

Acute altered sensorium in a young industrial worker with suspected polysubstance intoxication and possible occupational inhalational exposure: A case report

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ABSTRACT

Acute altered sensorium in an adolescent or young adult poses a broad differential diagnosis that includes recreational drug intoxication, substance abuse, hypoglycemia, occupational inhalational exposure, and less common causes such as viral encephalitis and non-convulsive seizures. We report the case of a 19-year-old male industrial worker who presented to the emergency department with disorientation, incoherent speech and relative bradycardia, with no focal neurological deficit. Initial work-up revealed hyperglycemia, transient hyponatremia and sinus bradycardia with right axis deviation on electrocardiography. Based on a broad initial differential, the clinical picture evolved to favor a working diagnosis of suspected cannabis intoxication, layered on a probable alcohol-use pattern and possible occupational inhalational exposure, pending confirmatory toxicology. The patient recovered fully with supportive care, empirical high-dose parenteral thiamine and psychiatric liaison. The case illustrates the diagnostic challenge posed by suspected polysubstance and occupational exposure in young workers and the value of a structured, hedge-appropriate diagnostic approach in the absence of immediate toxicological confirmation.

Key words: Altered sensorium, Occupational inhalational exposure, Substance abuse, Suspected cannabis intoxication, Wernicke encephalopathy, Young adult

Acute alteration of consciousness in young adults is a frequent and challenging presentation in the emergency department (ED). In India, it is often multifactorial, involving substance use, metabolic derangement, and less commonly, occupational inhalational exposure [1,2]. Cannabis (*Cannabis sativa*), known colloquially in India as ganja or marijuana, terms used here only for context and not elsewhere in this report, is the most widely abused illicit substance in India after alcohol, and its prevalence among young urban workers is rising steadily [3]. Although cannabis intoxication is rarely fatal by itself, it may present with a spectrum of neuropsychiatric manifestations including disorientation, incoherent speech, paranoia, and conjunctival injection [4]. Acute presentations are frequently complicated by concurrent alcohol consumption, which may mask the presenting features and delay diagnosis [5].

Young industrial workers are additionally at risk of exposure to volatile hydrocarbons, organic solvents

and refrigerant gases, all of which can produce an encephalopathy clinically indistinguishable from that of cannabis intoxication [6]. Hypoglycemia, hyponatremia, viral encephalitis, non-convulsive seizures, carbon monoxide poisoning, toxic alcohol ingestion and traumatic brain injury must also be considered. A structured approach, history from attenders, examination for stigmata of substance use, targeted laboratory workup, neuroimaging and psychiatric opinion, is therefore essential [7].

We describe a case of acute altered sensorium in a 19-year-old industrial worker who presented a diagnostic challenge because of overlapping features of possible occupational inhalational exposure, suspected cannabis intoxication, and a probable alcohol-use pattern. We report this case because it illustrates a diagnostic overlap that is, in our experience, under-represented in the Indian case-report literature: The simultaneous consideration of recreational substance intoxication and occupational toxicological exposure in a young worker managed in a resource-limited ED setting. The case is intended to reinforce the importance of a broad, appropriately

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hedged differential diagnosis; the rationale for empirical parenteral thiamine as low-risk supportive therapy; and the role of multidisciplinary (emergency medicine, internal medicine and psychiatry) evaluation in such presentations, pending toxicological confirmation.

CASE REPORT

A 19-year-old male, a daily-wage industrial worker, was brought to the ED of a tertiary-care teaching hospital at 13:35 h with a history of altered behavior noticed by his brother approximately 6 h before admission. The attender reported that the patient had been incoherent, disoriented, and shouting intermittently since the morning. There was no history of trauma, fever, seizure, vomiting at the time of arrival, or prior neurological illness. Collateral history obtained from the brother was as follows: The patient had a history of cannabis use, 1–2 times per week, of unknown quantity, with the last reported intake approximately 3 days before admission. He also had a history of alcohol consumption, 1–2 times per week in unknown quantity, with the last bout again approximately 3 days prior. He was an intermittent smoker, consuming approximately 2–3 cigarettes on the days he smoked (about 1–2 days per week) for an estimated preceding period of 2 years. This corresponds to an average daily consumption of well under 1 cigarette/day and, using the standard formula (pack-years = [cigarettes per day ÷ 20] × years smoked), a cumulative exposure of approximately 0.03–0.05 pack-years. The exact duration and pattern of smoking should, however, be confirmed if possible. There was no known past psychiatric history, no chronic medical comorbidity, no regular medication, and no family history of psychiatric illness.

