexuality.[48]

Anxiety: Anxiety may occur as an ictal, perictal phenomenon, associated with depression or an isolated anxiety disorder. It is more common in focal epilepsy (TLE) as compared to generalized epilepsy.[48]

Suicide: Risk of completed suicide is four to five times higher as compared to general population. Patients with TLE, BPD, and PIP are predisposed to attempt suicide.[48]

Psychotropic Drugs and Epilepsy[57]

The effects of psychotropic drugs on seizure threshold are described in **Table 6**.

Pseudoseizures

Psychogenic nonepileptic seizures (PNESs)/pseudoseizures are episodic seizure-like behavioral events that occur in the absence of abnormal electrical discharge in the brain. An underlying psychological stress phenomenon or psychological conflict is usually present. Around 30–50% of patients who have PNESs have epilepsy,[56,58] and 20–60% of patients who have epilepsy have PNESs.[56,59] Differences between true and pseudoseizures are described in **Table 7**.

TABLE 6: Effects of psychotropic drugs on seizure threshold.

Fluoxetine, fluvoxamine	Lowers seizure threshold
Clomipramine, amitriptyline, dosulepin	Epileptogenic, particularly at high doses
Bupropion	Epileptogenic, should be avoided
Lithium	Low proconvulsive effect at therapeutic dose; epileptogenic in overdose
Risperidone, olanzapine, quetiapine	Caution required. May lower seizure threshold
Clozapine	Highly epileptogenic. Avoid if possible
Chlorpromazine	Most epileptogenic in first-generation antipsychotics

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TABLE 7: Differentiating features between true and pseudoseizures.[56,60]

Clinical features	*True seizures*	*Pseudoseizures*
Ictal features		
Stereotyped nature	Yes	No
Episodes during sleep	Yes	No
Duration	Brief (1–2 min)	Usually long
Onset	Abrupt	Usually gradual
Precipitant	Usually absent	Usually present
Consciousness	Lost	Preserved
Aura	Usually present	Absent
Synchronous movements	Yes	No
Jactitation	No	Yes
Pelvic thrusting	No, though occurs only in around 24% of frontal lobe epilepsy and 2–4% of TLE[61]	Yes
Prolonged body flaccidity	No	Yes
Eye closure in tonic phase	No	Yes
Jaw clenching in tonic phase	No	Yes
Tongue bites	Yes, lateral	Usually absent. Rarely tip of tongue bite may be present
Pupillary reflex	May be normal	Normal
Plantar	Extensor	Flexor
Ictal moaning and crying	Absent	Present
Emotive speech	Absent	Present
Ictal stuttering	Absent	Present
Postictal features		
Stertorous breathing	Yes	No
Postictal nose rubbing	Yes	No
Postictal headache	Yes	No
Recall of items during ictus	No	Yes
Postictal confusion	Yes	No
Investigations		
EEG ictal changes and postictal slowing	Yes	No
pH after attack	May change	Same
Prolactin after attack	Rises	Normal

Pseudoseizures are diagnosed clinically by eliciting detailed history and examination. EEG and brain scanning may be done in confusing cases.

Treatment involves establishing rapport and therapeutic alliance, treat the underlying psychiatric comorbidity, development of insight into the symptoms, psychosocial explanation of symptoms, solving the psychosocial problems, cut secondary gains.

Parkinson's Disease

An essay on the shaking palsy by James Parkinson reported 6 cases of *paralysis agitans* in 1817 and described predominant motor symptoms of Parkinson's disease (PD).[62] Today we know that PD has a variety of motor and nonmotor symptoms. Motor symptoms include tremors, rigidity, bradykinesia and nonmotor symptoms include the cognitive and behavioral abnormalities.[63]

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TABLE 8: Various psychiatric disorders associated with Parkinson's disease (PD).

Psychiatric disorder	*Prevalence*
Depression	20–40%[64,65]
Anxiety	40%[65,66]
Psychosis	• 10% of untreated PD • 15–40% of treated PD[65,67]
Dementia	• 30% • 80% with course >10 years[65,68]
Impulse control disorders	15%[65]
Sleep disorders	40–90%[65]
Apathy	40%[69]

Various psychiatric disorders associated with PD are enlisted in **Table 8**.

Depression

Depression and PD follow a vicious cycle increasing the risk of each other. PD is 3.24 times more likely to occur in patients with depression.[63,70] Patients on antiparkinsonian medications have higher rates of depression as compared to drug naïve patients (75.5% vs. 59.2%).[63,71] Features of depression in PD differ from idiopathic depression. Patients with PD have higher rates of anxiety, dysphoria, irritability, pessimism, suicidal ideation without suicidal behavior, and less rates of self-guilt and reproach.[65,72] Depressive symptoms usually fluctuate with motor symptoms and may be present only in the off phase. Risk factors include female gender, right-sided motor symptoms, akinetic rigid subtype, severe cognitive decline, advanced disease, longer duration of illness, associated anxiety or psychotic symptoms, younger age, motor disability, higher daily doses of levodopa.[63,65]

Dysfunction of the striatothalamic frontal and basotemporal limbic circuits, disruption in serotonergic and dopaminergic pathways have been implicated in development of depression in PD.[65]

Treatment involves assessing the relationship between depression and the antiparkinsonian drugs being given and adjust their dose accordingly.[63]

- *Mild depression:* Supportive therapy, CBT
- *Moderate depression:*
  - Antidepressants or CBT
  - Tricyclics have more evidences as compared to other antidepressants.
  - TCAs with least sedative and anticholinergic effects such as desipramine and nortriptyline are recommended.
- *Severe depression:*
  - ECT may be considered along with antidepressants.
  - Deep brain stimulation (DBS) and repetitive transcranial magnetic stimulation (rTMS) have also been found effective.[63]

Anxiety

The most common anxiety disorders associated with PD are panic disorder, generalized anxiety disorder (GAD), and social phobia.73 Risk factors include female gender, younger age, young age at onset, severity of illness, comorbid depressive symptoms, sleep disturbances, motor fluctuations, dystonia, and dyskinesias.[63] Noradrenergic dysfunction has a role in pathophysiology of anxiety in PD.[74] These symptoms may occur as a part of off periods where adjustment of antiparkinsonian drugs is required. SSRIs are considered first-line treatment. Benzodiazepines may be used with caution for acute control of symptoms. TCAs, DBS, rTMS, and CBT also have some evidences.[63]

Psychosis

The most common presentation of psychosis in PD is visual hallucinations; benign hallucinosis

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