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THE STUDY ON RAPID EYE MOVEMENT SLEEP DISORDER

MELAM.YESURATNAM [1], KATTA.MADHURI [2], Y.DHARANI [3], D.JAGADEESH [4], SHAIK.RESHMA [5,]

1. MELAM.YESURATNAM. Assistant Professor, Department of Pharmacy Practice, A.M Reddy Memorial college of Pharmacy, Petlurivaripalem, NarasaraoPeta, Palnadu Dist, Pin:522601, A.P.

2,3,4,5,Pharm.D Students, A.M.Reddy Memorial College of Pharmacy,Petlurivararipalem,Narasaraopet, Palnadu-522601, Andhra Pradesh.

ABSTRACT:-

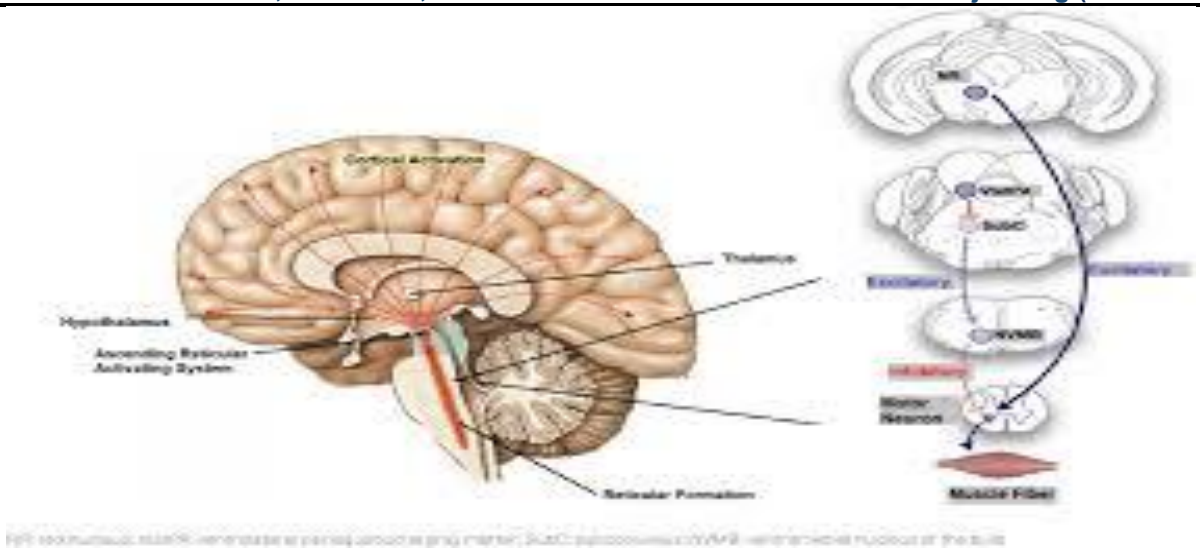
(RBD) occurs when the gross motor paralysis typically associated with REM sleep is intermittently absent. As a result, the sleeper may laugh, yell, kick, punch, or engage in other elaborate behaviors as they respond to dream content. RBD-related behavior typically occurs during the second half of the night, when REM periods are more frequent. These behaviors can often be violent or aggressive, and can result in injury to the patient or to bed partners. RBD occurs almost exclusively in older males,76 and is highly prevalent in Parkinson's disease and related neurodegenerative conditions. In some cases, RBD may signal the onset of one of these neurologic conditions, and may occur years before diagnosis.76,77 In cases of acute onset, RBD has been associated with posttraumatic stress disorder.78 The overall estimated prevalence of RBD is 0.5% in the general population.