The patient had been employed for approximately 8 months as a helper in a small-scale industrial unit involving solvent-based adhesives and surface-coating/painting work. His reported duties involved daily 8-h shifts with regular handling of solvent-based adhesives and paints, plausibly containing aromatic hydrocarbons such as toluene and xylene. The workspace was reported by the attender to be poorly ventilated, without a local exhaust ventilation system. The patient did not routinely use respiratory protective equipment, apart from occasional use of a simple cloth mask. Two co-workers were reported to have experienced intermittent headache and dizziness during shifts in the preceding weeks but had not sought medical attention. This history, although obtained from collateral source and not independently verified with the employer, raises the possibility of a chronic low-grade solvent exposure background against which an acute event occurred.

On examination, the patient was conscious but not oriented to time, place or person, non-coherent and non-cooperative. He was afebrile (temperature 98.4°F), pulse rate 74 beats/min, blood pressure 110/70 mmHg, respiratory rate 18/min, and peripheral oxygen saturation (SpO₂) 98% on room air. The Glasgow Coma Scale (GCS)

was E4V4M5 at initial assessment (total 13). Pupils were 3 mm bilaterally, equal and reactive to light. There were no signs of head injury, no icterus, no cyanosis, and no clubbing. Cardiovascular and respiratory examinations were within normal limits; the abdomen was soft and non-tender. Central nervous system examination revealed no focal neurological deficit, symmetrical deep tendon reflexes, and flexor plantar responses bilaterally. There was no meningeal irritation.

The provisional working diagnoses at admission were: (i) altered sensorium secondary to suspected cannabis intoxication, considered together with possible occupational inhalational exposure to an unidentified industrial solvent; (ii) polysubstance use; and (iii) hypoglycemia, considered as an immediately reversible cause pending bedside glucose testing. Hypoglycemia was subsequently excluded once the random blood sugar (RBS) returned markedly elevated at 291 mg/dL (see Investigations below), and this diagnosis was removed from the active differential at that point. A high-risk consent was obtained from the attender, explaining the possibility of respiratory failure, aspiration pneumonia, seizures, need for mechanical ventilation, and sudden cardiac events.

Baseline investigations are summarized in Table 1. Laboratory evaluation revealed a RBS of 291 mg/dL, hemoglobin 14.9 g/dL, total leukocyte count 4,300/cumm (with relative neutrophilia on subsequent samples), platelet count 1.9 lakhs/cumm, blood urea 28 mg/dL, and serum creatinine 0.6 mg/dL. Initial serum sodium was 138 mmol/L; this fell transiently to 128 mmol/L on day 3 before returning to 133 mmol/L. Serum and urine osmolality and urine sodium were not measured, and syndrome of inappropriate antidiuretic hormone secretion (SIADH) could therefore not be biochemically confirmed. Liver function tests (serum glutamic oxaloacetic transaminase 19, serum glutamic pyruvic transaminase 10, alkaline phosphatase 35 U/L, total bilirubin 0.3 mg/dL, albumin 3.8 g/dL) were normal. Urinalysis showed pus cells 2–4/HPF, epithelial cells 1–2/HPF and a 3+ glucose result; no ketones. Human immunodeficiency virus, hepatitis B surface antigen and hepatitis C virus serology were non-reactive.

Twelve-lead electrocardiography (ECG) at admission (Fig. 1) demonstrated sinus bradycardia with a ventricular rate of approximately 58–60 bpm and right axis deviation, with no ischemic changes or QTc prolongation. Repeat ECG after 24 h (Fig. 2) showed a return to normal sinus rhythm. Chest X-ray in the posteroanterior view showed clear lung fields; two-dimensional (2D) echocardiography and ultrasonography of the abdomen and pelvis were unremarkable. Non-contrast computed tomography (CT) of the brain was normal. Cerebrospinal fluid analysis, electroencephalography (EEG) and carboxyhemoglobin measurement, was not performed, as clinical suspicion for encephalitis, non-convulsive seizure and carbon monoxide poisoning was low at the time. A urine drug screen for cannabinoids, opioids, benzodiazepines,

