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Review Article

Expert opinion on management of alcohol withdrawal syndrome in India

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ABSTRACT

Alcohol dependence is an escalating and pervasive problem worldwide. Alcohol withdrawal syndrome (AWS) is a distressing condition, occurring due to abrupt discontinuation of alcohol in alcohol dependents. It is a common management problem encountered in general hospital settings. The symptoms of AWS range from mild to severe. Proper diagnosis and choice of an appropriate drug regimen for AWS play a key role in effective management. Severe complicated alcohol withdrawal may present with hallucinations, seizures, or delirium tremens. Benzodiazepines (BZDs), considered as the gold standard, have the largest evidence base as treatment for alcohol withdrawal. Other management strategies include use of essential vitamins (as supportive care), anticonvulsants, barbiturates, adrenergic drugs, and gamma-aminobutyric acid (GABA) agonists. Chlordiazepoxide is an effective long-acting benzodiazepine agent and offers a slight advantage over other BZDs. The present review aims to provide an evidence-based consensus on management algorithm for AWS according to the Indian clinical scenario.

Keywords: Alcohol dependence, AWS, Delirium tremens, BZDs, Anticonvulsants

INTRODUCTION

Alcohol dependence is a crucial, multifaceted problem worldwide. The incidence and prevalence of alcohol dependence vary across countries. Consumption of alcohol is one of the leading risk factors for disease and disability in the world, especially with a greater risk in middle-income countries.[1] As per the world health organization, 5.1% of the global burden of disease and injury, which represents 132.6 million disability-adjusted life years is attributable to alcohol use.[2] Harmful use of alcohol results in 3 million deaths every year worldwide, which is equivalent to 5.3% of all deaths.[3] Developing countries like India have seen tremendous upsurge in alcohol consumption especially among younger population.[1] In India, alcohol consumption prevalence ranges from 10% to 60% with a predominant male predilection.[4] The key objective in the management of alcohol dependence is to attain complete abstinence, which can be accomplished by various behavioral and pharmacological strategies. Optimal treatment of alcoholism can be achieved only when the alcoholic is motivated; it comprises of detoxification, rehabilitation, and maintenance of abstinence. Abrupt discontinuation of alcohol in alcohol dependents may result in a distressing and life-threatening condition termed as AWS.[1]

RATIONALE

The objective of consensus document is to provide insight on AWS prevalence, challenges associated with diagnosis and management among the Indian clinical setting.

Expert group meetings involving 88 doctors were conducted across major 9 cities in India in 2021. Evidence-based concept of AWS was discussed and experts' clinical

insights were sought on prevalence, diagnosis, and management strategies of AWS in the Indian scenario.

AWS: CONCEPT AND PREVALENCE

AWS is a cluster of symptoms that arise in alcohol-dependent people, post cessation or reduction in heavy or prolonged alcohol use. The clinical presentation differs from mild to severe and the onset of symptoms usually begins a few hours after the last alcohol intake.[5] The diagnostic and statistical manual of mental disorders (DSM-5) describes AWS as 2 or more distinctive symptoms happening within hours to days following significant reduction in consumption of alcohol after an extended period of heavy drinking. These symptoms comprise of nausea or vomiting, autonomic hyperactivity, insomnia, elevated anxiety and agitation, tremor, perceptual disturbances, and seizures.[6] A minority of 3%-5% patients progress into very severe AWS, including delirium tremens, which involve symptoms like disturbances in various neurotransmitter circuits implicated in the alcohol pathway and reflect a homeostatic readjustment of the central nervous system.[5,6]

Consensus point 1

According to the experts', 3-5 is the approximate number of patients who present with AWS on a weekly basis in Indian clinical practice, with a ratio of 9:1 for men: women. Most patients suffering from severe forms of AWS are between age of 20 and 40 years. Among patients with AWS, a minority of only 5% requires hospitalization, while the rest are treated in the out-patient department.