Treatment of RBD is primarily pharmacologic, although strategies that decrease the likelihood of injury include placing pillows or cushions around the sleeper's bed, or having partners sleep in another room if behaviors are especially violent, can also be helpful. The pharmacologic treatment of choice is the clonazepam, a sedating benzodiazepine, which has been found to be beneficial for patients with RBD in up to 79% of cases.80 In patients with RBD for whom clonazepam is contraindicated, melatonin has also shown promise in RBD treatment

KEYWORDS:- REM, Behaviour,Frequent,Neurologic,Disorder,Treatment.

Introduction:-

Rapid eye movement behavior disorder (RBD) is a parasomnia involving dream enactment behavior associated with loss of atonia during rapid eye movement (REM) sleep. These symptoms may bring serious harm to the individual themselves and their sleeping partners. RBD has been associated with antidepressant use as well as narcolepsy. The strongest correlation exists between RBD and comorbid neurodegenerative alpha-synucleinopathies (i.e., Parkinson's disease, dementia with Lewy bodies, and multiple system atrophy). Symptoms of RBD may precede neurodegenerative disorders by decades; therefore, a careful history is significant in assessing these patients.

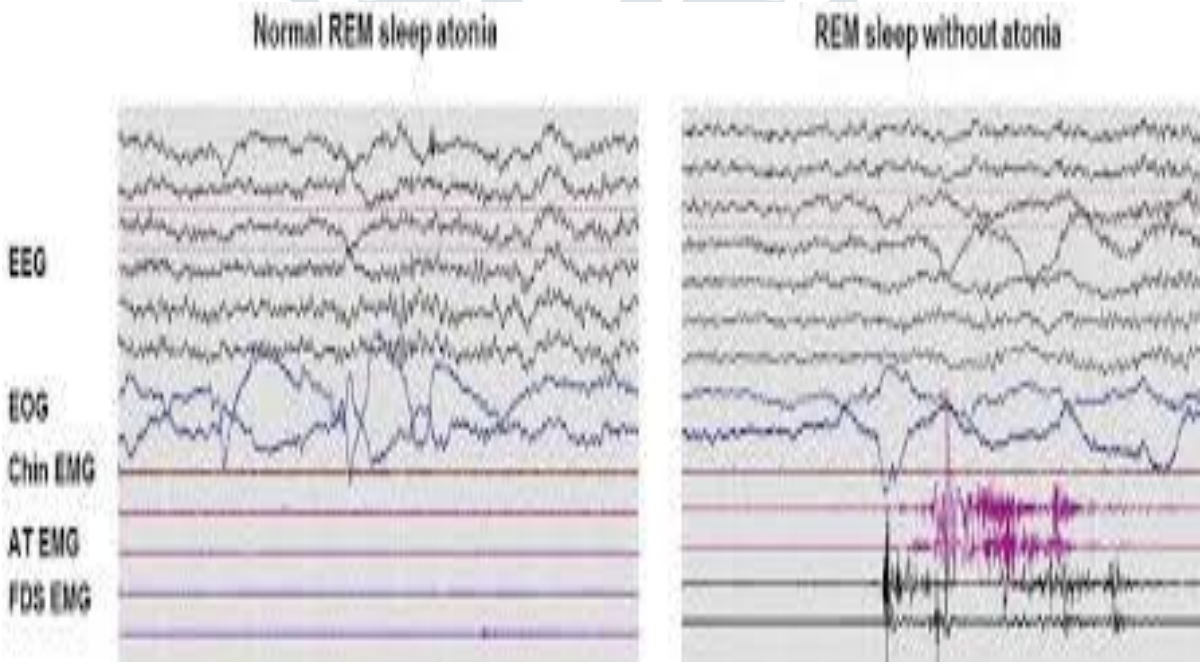


The diagnosis requires confirmation by an in-laboratory sleep study (polysomnography) with video recording, which helps assert abnormal behaviors during REM sleep and excludes other sleep disorders. Counseling and management of RBD focus on injury prevention and the treatment of underlying precipitating disorders in addition to pharmacological treatment of severe cases using oral medications such as melatonin or clonazepam. This topic will review the etiology, epidemiology, pathogenesis, clinical features, evaluation, management, and prognosis of RBD in adults.