Table 1: Trend of key laboratory parameters during hospital stay

Parameter (unit)	Day 1 (23/01)	Day 2 (24/01)	Day 3 (26/01)	Day 4 (27/01)	Day 5 (28/01)
Haemoglobin (g/dL)	14.9	14.0	14.0	13.8	—
TLC (cells/mm ³)	4,300	6,560	6,900	7,900	—
Platelets (lakh/mm ³)	1.9	2.2	1.85	2.05	—
RBS (mg/dL)	291	—	—	—	—
Blood urea (mg/dL)	28	—	11	22	23
Serum creatinine (mg/dL)	0.6	0.7	0.6	0.7	0.6
Sodium (mmol/L)	138	134	128	134	133
Potassium (mmol/L)	4.1	4.0	3.6	4.2	4.4
Chloride (mmol/L)	96	101	107	94	98
SGOT/SGPT (U/L)	19/10	22/12	19/12	—	—
Total bilirubin (mg/dL)	0.3	0.3	0.3	—	—

TLC: Total leukocyte count, RBS: Random blood sugar, SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamic pyruvic transaminase. Serum/urine osmolality and urine sodium were not measured (see Discussion and Limitations)

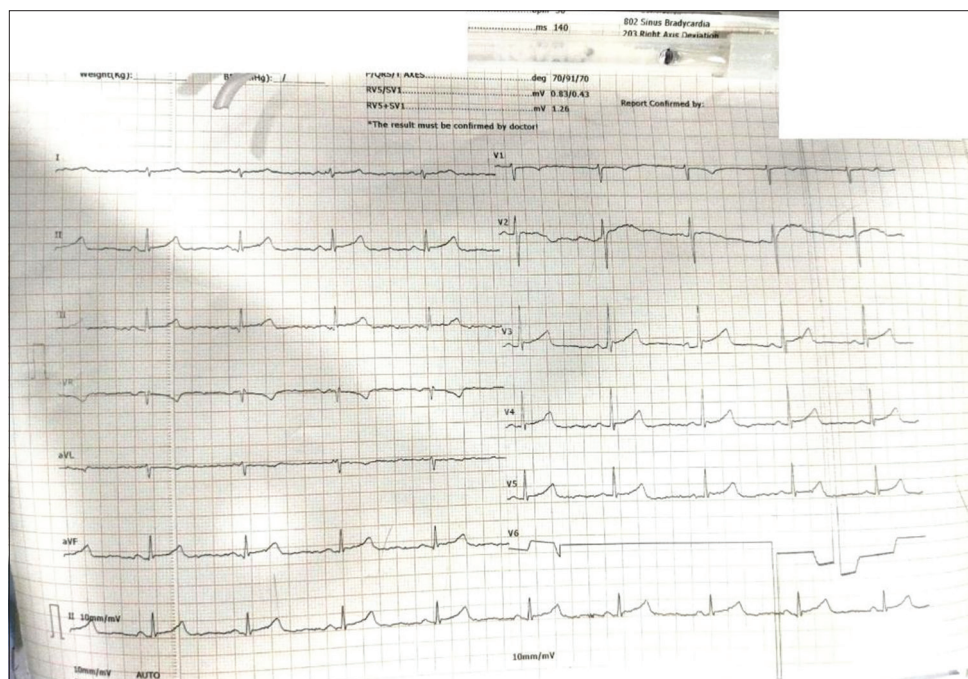


Figure 1: Twelve-lead electrocardiogram at admission showing sinus bradycardia (rate ~58 bpm) with right axis deviation, no ischaemic ST-T changes, and a normal QTc interval. bpm: Beats per minute, QT: QT interval, ST: ST segment



Figure 2: Follow-up twelve-lead electrocardiogram 24 h later showing return to normal sinus rhythm at 72 bpm, with resolution of the axis deviation. bpm: Beats per minute, QT: QT interval, ST: ST segment

amphetamines, and barbiturates, as well as a blood ethanol level, was sent to investigate the etiology. This screen does not test for synthetic cannabinoids, which cannot be excluded based on a negative standard panel alone.