DIAGNOSIS OF AWS AND ASSOCIATED CHALLENGES

AWS is diagnosed after collection of complete medical history and thorough clinical examination. Diagnosis requires adequate collection of history of the quantity, frequency of alcohol intake, the temporal relation between cessation or reduction of alcohol intake and the onset of withdrawal symptoms (Figure 1).[5] Withdrawal symptoms usually begin 6-24 hours post the last alcohol intake.[7]

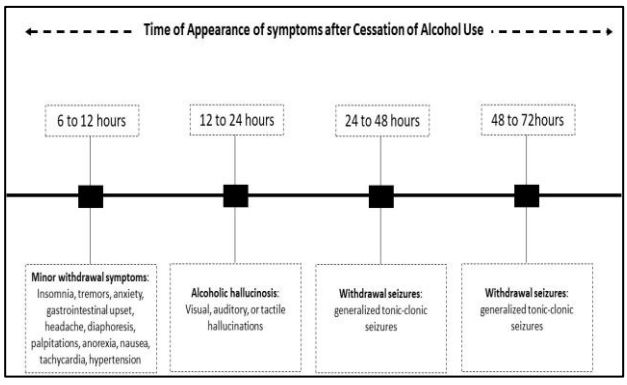

Figure 1: Chronology of alcohol withdrawal syndrome.

If the onset of withdrawal-like symptoms is seen after 1 week of complete cessation of alcohol, then the diagnosis of AWS becomes untenable, irrespective of the amount and severity of alcohol dependence.[5] For establishing a diagnosis of AWS, at least two of the following symptoms should be present, post reduction or discontinuation of alcohol use: autonomic hyperactivity (sweating, tachycardia); increased hand tremor; insomnia; nausea or vomiting; transient visual, tactile, auditory hallucinations or illusions; psychomotor agitation; anxiety; or tonic clonic seizures.[7]

Objective assessment of AWS severity can be done using scale-based measurements. The clinical institute withdrawal assessment of alcohol scale, revised (CIWA-Ar) is one of the most reliable scales used in common clinical practice. It is used to quantity the severity of alcohol withdrawal in a patient diagnosed to have AWS. The CIWA-Ar is a 10-item scale used to measure alcohol withdrawal severity. It is also used to monitor withdrawal and medicate accordingly. Each item is scored by the clinician using a Likert-type scale (mostly ranging 0-7), with a maximum possible total score of 67. The evaluation of the scale is easy and consumes <2 minutes. Scores of 0-8 interpret absent to minimal withdrawal, scores of 9-15 interpret moderate withdrawal, and scores ≥16 interpret severe withdrawal (impending delirium tremens).[8]

In the initial patient assessment of suspected AWS patients, clinical evaluations of elevated heart rate, systolic blood pressure, and temperature are easily verifiable. Evaluating laboratory biomarkers is one of the most important methods for the identification of patients at high risk. To evaluate alcohol consumption in the acute clinical settings, direct and indirect alcohol markers include measurement of ethanol, hypokalemia, thrombocytopenia, ratio aspartate aminotransferase (AST)/alanine aminotransferase (ALT) >2 and mean corpuscular volume. There are even more specific alternatives, which focus on metabolic markers comprising direct products of alcohol degradation and these includes carbohydrate-deficient transferrin, ethylglucuronid, ethylsulfate, phosphatidylethanol, fatty acid ethyl esters, 5-hydroxytryptophol:5-hydroxyindole-3-acetic acid, whole blood acetaldehyde, total sialic acid and homocysteine levels.[9] Moreover, the suspected patients should be evaluated for other underlying disease conditions such as dehydration, infection, cardiac issues, electrolyte abnormalities, gastrointestinal bleeding, and traumatic injury. Due to poor nutritional status, chronic alcoholics may report baseline ketoacidosis, and laboratory findings may show acidemia with ketone production similar to a diabetic patient but with conditions such as euglycemia or hypoglycemia due to lack of liver glycogen stores.[10]