Etiology:-

Predisposing factors that increase RBD diathesis include elderly age, male sex, narcolepsy, antidepressant use, and neurological disorders. RBD can be divided into three categories:

Idiopathic RBD is most suggestive in neurodegenerative synucleinopathies, including dementia with Lewy bodies, Parkinson's disease, olivopontocerebellar degeneration, multiple-system atrophy, and Shy-Drager syndrome. The literature suggests that RBD is precipitated by aberrant connections between the brainstem control of muscle tonicity and the cortex. Studies have also suggested associations with traumatic brain injury (TBI), post-traumatic stress syndrome (PTSD), and congenital and neurodevelopmental disorders.



Drug-induced RBD is common in individuals who are taking antidepressants. The most likely antidepressants that will incite an RBD episode are serotonin reuptake inhibitors (fluoxetine), tricyclic antidepressants (mirtazapine, protriptyline, amitriptyline, nortriptyline, desipramine, imipramine), and monoamine oxidase inhibitors (phenelzine and selegiline). Other acute transient forms of RBD involve toxic metabolic encephalopathy—most commonly involving ethanol use.

RBD with concomitant narcolepsy may be considered a distinct phenotype of RBD. It is characterized by less violent or complex behavior during REM sleep, earlier age of onset, equal sex distribution, and hypocretin (orexin) deficiency (a lab diagnosis specific for narcolepsy type

Epidemiology:-

Studies on RBD, although limited in quantity, suggest a prevalence of 0.5% in the general population.The prevalence significantly increases in the elderly population, with RBD presenting between 5% and 13% in adults aged 60 to 99.

Among the elderly population, approximately 60% of cases are idiopathic, while 40% of cases are suggestive of an underlying neurologic disorder.The onset of symptoms is typically seen during the sixth or seventh decade of life. There is a male predominance amongst the older population, but there is an equal distribution between males and females under 50 years.

There is a strong link between RBD and psychiatric disorders. RBD is found to be 5-fold and 10-fold more likely to develop in patients receiving antidepressants and patients with psychiatric disorders, respectively.[16] Some reports linked RBD in childhood to narcolepsy or idiopathic hypersomnia.

RBD has been found in nearly 30 % of young individuals with narcolepsy type I.REM sleep without atonia (RSWA) without clinical symptoms of RBD is not uncommon (2% of the general population) and is more common in those on antidepressants (12%) and older men (25%).[20]

Pathophysiology:-

A prime feature of rapid eye movement behavior disorder is the intermittent loss of atonia during REM sleep, which leads to dreaming-related motor behaviors. Muscle atonia during normal REM sleep is controlled within the pontine tegmentum and medial medulla. Excitatory glutamatergic neurons within the dorsal pre-coeruleus nucleus activate the spinal cord inhibitory interneurons, thereby initiating REM sleep atonia. Animal models and diagnostic imaging of case reports suggesting interruption or disinhibition of these brainstem areas is the pathophysiology of RBD.

The relationship between RBD and neurodegenerative synucleinopathies is very strong, with an estimated conversion rate from RBD to a neurodegenerative syndrome of 6.3% per year and a total of 74% converting after a 12-year follow-up.

The neurodegenerative alpha-synucleinopathies consist of glial cytoplasmic inclusions aggregates of insoluble alpha-synuclein protein. However, whether the link between this neurodegenerative classification and RBD results from these accumulated aggregates or through another pathology is unclear. Animal studies showed that progressive alpha-synuclein aggregation and neuronal apoptosis occurred in the ventricular reticular nucleus in the brainstem and resulted in RBD symptoms in a chronic rat model of Parkinson disease.There was also a strong association described recently between RBD and synucleinopathies in an extensive series of RBD cases that had an autopsy.