The patient was managed in the medical intensive care unit with a standard supportive protocol (Table 2): continuous oxygen supplementation to maintain $\text{SpO}_2 \geq 94\%$, intravenous fluid with 10% dextrose in normal saline at 50 mL/h, intravenous thiamine 300 mg in 100 mL normal saline 3 times daily (TID) as empirical, low-risk supportive cover for possible Wernicke encephalopathy in the context of probable chronic alcohol use (administered ahead of dextrose-containing fluids), intravenous pantoprazole 40 mg twice daily (BD), intravenous ondansetron 4 mg TID, intravenous lorazepam 1 mg as required (PRN) for agitation, and hourly monitoring of vitals, two-hourly monitoring of GCS and capillary blood glucose (GRBS), and six-hourly ECG monitoring. He was maintained nil-per-oral initially and then advanced to slow oral feeds once the sensorium improved. Multivitamin supplementation (Optineuron 1 ampoule once daily) was continued.

A psychiatry opinion was sought in view of the suspected substance-abuse spectrum. On initial mental status examination, the patient was disoriented and intermittently shouting, with incoherent speech; formal assessment of mood and thought content could not be completed owing to non-cooperation, and no hallucinatory behavior was witnessed by staff. Cognition could not be formally tested at this stage. In the absence of a reliable first-hand history, the working psychiatric impression was suspected cannabis intoxication with possible underlying alcohol-use disorder, pending formal evaluation. Once the sensorium normalized (day 3), a structured substance-use assessment was planned using standard screening instruments (CAGE and AUDIT) to characterize the pattern and severity of alcohol and

cannabis use; the patient was also monitored clinically for alcohol-withdrawal features using the Clinical Institute Withdrawal Assessment for Alcohol, revised, and none developed. Before discharge, the patient and his attender received psychoeducation regarding substance use and its risks, and a formal referral was made to the hospital's de-addiction counseling service for outpatient follow-up.

The patient's sensorium improved progressively over 48 h. By day 3, he was conscious, coherent, and oriented, with normal vital parameters and a GCS of 15/15. He was subsequently shifted to the general ward and counseled regarding substance use. He was discharged on day 5 in a hemodynamically and neurologically stable condition, with advice for outpatient psychiatric follow-up, de-addiction counseling, and avoidance of further cannabis and alcohol use, together with a recommendation for occupational health assessment of his workplace given the possible inhalational exposure history.

DISCUSSION

This case illustrates the diagnostic complexity of acute altered sensorium in a young industrial worker and highlights three important teaching points. First, the differential diagnosis of altered sensorium in this age group is broad, and suspected cannabis intoxication, though common, remains a diagnosis of exclusion in the ED until confirmed toxicologically [3,4]. Second, possible inhalational exposure to industrial compounds is an under-recognized cause of encephalopathy in young Indian workers, and a detailed occupational history is therefore mandatory [6,8]. Third, empirical parenteral thiamine may reasonably be considered as low-risk supportive therapy in a patient with altered sensorium and a probable history of chronic alcohol consumption, even when classical Wernicke features are absent, though this should not be interpreted as evidence of a confirmed Wernicke diagnosis.

Table 2: Summary of supportive management in the intensive care unit

Sr. No.	Intervention	Dose/route	Indication/rationale
1	Oxygen supplementation (nasal cannula)	Titrated to $\text{SpO}_2 \geq 94\%$	Maintain oxygenation; precaution against aspiration
2	IV 10% dextrose-normal saline	500 mL at 50 mL/h	Maintain hydration; glucose already confirmed elevated, so infusion was not intended to treat hypoglycaemia
3	IV thiamine (vitamin B ₁)	300 mg in 100 mL NS, TID	Empirical, low-risk supportive cover against possible Wernicke encephalopathy
4	IV pantoprazole	40 mg BD	Gastric protection in stressed, NPO patient
5	IV ondansetron	4 mg TID	Nausea/vomiting prophylaxis
6	IV lorazepam	1 mg PRN	Agitation control; also alcohol-withdrawal cover
7	Multivitamin injection (Optineuron)	1 ampoule IV OD in 100 mL NS	Nutritional supplementation
8	Continuous cardiac/ECG monitoring	6-hourly 12-lead+telemetry	Monitor arrhythmia, QTc prolongation
9	GRBS monitoring	2-hourly	Detection of recurrent hypo-/hyperglycaemia
10	GCS monitoring	2-hourly	Neurological trend
11	Vitals monitoring (PR, BP, SpO_2)	Hourly	Haemodynamic surveillance

NS: Normal saline, OD: Once daily, BD: Twice daily, TID: Three times daily, PRN: As required, IV: Intravenous, GRBS: General random blood sugar, GCS: Glasgow Coma scale, PR: Pulse rate, BP: Blood pressure, SpO_2 : Peripheral oxygen saturation