In patients with severe AWS, comorbid or other medical illnesses must be ruled out. This is especially important in patients who have not had a previous history of delirium tremens. Differential diagnoses for severe alcohol withdrawal are hyponatremia, hepatic encephalopathy,

pneumonia, encephalitis/meningitis, head injury, thyrotoxicosis, lithium intoxication, atropine/tricyclic intoxication, psychosis, antidepressant intoxication, subacute encephalopathy with seizures in alcohol use disorders (AUD), status epilepticus, and withdrawal from sedative-hypnotics.[9,10]

Consensus point 2

Experts stated that the diagnosis of alcohol withdrawal can be made after collection of complete history and thorough physical examination. The common AWS presentations noted in patients in the clinics are anxiety, tremors of body and hands, high blood pressure, tachycardia, insomnia, high body temperature, sweating, hallucinations, dilated pupils' nausea, lack of orientation, headache, and grand mal seizures.

The severity of AWS can be classified into three stages: stage 1 (mild) where symptoms are mild and not usually associated with abnormal vital signs; stage 2 (moderate) where symptoms are more intense and associated with abnormal vital signs (e.g., elevated blood pressure, respiration, and body temperature); and stage 3 (severe) that includes delirium tremens or seizures.

Only 5% of the experts stated that they use the CIWA-Ar to assess severity of alcohol withdrawal symptoms in their clinical practice to monitor signs and symptoms of withdrawal and identify patients requiring medical therapy. The remaining panelists opined that the scale is time consuming.

The experts further discussed the significance of ruling out above-mentioned differential diagnosis in AWS management and advised routine laboratory analysis for changes in electrolytes (especially potassium), AST, ALT, mean corpuscular volume, gamma-glutamyl transpeptidase and thrombocytopenia, cardiac issues, or other underlying disease processes. Routine examination should include evaluation of blood (or breath) alcohol concentration, complete blood count, renal function, electrolytes, glucose, liver enzymes, urinalysis, and urine toxicology.

MANAGEMENT OF AWS

AWS is a reason of extreme discomfort to patients; as symptoms are disabling and patients who had experienced withdrawal are scared to stop drinking for fear of acquiring withdrawal symptoms again. The main target of treatment is to lessen the severity of symptoms so as to prevent severe manifestations such as seizure, delirium, and death and to improve patients' quality of life. Additionally, effective AWS treatment should also include efforts in increasing patient motivation to preserve long-term alcohol abstinence. The first-line approach is the non-pharmacologic interventions and sometimes, these are the only approach required in certain patients. It includes frequent reassurance, reality orientation, and nursing care in a quiet room without dark shadows, noises, and other excessive stimuli (i.e. bright lights) is advised. The pharmacological approach of AWS comprises of the use of a long-acting drug as a substitutive agent to be gradually tapered off.[11]

A large number of patients if correctly identified in the early stages of AWS, can be managed on a reduced regime of BZDs without a complicated clinical course. Though, for a minority of patients, consequences are complicated by the development of delirium tremens or Wernicke–Korsakoff syndrome, and those at high risk (moderate-to-severe) will require hospitalization facility to manage them.[12]

BZDs

BZDs are the "gold-standard" in the treatment of AWS. BZDs are the only drug class with proven efficacy in preventing the development of complicated forms of AWS, with a reduction in the incidence of seizures, delirium tremens and associated mortality risk. The efficacy of BZDs in the treatment of AWS appears to be mediated via alcohol mimicking effects that stimulate GABAA receptors.[11] Thus, BZDs substitute the repressive effect of discontinued ethanol in AWS patients. Most BZDs are rapidly absorbed after oral administration, with bioavailability ranging from 80% to 100%.[9]

The ideal drug for AWS treatment should have a rapid onset, long duration of action, wide margin of safety, metabolism independent of liver function, and absence of abuse potential. Several BZDs are effective in preventing agitation and alcohol withdrawal seizures, preventing delirium tremens, and they act as cross-tolerant agents with ethanol. BZDs are effective in the treatment of AWS, owing to the wide margin of safety and low potential to produce physical dependence and tolerance in short-course therapy. Selecting a BZD majorly depends on selection of preferred pharmacokinetic properties in relation to the patient being treated. Chlordiazepoxide, diazepam (long acting) and lorazepam, and oxazepam (short/intermediate acting) are the most commonly used BZDs for alcohol detoxification.[8]