While the relationship between RBD and synucleinopathies is more frequently described, other non-synucleinopathies such as progressive supranuclear palsy (PSP), familial amyotrophic lateral sclerosis, frontotemporal dementia, myotonic dystrophy were also reported but less commonly.

RBD has also been associated with Wilson disease, cerebellar degeneration, and autoimmune encephalitis.Some studies suggested that secondary RBD due to paraneoplastic cerebellar degeneration could be immune-mediated, which improved after immunotherapy.

More recently, a novel association between antibodies (mainly IgG4) to a neuronal antigen against IgLON5 (a neuronal cell adhesion molecule) and RBD has been reported suggesting a tauopathy. The more usual presentation includes gait instability followed by dysarthria, dysphagia, ataxia, or chorea, which progress gradually (the median duration between the symptom onset and death is five years).Neural histopathology studies in cases of autoimmune-mediated RBD showed neuronal loss and extensive deposits of hyperphosphorylated tau in the tegmentum of the brainstem and hypothalamus.

The pathogenesis of RBD is distinct in cases of narcolepsy as it is linked to orexin deficiency. In contrast to idiopathic RBD, in cases of RBD-narcolepsy combination, alpha-synuclein biomarkers are usually not detected.

Autoimmune disorder plays an important role in the pathogenesis of RBD (such as in anti-IgLON5 disease).[33] Anti-IgLON5 is a degenerative neurological disorder characterized by neurological (bulbar symptoms, gait abnormalities, cognitive dysfunction) in addition to sleep manifestations (sleep apnea, non-rapid eye movement sleep parasomnia, and RBD.Serotonergic agents can lead to dream enactment.

Stertenrogic RBD can also present in patients with alpha-synuclein neurodegeneration (such as abnormalities in color vision, anosmia, constipation, and motor impairments). These deficits are not well explained by serotonergic mechanisms, suggesting that serotonergic antidepressants may unmask RBD in individuals at risk of underlying neurodegeneration.

History :-

Rapid eye movement behavior disorder exhibits abnormal behaviors during REM sleep that may disrupt sleep and cause injury. Dream enactment is the most distinct abnormal behavior associated with RBD, characterized by purposeful movements during REM sleep with or without vocalization linked to a dream. These sleep-related movements could be violent and lead to self-injuries or injuries to the bed partner. These complex behaviors could result in walking out of bed or falling. Symptoms are usually associated with enacting undesirable or violent dreams where the patient is attacked, chased, or compromised. The patient often wakes up abruptly with prompt alertness and can recount the dream coherently. More than half of the patients are usually aware of their dream-enactment behaviors.

The dream enactment events are variable in frequency (nightly to annually) and usually appear at least 90 minutes after sleep onset and more frequently during the latter half of sleep due to the typical pattern of REM sleep. The eyes are typically closed during an event, and the patient would not normally interact with the environment but rather only behave with the dream. Increased periodic limb movement may occur that may disrupt the sleeping partner.Other sleep behaviors were reported, including stereotypical hand motions, reaching gestures, and punching or kicking.

Acute forms of RBD can be exhibited with intense REM sleep rebound due to withdrawal effects from REM-suppressing drugs. These include alcohol, sedative-hypnotic medications, drug intoxication, or antidepressants. In cases of idiopathic RBD, associated neurological findings could be present such as gait abnormalities or signs of parkinsonism (cogwheel sign, rigidity, or resting tremor).

The cognitive impairments associated with RBD are similar to Parkinson disease and dementia with Lewy bodies.However, the progression of cognitive impairment related to the RBD-PD combination has been reported to be more rapid with less response to treatment than in patients without RBD.

Evaluation:-

The diagnosis of rapid eye movement behavior disorder comprises two features: loss of normal atonia during REM sleep and dream enactment behavior. The suspicion of RBD is usually due to presentation consistent with dream enactment behavior. The diagnosis requires, however, confirmation with in-lab polysomnography (PSG) with video recording.