A systematic consideration of alternative diagnoses is essential before attributing this presentation to any single cause. Viral encephalitis was considered less likely given the absence of fever and meningismus and a normal non-contrast CT brain; however, cerebrospinal fluid analysis was not performed, and this diagnosis cannot be formally excluded, a limitation of this report. Toxic alcohol ingestion (methanol/ethylene glycol) was not directly excluded biochemically, as an anion-gap/osmolar-gap calculation and specific toxin levels were not obtained; the absence of visual symptoms and metabolic acidosis on available electrolytes makes this less likely but does not exclude it definitively [9]. Synthetic cannabinoid exposure is an important consideration, particularly as standard urine drug screens (including the one sent in this case) do not reliably detect synthetic cannabinoids; this possibility therefore cannot be excluded based on a negative or pending standard panel. Carbon monoxide poisoning was considered unlikely given a normal SpO₂ and no history of exposure to combustion sources, although a normal pulse oximetry reading does not reliably exclude carboxyhaemoglobinemia, which was not directly measured. Non-convulsive seizures were considered less likely in the absence of witnessed convulsive activity and given progressive spontaneous improvement, but EEG was not performed and cannot be used to formally exclude this diagnosis. Metabolic encephalopathy secondary to hepatic or uremic causes was considered unlikely given essentially normal renal and liver function; hypoglycemic encephalopathy was excluded by the markedly elevated admission RBS.

Cannabis is the most widely used illicit substance in India, with the 2019 National Survey on Extent and Pattern of Substance Use estimating that 2.8% of Indians aged 10–75 years had used a cannabis product in the past year [3]. Acute cannabis intoxication typically presents within minutes to hours of inhalation, with a clinical syndrome of euphoria followed by disorientation, incoherent speech, perceptual distortions, conjunctival injection, tachycardia, and less commonly, bradycardia, hypotension, and even syncope [4,10]. Acute psychosis, anxiety and paranoid ideation are recognized with higher-potency preparations [4]. In the present case, the patient presented with a clinical picture broadly consistent with suspected cannabis intoxication, disorientation, incoherence, and relative bradycardia, though the classical conjunctival injection and drug-seeking behavior were absent, and confirmatory toxicology remained pending at the time of writing.

An important diagnostic consideration in a young industrial worker is possible inhalational exposure to volatile organic compounds. Solvents such as toluene, xylene, and halogenated hydrocarbons produce an acute encephalopathy syndrome that can closely mimic cannabis intoxication [6,8]. Chronic exposure may cause cerebellar signs, cognitive impairment, and hepatic dysfunction. In the present case, the patient worked at

an industrial facility with reported use of solvent-based adhesives and paints under poorly ventilated conditions and minimal respiratory protection (see occupational history above); the exact chemical agents and cumulative exposure could not be independently verified, and this aspect warrants further investigation, ideally through direct occupational-hygiene assessment, in similar future presentations. Occupational health services should play a larger role in the work-up of such young workers.

The sinus bradycardia and right axis deviation observed on admission, with full resolution within 24 h, are of uncertain but plausibly functional significance. While acute cannabis intoxication is more classically associated with tachycardia, bradycardia and transient conduction/axis changes have also been described, particularly with higher doses or with concurrent vagal predominance [9,10]. The transient, fully reversible nature of the ECG changes in this case favors an autonomic or functional mechanism over structural cardiac disease, but this correlation remains hypothesis-generating rather than conclusive, and serial troponin measurements or further electrophysiological evaluation were not available.

Concurrent alcohol and tobacco use is common among young cannabis users in India, and a probable pattern of polysubstance use was evident in our patient [3,5]. This raises the practical issue of Wernicke encephalopathy prophylaxis, though we emphasize that the classical triad of ophthalmoplegia, ataxia and confusion, present in fewer than 10% of patients with biopsy-proven Wernicke encephalopathy, was absent in our patient, and no formal diagnosis of Wernicke encephalopathy was made [9]. Parenteral thiamine is inexpensive, safe, and effective; delayed administration, especially if glucose-containing fluids are given first, may precipitate or worsen an evolving Wernicke syndrome in at-risk patients. For this reason, thiamine was administered empirically as a precautionary, low-risk supportive measure ahead of dextrose-containing fluids, consistent with a cautious rather than diagnostically confirmed approach [9].