Chlordiazepoxide

Chlordiazepoxide, a long-acting BZD, is an approved medication for patients with AWS.[13] Due to its long half-life of 24-48 hours, blood levels are reasonably uniform across the course of the day, thereby lowering the risk of withdrawal symptoms, including seizures that may arise when blood levels drop. An added advantage of the long half-life is the less likelihood of occurrence of drug discontinuation reactions.[14] A landmark study by Kaim et al proved that chlordiazepoxide is better than chlorpromazine, hydroxyzine, thiamine, or placebo in preventing seizures and delirium tremens in patients with AWS.[15] Chlordiazepoxide ranks among the safer of the effective psychopharmacologic BZD compounds.[13]

Consensus point 3

The panelists agreed that BZDs are currently recognized as the first-line treatment for AWS, with chlordiazepoxide and lorazepam as the preferred choice of agents. The likelihood of multiple administration routes viz., oral, intramuscular, or intravenous signifies an advantage of BZDs. For moderate to severe AWS, intravenous route should be preferred because of the fast onset of action, while the oral route can be used for mild and even moderate forms of AWS. Long-acting agents like chlordiazepoxide offers a smooth course of treatment without the risk of rebound symptoms which occur late during withdrawal therapy, as blood levels are considerably uniform across the day. However, the long half-lives, and the presence of active metabolites results in drug accumulation in patients with liver disease. Hence, short-acting BZDs like lorazepam are preferred option in patients with severe liver dysfunction and in patients at high risk of experiencing serious medical consequences following sedation, viz. severe lung disease or elderly patients. The advantage of lorazepam is that it has no active metabolites and its bioavailability is not largely affected by liver metabolism. Although short-acting BZDs are effective, they are linked with greater symptom rebound risk, hence these drugs are given in gradually decreasing /tapering doses before they can be discontinued.

Dosage regimens for BZDs

Three regimens for alcohol detoxification using BZDs are most commonly followed, namely loading dose regimen, fixed tapering dose regimen, and symptom-triggered regimen:

Loading dose regimen /front loading: These regimens usually use long-acting BZDs to reduce complication risks such as seizures and delirium. Administration of 20 mg diazepam oral loading dose, every 2 hours was found useful in treating AWS. Before every dose, the withdrawal severity and the clinical condition need to be monitored.[8]

Fixed tapering dose regimen: Fixed dose regimens of BZDs are given at scheduled intervals irrespective of symptom severity. According to the presenting severity of withdrawal and the time since last intake, the starting doses are decided. This regimen is best suitable for out-patient settings where close monitoring is not feasible.[8]

Symptom triggered regimen: BZDs may be administered as per the presence of withdrawal symptoms as assessed by withdrawal rating scales e.g. CIWA- AR. The ratings are assessed at fixed schedules, and drug doses are given as per withdrawal severity. Administration of these regimens requires training in applying scales and trained personnel. This type of dosing regimen is preferred in most cases of AWS as it results in the administration of less medication and requires shorter duration of treatment. This regimen may also lower the risk of undermedication or overmedication because dosing is based upon withdrawal symptoms.[8]

American society of addiction medicine (ASAM) guidelines recommend symptom-triggered treatment by trained staff as the preferred BZD dosing methodology, at short-term observational settings with continuous monitoring. Front loading while under clinical supervision or fixed dosing with additional, as-needed medication are also considered appropriate. Front loading is recommended for patients experiencing severe AWS.[16]

Consensus point 4

A "fixed-dose" regimen, rather than a "loading dose" or a "symptom-triggered" regimen was the preferred dosing adopted by most experts in Indian clinical practice. With the fixed-dose regimen, the chosen drugs are diazepam (10 mg once daily [QID] for 1 day, followed by 5 mg QID for 2 days and then tapering off) and chlordiazepoxide (50-100 mg QID for 1 day, followed by 25-50 mg QID for 2 days, with subsequent tapering off). These are given at regular intervals independent of symptoms, and are tapering off by 25% per day from day 4 to day 7. If symptoms are not adequately controlled, additional doses can be administered. Fixed-dose regimen is highly effective and can be ideal in patients at risk for severe AWS or in patients with history of seizures or delirium tremens. However, patients should be monitored carefully for the risk of excessive sedation and respiratory depression.