Polysomnography involves simultaneously recording many physiologic variables, including electroencephalography (EEG); therefore, home sleep tests can not be used. PSG is also helpful in excluding other sleep disorders such as sleep-disordered breathing, seizure, and other non-REM sleep disorders or parasomnia. In cases when abnormal behavior does not occur during the sleep study, REM sleep without atonia is required for the diagnosis. REM sleep without atonia (RSWA) is defined as increased tone during REM sleep using electromyography (EMG) of the chin and/or limb leads (either sustained increased chin EMG during sleep or increased phasic chin or limb twitching).

The presence of isolated RSWA is considered an incidental finding in normal individuals, especially in patients receiving antidepressant therapy (SSRI) excessive caffeine or alcohol, or older men.[20] RSWA is often associated with neurodegenerative disorders, which include idiopathic Parkinson disease, Lewy body dementia, and multiple system atrophy.

The scoring of ESWA requires at least 1 of the following features developed by the American Academy of Sleep Medicine (AASM):

The International Classification of Sleep Disorders, 3 ed., states that diagnostic criteria for RBD must include the following:

Repeated episodes of sleep-related behaviors include vocalization and/or complex motor behaviors.

Documenting behaviors by polysomnography during REM sleep or based on clinical history.

Recordings of polysomnography that demonstrate REM sleep without atonia via submental or limb leads

Behaviors are not better explained by another sleep disorder, mental disorder, medication, substance use, or epilepsy.

Validated questionnaires have been established to screen patients for RBD. Two single-question questionnaires have been established; the Mayo Sleep Questionnaire and the RBD1Q. The Mayo sleep question was tested in the general population and queried the bed partners (sensitivity 98% and specificity 74%); it asks, “Have you ever seen the patient appear to ‘act out his or her dreams’ (punched or flailed arms in the air, shouted, or screamed) while sleeping?”

The RBD1Q was tested in a sleep center and queried the patient directly (sensitivity 98% and specificity 87%); it asks, “Have you ever been told, or suspected yourself, that you seem to ‘act out your dreams while asleep (for example, punching, flailing your arms in the air, making running movements, etc.)?”

Other questionnaires included the 13-item RBDQ-HK (82% sensitivity and 87% specificity), 14-item RBDSQ (96% sensitivity and 56% specificity), and the 5-item Innsbruck Questionnaire (91% sensitivity, 86% specificity).[45][46][47]

Idiopathic RBD is uncommon. Therefore, the positive predictive value of questionnaires with the highest specificity may be low in the general population, which suggests the continued importance of evaluation with polysomnography.

Management:-

The primary treatment goal of rapid eye movement behavior disorder is to reduce the risk of injury to the patient and bed partners. Changing routine sleep habits may prove challenging for the patient and their bed partners. Their risk for injury, however, needs to be emphasized. Patients and sleeping partners should be educated on the following:

Mitigating fall risk by lowering the bed closer to the floor

Safe-guarding any firearms, knives, and other weapons

Cushioning or padding the floor or sharp furniture surfaces.

Placing patients in restraining clothes or sleeping bags

Separating the sleeping partner from the patient to reduce the risk of injury

Based on current guidelines from the American Academy of Sleep Medicine (AASM), the recommended pharmacological treatment of isolated RBD in adults is immediate-release melatonin, clonazepam, or pramipexole (Conditional recommendation). Melatonin is an endogenous hormone secreted from the pineal gland associated with circadian rhythm, and its secretion is influenced by light exposure. Although the mechanism of action is unclear, 3 to 12 mg of melatonin at bedtime is recommended and appears effective in reducing RBD symptoms.

Ramelteon is a melatonin M1/M2 receptor agonist that resembles melatonin FDA FDA-approved for insomnia. Ramelteon at a dose of 8mg at bedtime was studied in an open-label study on individuals with idiopathic RBD. Although there was no significant effect on rates of dream enactment or RSWA, subjective improvement was reported, and no side effects were noted.