The transient hyponatremia (nadir 128 mmol/L on day 3) seen in our patient is noteworthy but cannot be definitively attributed to a specific mechanism. Cannabis use has been associated with SIADH and with water intoxication in some reports [11]; alternatively, the hyponatraemia may have reflected inappropriate free-water administration during early resuscitation. Because serum/urine osmolality and urine sodium were not measured in this case, SIADH could not be biochemically confirmed, and this should be regarded as a plausible hypothesis rather than an established mechanism. The electrolyte abnormality was corrected with fluid restriction and switching to isotonic saline, without requiring hypertonic saline.

From a management perspective, this case reinforces the value of multidisciplinary care. Emergency physicians, intensivists, and psychiatrists need to work

Table 3: Comparison with previously published literature

Feature	Present case	Typical acute cannabis intoxication (Volkow <i>et al.</i> , [4]; Hollister [10])	Occupational solvent-related encephalopathy (Filley <i>et al.</i> , [6]; Rath <i>et al.</i> , [8])	Cannabis-associated hyponatraemia (Asplund <i>et al.</i> , [11])
Population	19-year-old male industrial worker	Variable age; often young adults	Occupationally exposed workers, variable age	Young adults, case-series level evidence
Onset	Acute (hours)	Acute (minutes–hours after use)	Acute or chronic, depending on exposure pattern	Acute, following heavy water intake and cannabis use
Key features	Disorientation, incoherence, relative bradycardia, transient hyponatraemia, transient ECG changes	Euphoria, disorientation, tachycardia (more common than bradycardia), conjunctival injection	Encephalopathy, cognitive impairment; chronic exposure may add cerebellar/hepatic features	Hyponatraemia/water intoxication in the context of cannabis use
Confirmatory testing	Pending at time of writing	Urine cannabinoid screen	Occupational exposure history/hygiene assessment; solvent levels where available	Serum/urine osmolality, urine sodium (obtained in reported cases)
Outcome	Full recovery with supportive care	Generally self-limiting; rarely fatal alone	Variable; may become chronic with continued exposure	Resolved with fluid management in reported cases

This table synthesises previously published, generally described features from the cited literature for qualitative comparison; it is not a systematic review, and exact figures from individual studies should be verified by the author against original sources before final submission. ECG: Electrocardiography

together; a structured psychiatric liaison is especially important to bridge the transition from acute management to de-addiction counselling [7,12]. Formal toxicological confirmation (urine drug screen, blood alcohol) should be pursued in every such case, as it has both clinical and medicolegal implications, and should ideally precede, rather than follow, firm diagnostic labelling in the medical record. Comparison with previously published literature is shown in Table 3.

This report has several limitations. Confirmatory toxicology (urine drug screen for standard substances, blood ethanol level) was pending at the time of writing, and the diagnosis of cannabis intoxication therefore remains presumptive rather than confirmed. Standard urine drug screens do not detect synthetic cannabinoids, which cannot be excluded. Serum/urine osmolality and urine sodium were not measured, limiting biochemical confirmation of SIADH as the mechanism of hyponatraemia. Cerebrospinal fluid analysis, EEG and carboxyhaemoglobin level were not obtained, limiting formal exclusion of viral encephalitis, non-convulsive seizures, and carbon monoxide poisoning, respectively. The occupational exposure history relies on collateral report from the attender and could not be independently verified with the employer or through workplace hygiene assessment. Finally, substance-use history in general relied on collateral rather than first-hand report, given the patient's non-cooperation at presentation. Despite these limitations, the case adds to the Indian literature on suspected polysubstance and occupational-exposure presentations in young workers and may help emergency physicians anticipate similar diagnostic challenges.

CONCLUSION

Acute altered sensorium in young industrial workers should prompt a broad, systematically considered

differential diagnosis that includes suspected cannabis intoxication, polysubstance use, occupational inhalational exposure, and important alternative diagnoses such as viral encephalitis, non-convulsive seizures and toxic-metabolic causes. A structured clinical approach, empirical low-risk parenteral thiamine where alcohol use is probable, and early psychiatric liaison form reasonable pillars of initial management. Clinicians managing young adults with unexplained altered sensorium, particularly those in industrial occupations, should maintain a broad differential rather than prematurely anchoring on a single diagnosis, and should pursue timely toxicological confirmation before attributing the presentation definitively to any single agent. De-addiction counselling and occupational safety review are essential components of follow-up to reduce the risk of recurrence.

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