The loading-dose approach needs administration of moderate-to-high doses of long-acting BZDs (i.e. diazepam 10-20 mg or chlordiazepoxide 100 mg, every 1-2 hours) in order to induce sedation; drug levels will successively decrease (auto-taper) via metabolism. In the early phase of the treatment, the risk of BZD toxicity is high, hence strict clinical monitoring is required to prevent toxicity. However, this approach yields a shorter treatment course unlike the progressive auto-tapering in other regimen and promotes recovery from AWS by reducing the incidence of severe AWS.

Anticonvulsants

Though BZDs are the drugs of choice for AWS in most treatment settings, anticonvulsant drugs represent suitable alternatives. There are various potential advantages of using anticonvulsants. Administration of an anticonvulsant lowers the likelihood of a patient experiencing withdrawal seizure, thereby reducing AWS complications. Anti-convulsant drugs also reduce craving and have shown to block kindling in brain cells.

They do not appear to have abuse potential, and are effectively used to treat mood disorders, which share some symptoms with AWS, such as depression, irritability, and anxiety. The inclination of anticonvulsant drugs to cause sedation is much less as that of BZDs.[8]

Compared to oxazepam, carbamazepine is superior in ameliorating global psychological distress and reducing aggression and anxiety. It is an effective alternative to BZDs in the treatment of mild to moderate AWS patients. Carbamazepine is also effective in decreasing the craving for alcohol after withdrawal. Carbamazepine is better than BZDs in preventing rebound withdrawal symptoms and reducing post-treatment alcohol consumption, viz., in patients with multiple repeated withdrawals. However, use of it is limited due to its interaction with various medications that undergo hepatic oxidative metabolism, which makes it less helpful in elderly patients and patients with comorbidities.[8]

Valproic acid significantly impacts the course of alcohol withdrawal and diminishes the need for treatment with a BZD. Studies have shown that patients treated with valproic acid for 4 to 7 days drop out less frequently, have less severe withdrawal symptoms such as fewer seizures, and require less oxazepam than patients receiving either carbamazepine or placebo. Though valproic acid is effective, its use is limited due to medication side effects, such as somnolence, gastrointestinal disturbances, confusion, and tremor-which are comparable to AWS, making assessment of improvement complicated.[8]

Gabapentin has structural similarity to GABA and is effective in the treatment of alcohol withdrawal. Gabapentin was found to be as effective as lorazepam in a randomized, double blind controlled study involving 46 in-patients with acute mild to moderate AWS. Its low toxicity makes it a promising agent. Vigabatrin, an anticonvulsant agent, irreversibly blocks GABA transaminase, and improves withdrawal symptoms within three days of treatment.[8]

Consensus point 5

Carbamazepine, gabapentin, or phenobarbital are appropriate for patients contraindicated for BZDs. Carbamazepine, gabapentin, or valproic acid (for patients with no liver disease or women with childbearing potential) may be used as adjunct to BZDs in the mild AWS.

OTHER MEDICATIONS

Although baclofen is effective in reducing AWS symptoms and reducing the risk of relapse, overall data are diverse. As adjunctive therapy, beta blockers and the alpha-adrenergic agonist clonidine lower adrenergic symptoms, but do not prevent occurrence of seizures. For outpatient treatment of AWS, phenothiazines and barbiturates are not recommended. Moreover, phenytoin is ineffective in the treatment or prevention of seizures.[7]