Clonazepam is an effective treatment for RBD and has been considered for a long time as first-line pharmacological therapy.Clonazepam is a long-acting benzodiazepine with a half-life of 30 to 40 hours and a peak effect within 1 to 4 hours after ingestion. The recommended initial dose is 0.25 mg 30 minutes before bedtime, and a gradually increased dose to as high as 4 mg has been reported. Studies show that the low dose required for treatment offers a low concern for abuse and tolerance. However, common side effects have been observed, including residual morning sleepiness, increased fall risk, memory dysfunction, impotence, and unstable gait. Responders may note an initial period of symptomatic suppression with the re-emergence of more complex behavior. Caution should be considered in those with dementia, fall risk, and obstructive sleep apnea due to worsening associated symptoms. The efficacy was not confirmed in a randomized control trial as both Clonazepam 0.5 mg and placebo improved RBD symptoms.

Clonazepam can significantly reduce periodic limb movement syndrome but not normalize REM sleep atonia.Other benzodiazepine agents that have been used in RBD include temazepam, triazolam, alprazolam, and the so-called "z-drugs" (such as zopiclone and zolpidem).

Other therapies for isolated RBD include dopaminergic agents (such as L-DOPA and pramipexole), paroxetine, acetylcholinesterase inhibitors (such as donepezil and rivastigmine), desipramine, clozapine, antiepileptic drugs (e.g., carbamazepine, levetiracetam), and antihypertensive medications (e.g., prazosin, clonidine). While these medications may improve nightmares and some motor movements during wakefulness, they are rarely effective in RBD.

In secondary RBD due to medical conditions in adults, the recommended therapy includes immediate-release melatonin, clonazepam, or transdermal rivastigmine (conditional). While it is not recommended to use deep brain stimulation as a treatment for secondary RBD. In older individuals with RBD (isolated or secondary), immediate-release melatonin remains the preferred choice for safety profile.

Other non-pharmacologic treatments under investigation include Plasma exchange and deep brain stimulation. In a study on patients with RBD due to Parkinson disease, deep brain stimulation had a different effect on complex behaviors and RSWA events.

Treating comorbid obstructive sleep apnea with continuous positive airway pressure may decrease the frequency and severity of RBD symptoms. Medication reconciliation is necessary to identify medications prone to inducing loss of REM atonia. Management should be discussed with ordering providers and the patient that may require discontinuation, reduction of dose, or earlier medication intake.

Differential Diagnosis:-

Many conditions may mimic the presentation of rapid eye movement behavior disorder symptoms.[65] Take an appropriate history and conduct in-lab video polysomnography to distinguish the definitive diagnosis. For example, isolated REM sleep without atonia (RSWA) noted incidentally during (as increased EMG chin tone or as twitches in the limbs EMG) could occur in the context of OSA (as known pseudo-RBD).

The twitches in limbs EMG could mimic periodic leg movements (PLMS). However, PLMs are periodic and come as series (≥ four limb movements separated by 5 to 90 seconds), usually occur during non-REM sleep, and are not associated with abnormal behaviors (dream enactment). Dream enactment behavior (DEB) does not exclusively present in patients with RBD. Other manifestations that have been associated with DEB include:

Substance use and/or withdrawal

Benign childhood epilepsy

Complex partial seizures

Confusional arousals

Delirium

Epilepsia partialis continua

Epileptic encephalopathy

Juvenile myoclonic epilepsy

Malingering

Obstructive sleep apnea (OSA)

Parasomnia overlap syndrome

Periodic limb movement disorder

Posttraumatic stress disorder

Psychogenic nonepileptic seizure

Sleep terror

Sleepwalking

Trauma-associated sleep disorder:-

Non-REM sleep parasomnias are common and could mimic RBD, including confusional arousals, sleepwalking, and sleep terrors. These disorders typically occur during childhood in the first half of the night during non-REM sleep without dream recall. However, these clinical findings may be observed concomitantly with objective results of REM without atonia and have been characterized as parasomnia overlap syndrome.