Consensus point 6

Adrenergic medicines, which alter the function of adrenergic receptors, are thought to significantly improve symptoms of AWS and are used as adjuncts to BZDs in the management of AWS. Barbiturates such as phenobarbitone act via GABA pathways, are cross tolerant to alcohol and can ease withdrawal symptoms significantly. However, there is a lack of controlled clinical studies to demonstrate that these agents can prevent occurrence of seizures or delirium tremens. Additionally, barbiturates have a narrow therapeutic index, i.e. the difference between the minimum doses required for a therapeutic effect and the dose at which the agents become toxic is very narrow, as compared to BZDs; hence, the barbiturates are not used in the common practice. Baclofen helps in relief of severe withdrawal symptoms. Baclofen also lowers craving in the alcohol-dependent patients.[8]

Adjunctive supplements

Chronic alcohol use is correlated with reduction of body stores of thiamine, magnesium and niacin. Chronic thiamine deficiency may translate into a classical triad of confusion, ataxia, and ophthalmoplegia, better known as Wernicke's encephalopathy (WE).[5] WE is distressed with high morbidity and mortality and is prevalent only in rare cases. Principally, in severe AWS with predominant symptoms of delirium tremens, differentiation from WE is impossible. Due to its easy and uncomplicated treatment, prevention of WE with parenteral thiamine prior to administration of parenteral carbohydrate-containing fluids should be considered for all patients at risk, including those experiencing AWS. The sooner thiamine supplementation is started, the faster is the recovery, regardless of initial clinical presentation.[9] Typical dosing is 100 mg intravenous/intramuscular or oral per day for 3-5 days.[16] As a result of chronic malnutrition and gastric malabsorption that proceeds chronic alcohol abuse, many clinicians recommend multivitamin supplements (B1 + B2 + B6 + nicotinamide + vitamin C) in parenteral form for the initial 3-5 days.[15]

Chronic use of alcohol is also associated with abnormal metabolism and absorption of magnesium and niacin. As a result, these patients are deficient in vitamins and electrolytes, such as magnesium and niacin. Hence, their deficiencies need appropriate correction. Intravenous multivitamin injections in addition to magnesium replacement should be given if symptoms are present or administered prophylactically severe AWS cases.[5]

Consensus point 7

Experts stated that due to the higher likelihood of nutritional deficiencies in AWS patients, nutritional support should be provided, as per tolerability. Thiamine supplementation prevents WE development, and a dose of 100 mg daily should be recommended to all patients orally for mild AWS and intravenously for moderate-severe AWS. Multivitamin injections should also be given to AWS patients.

COMORBIDITIES ASSOCIATED WITH AWS

AUD or alcohol dependence is one of the most prevalent comorbidities in people with mental illness. Common psychiatric comorbidities associated are depression, schizophrenia, bipolar disorder, and psychoses. Antidepressant medications are available to treat depressive illness, with the selective serotonin reuptake inhibitors (SSRIs) being regarded as a first-line pharmacologic treatment.[17] The selection of medication for managing psychotic or affective symptoms in people with psychotic disorders may have effect on alcohol consumption. First-generation antipsychotic medications do not report a reduction in alcohol use and may increase substance use and craving in people with schizophrenia. Long-acting injectable formulations of second-generation antipsychotics, as well as clozapine, a second-generation antipsychotic, may be considered as a preferred choice.[18] In bipolar disorders, anticonvulsants, viz., valproate, carbamazepine and lamotrigine, are the first line alternatives to lithium for a long-term use.[19]

Consensus point 8

Experts mentioned that antipsychotics such as olanzapine and risperidone, should be prescribed as preferred drugs in psychiatric symptoms and haloperidol in aggressive psychiatric symptoms. The SSRI sertraline at 15 to 100 mg is doses given as first line treatment and serotonin-norepinephrine reuptake inhibitor (SNRI) as second-line treatment in depressive patients. Anticonvulsants used are carbamazepine as first-line treatment and sodium valproate as second-line treatment in epilepsy.

NEWER MEDICATIONS IN AWS

Many of the recently developed drugs in AWS are being used as adjuncts to BZDs. The N-methyl-D-aspartate antagonist ketamine reduces BZD requirements and is well tolerated at low doses. Though there is insufficient published data, it is still a promising new agent. Levetiracetam which has also been tried in clinical trials have resulted in negative outcomes. A review found that sodium oxybate and sodium salt of gamma-hydroxybutyric acid are useful options for the treatment of AWS. Dexmedetomidine is a drug that acts on the noradrenergic system and is currently used in the United States for the treatment of AWS in an emergency clinical setting. It may lower the need for BZDs and proposed to be a promising and effective adjuvant treatment for AWS.[8]

MANAGEMENT ALGORITHM FOR AWS

AWS is a frequent consequence of sudden alcohol cessation in patients with moderate to severe alcohol dependence syndrome.[20] Table 1 illustrates how to proceed in the clinical setting of suspected AWS to confirm the diagnosis and to start sufficient therapy. Mild AWS may not need pharmacotherapy in most cases. The patient needs supportive care in a calm and quiet environment and observation for a period of up to 36 h, after which the patient is unlikely to develop withdrawal symptoms. In the presence of risk factors such as a history of significant alcohol withdrawal symptoms, high levels of recent drinking, a history of withdrawal seizures or delirium tremens, and the co-occurrence of a serious medical or psychiatric illness, diazepam 20 mg loading dose is recommended. In moderate AWS, patients should be started immediately on a symptom-triggered dosing regimen, along with monitoring the withdrawal severity (CIWA-Ar ratings) and clinical signs and symptoms. In severe AWS patients, it is advised to initiate with loading dose regimen and eventually shift to symptom-triggered regimen based on the withdrawal severity scoring.[8,9]

Table 1: Management algorithm for AWS.

Step	Actions	
1	Physical examination and AWS investigation according to DSM-5	
2	Exclusion of differential diagnosis	
3	Laboratory investigation to support suspected diagnosis of AWS	
4	Confirmation of AWS diagnosis	
5	CIWA-AR questionnaire-based assessment to objectify severity of AWS	
6	Targeted therapy based on severity of symptoms	
	Mild AWS	**Moderate to severe AWS**
	Supportive care in a calm and quiet environment	Monitor patient
	Use of questionnaires to score symptoms	Use of pharmacotherapy (BZDs and others as per symptoms and other comorbidities) Moderate AWS: Symptom-triggered treatment Severe AWS: Initiate with loading dose and switch to symptom-triggered treatment when goal is achieved
	Pharmacotherapy, if needed (in presence of risk factors)	Use of questionnaires to score symptoms, in regular intervals

AWS-alcohol withdrawal syndrome, BZDs-benzodiazepines, CIWA-AR-Clinical institute withdrawal assessment for alcohol revised, DMS-5-Diagnostic and statistical manual of mental disorders-5th Edition.

CONCLUSION

AWS is a syndrome caused by sudden cessation or reduction of alcohol intake in people with AUD. AWS carries significant morbidity and mortality by itself, and the syndrome may complicate co-morbid treatment of

medical illness. The clinical presentation of alcohol withdrawal can often be mild; however, serious complications and death are certainly possible. Providing care for a patient who is experiencing alcohol withdrawal is very challenging. While BZDs have long been the mainstay of AWS treatment, novel approaches have garnered increasing evidence and acceptance. Clinicians must recognize their vital role not only in treating life-threatening withdrawal but also in setting the patient on the path towards recovery. AWS are best monitored by regular scale-based assessments such as CIWA-Ar but as panelists opined that the scale is time consuming, we need to develop a simpler scale for diagnosis which can be used in routine clinical practice. There is room in the research body for further exploration into separate screening checklist or criteria for physicians or GPs and for the Psychiatrists that can be looked. Clinicians knowledgeable about alcohol use and withdrawal need to be flexible in modifying management strategy towards patient care in light of evolving clinical conditions.