In contrast to RBD, sleep terrors occur predominantly in children and present with sudden screening during sleep that can last for several minutes. Sleep talking can be confused with RBD. However, the tone of voice mimics usual conversation and occurs during NREM and REM sleep. Nightmares are vivid dreams that do not have a motor activity. Other associated parasomnias may include sleep-related eating disorders, sexsomnia, or rhythmic movement disorder.

Trauma-associated sleep disorder has recently been characterized as a novel parasomnia. The patient demographics tend to be younger males with traumatic experiences as the temporal onset of disturbing nocturnal behaviors and nightmares. They may or may not have posttraumatic disorders. Objective findings include REM without atonia. Disruptive nocturnal behaviors captured in the lab are rare.

It is important to differentiate RBD behaviors from nocturnal seizures, including juvenile myoclonic epilepsy, grand mal seizure, benign rolandic, Landau-Kleffner syndrome, and sleep-related hyper motor epilepsy (formerly known as nocturnal frontal lobe epilepsy). Patients with epilepsy may exhibit prodromal symptoms such as mood changes and auras, such as irregular blinking or bladder/bowel control loss. Another differentiating behavior between RBD and seizures is the presentation of postictal states that may appear as confusion, headaches, nausea, temporary neurological deficits, sensory deficits, and/or suppressed alertness following the seizure.

Prognosis:-

The clinical progression of RBD is dependent on the etiology. Idiopathic RBD and RBD associated with neurodegenerative diseases are often slowly progressive. At the same time, medication-induced RBD may occur acutely and improve upon discontinuing the medication. In RBD related to neurodegenerative disorders, the prognosis depends on the underlying condition.

Among those with a confirmed diagnosis of neurodegenerative condition due to an underlying synucleinopathy, it is estimated that the correlation with RBD is significantly high: 30 to 50% of Parkinson disease, 75% of Lewy-body dementia, and 70% to 90% of multiple system atrophy. The prognosis of RBD with Parkinson disease has a higher risk for dementia, and RBD with Lewy-body dementia has a higher mortality risk.

Once RBD has been confirmed, discussing the associated neurodegenerative risks is appropriate if individual patient circumstances are considered and patients are closely monitored for neurodegenerative disorder findings. In these patients, early introduction to neuroprotective therapies should be discussed, including the importance of regular exercise (a total of 120 to 150 minutes/week), which may decrease the risk of Parkinson disease as it does in cardiovascular disease.

Complications:-

The primary complications concerning RBD are the risk of injury and the potential prodromal symptoms linking neurodegenerative alpha-synucleinopathies. The injuries sustained during an event may require immediate medical attention and/or involve sleeping partners causing legal implications. Management for RBD primarily involves injury risk mitigation through behavioral changes and/or pharmacological therapy. Early diagnosis and management of RBD provide a therapeutic window to treat the potential underlying neurodegenerative disorder.

Deterrence and Patient Education:-

Patients with rapid eye movement behavior disorder and their sleeping partners are prone to injuries during their sleep that can result in serious harm. The sleeping partner could target a violent dream enactment behavior leading to the patient being arrested on charges of domestic assault. Therapeutic management should include injury risk mitigation discussions with the patient and their sleeping partner.

It is ethically important to disclose potential risks for the development of neurodegenerative disease to patients diagnosed with RBD. An open discussion with family members allows for improved understanding and avoiding misinformation about the disease process. It is recommended to counsel patients that Parkinson disease and other neurodegenerative disorders can be treatable. The prompt diagnosis of RBD may provide an early therapeutic window for neuroprotective therapies. The International Rapid Eye Movement Sleep Behavior Disorder Study Group is conducting ongoing collaborative studies for symptomatic and neuroprotective treatment.