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REFERENCES

1. Girish K, Vikram Reddy K, Pandit LV, Pundarikaksha HP, Vijendra R, Vasundara K, et al. A randomized, open-label, standard controlled, parallel group study of efficacy and safety of baclofen, and chlordiazepoxide in uncomplicated alcohol withdrawal syndrome. Biomed J. 2016;39(1):72-80.
2. Glantz MD, Bharat C, Degenhardt L, Sampson NA, Scott KM, Lim CCW et al. The epidemiology of alcohol use disorders cross-nationally: Findings from theWorld Mental Health Surveys, Addictive Behaviors. 2018.
3. Alcohol. World health organization. Available at: https://www.who.int/news-room/fact-sheets/detail/alcohol, Accessed on 10 October 2024.
4. Jagadeesan S, Patel P. Epidemiology, pattern and prevalence of alcohol consumption in India: need for public health action. Int J Community Med Publ Health. 2021;8(4):2070-6.
5. Sachdeva A, Choudhary M, Chandra M. Alcohol Withdrawal Syndrome: Benzodiazepines and Beyond. J Clin Diagn Res. 2015;9(9):VE01-7.
6. Wolf C, Curry A, Nacht J, Simpson SA. Management of Alcohol Withdrawal in the Emergency Department: Current Perspectives. Emerg Med. 2020;12:53-65.
7. Muncie HL Jr, Yasinian Y, Oge' L. Outpatient management of alcohol withdrawal syndrome. Am Fam Physician. 2013;88(9):589-95.
8. Jan MM, Majeed N, Mufti S, Margoob MA. Management of patients with alcohol withdrawal syndrome. The Int J Indian Psychol. 2020;8(1):DIP:18.01.135/20200801.
9. Jesse S, Bråthen G, Ferrara M, Keindl M, Ben-Menachem E, Tanasescu R, et al. Alcohol withdrawal syndrome: mechanisms, manifestations, and management. Acta Neurol Scand. 2017;135(1):4-16
10. Newman RK, Stobart Gallagher MA, Gomez AE. Alcohol Withdrawal. 2021 May 29. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2021.
11. Mirijello A, D'Angelo C, Ferrulli A, Vassallo G, Antonelli M, Caputo F, Leggio L, Gasbarrini A, Addolorato G. Identification and management of alcohol withdrawal syndrome. Drugs. 2015;75(4):353-65.
12. Sinclair JMA, O'Neill A. Update on the management of alcohol use disorders. B J Psych Adv. 2020;26:82-91.
13. Ahwazi HH, Abdijadid S. Chlordiazepoxide. 2020 Nov 22. In: StatPearls. Treasure Island (FL): StatPearls Publishing. 2021.
14. Kumar CN, Andrade C, Murthy P. A randomized, double-blind comparison of lorazepam and chlordiazepoxide in patients with uncomplicated alcohol withdrawal. J Stud Alcohol Drugs. 2009;70(3):467-74.
15. Kattimani S, Bharadwaj B. Clinical management of alcohol withdrawal: A systematic review. Ind Psychiatr J. 2013;22(2):100-8.
16. Alvanzo A, Kleinschmidt K, Kmiec JA, Kolodner G, Marti GE, et al. The ASAM Clinical Practice Guideline on Alcohol Withdrawal Management. 2020. Available at: https://www.asam.org/docs/default-source/quality-science/the_asam_clinical_practice_guideline_on_alcohol-1.pdf?sfvrsn=ba255c2_2, Accessed on 13 August 2024.
17. DeVido JJ, Weiss RD. Treatment of the depressed alcoholic patient. Curr Psychiatry Rep. 2012;14(6):610-8.
18. Archibald L, Brunette MF, Wallin DJ, Green AI. Alcohol Use Disorder and Schizophrenia or Schizoaffective-Disorder. Alcohol Res. 2019;40(1):arcr.v40.1.06.
19. Grunze H, Schaefer M, Scherk H, Born C, Preuss UW. Comorbid Bipolar and Alcohol Use Disorder-A Therapeutic Challenge. Front Psychiatry. 2021;12:660432.
20. Cooney G, Heydtmann M, Smith ID. Baclofen and the Alcohol Withdrawal Syndrome-A Short Review. Front Psychiatry. 2019;9:773.

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