Pearls and Other Issues:-

After ten years from diagnosis, there is a high risk with rapid eye movement behavior disorder for developing alpha-synucleinopathy pathology.

There are currently no guidelines on counseling and management regarding the potential conversion of RBD to alpha-synucleinopathy. The approach may include a discussion with the patient to understand this risk. The patient should seek consultation from their primary care provider, sleep specialist, or neurologist if associated symptoms manifest, such as memory issues, constipation, gait abnormalities, orthostatic hypotension, or neurological deficits.

Prodromal alpha-synucleinopathy is possible in younger patients less than 50 years old, but nondegenerative disorders should be considered, including autoimmunity, narcolepsy, and REM-suppressing medication use.

There is a lack of controlled, randomized, double-blind studies for RBD treatment. However, injury prevention is critical in the management of RBD. Clonazepam is highly effective in treating RBD to reduce injury risks.

Low risk for adverse reaction favors melatonin over clonazepam as initial pharmacological therapy for RBD.

Other etiology may resemble RBD symptoms and behaviors, necessitating evaluation with a formal PSG to rule out malingering, psychogenic movement disorder, NREM parasomnias, sleep-related epilepsy syndrome, and obstructive sleep apnea. As physicians may be called upon for legal proceedings, this becomes more paramount when an illegal act is presented to occur during sleep.

Enhancing Healthcare Team Outcomes:-

Evaluating and managing newly diagnosed rapid eye movement behavior disorder requires a multi-disciplinary and interprofessional effort involving the patient's primary care provider, a sleep specialist, a neurologist, nursing staff, and pharmacists to maximize these patients' well-being. Injury prevention is paramount and may require both pharmacological and non-pharmacological interventions. The looming prognosis of a neurodegenerative disorder associated with RBD will likely provoke anxiety and mood disruption and require management on its own. Including family members is also a key element of support for these patients.

Clinicians from various disciplines will weigh in from their areas of expertise. Pharmacists must be involved because of the number of possible drug options; they can counsel the patient on their medications and perform medication reconciliation, alerting the prescriber of any interactions or other concerns. Nurses often take patient histories and should inform the clinician of any indications of the patient experiencing rapid eye movement sleep behavior disorder. These interventions require open communication among all team members, which will continue throughout the management process once the patient is diagnosed. Given the challenges of this condition, all interprofessional team members must be free to communicate with any other team member with no barriers to ensure optimal patient care.

Unfortunately, there are no established means of predicting the development of neurodegenerative disease or effective management to slow down the disease process. This makes the discussion with patients on the prognostic timeline and the decision of optimal treatment management truly difficult. Close follow-ups and interprofessional communication are critical.

Future research is needed to identify potential biomarkers and neuroprotective strategies to prevent further neurodegeneration in high-risk individuals.

Risk factors:-

Factors associated with the development of REM sleep behavior disorder include:

- **Being male and over 50 years old** — however, more women are now being diagnosed with the disorder, especially under age 50, and young adults and children can develop the disorder, usually in association with narcolepsy, antidepressant use or brain tumors
- **Having a certain type of neurodegenerative disorder,** such as Parkinson's disease, multiple system atrophy, stroke or dementia with Lewy bodies
- **Having narcolepsy,** a chronic sleep disorder characterized by overwhelming daytime drowsiness
- **Taking certain medications,** especially newer antidepressants, or the use or withdrawal of drugs or alcohol

Recent evidence suggests that there may also be several specific environmental or personal risk factors for REM sleep behavior disorder, including occupational pesticide exposure, farming, smoking or a previous head injury.

Complications:-

Complications caused by REM sleep behavior disorder may include:

- Distress to your sleeping partner or other people living in your home
- Social isolation for fear that others may become aware of your sleep disruption
- Injury to yourself or your sleeping partner

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