DOI: https://doi.org/10.54393/pjhs.v7i1.3481

PAKISTAN JOURNAL OF HEALTH SCIENCES
(LAHORE)
https://thejas.com.pk/index.php/pjhs
ISSN (E): 2790-9352, (P): 2790-9344
Volume 7, Issue 01 (January 2026)

Original Article

OPEN ACCESS

Clinical Practice of Treating Benzodiazepine Poisoning with Kahwa (Black Tea) in Acute Cases of Overdose

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ARTICLE INFO

Keywords:
Altered Consciousness, Benzodiazepine, Flumazenil, Intubation, Kahwa

How to Cite:
Khan, J., Khan, M. I., Khan, M. A., Sultan, O., Khan, A., & Zahra, D. (2026). Clinical Practice of Treating Benzodiazepine Poisoning with Kahwa (Black Tea) in Acute Cases of Overdose: Benzodiazepine Poisoning with Kahwa (Black Tea) in Acute Cases of Overdose. Pakistan Journal of Health Sciences, 7(1), 76-82. https://doi.org/10.54393/pjhs.v7i1.3481

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Received Date: 11th September, 2025
Revised Date: 3rd December, 2025
Acceptance Date: 2nd January, 2026
Published Date: 31st January, 2026

ABSTRACT

Recently, an increasing trend of using black tea (Kahwa) in many diseases has been noticed due to its numerous health benefits with less to no side effects and easy availability. **Objectives:** To describe the clinical characteristics, management practices, and short-term outcomes of patients with acute benzodiazepine overdose who received Kahwa as part of supportive care. **Methods:** This retrospective descriptive study was conducted at the National Poison Control Center, Jinnah Postgraduate Medical Center, Karachi. Patients aged 13–70 years with confirmed acute benzodiazepine overdose were included using a non-probability consecutive sampling technique. All patients received standard supportive care plus enteral feeding of Kahwa via nasogastric tube (NG). Data were analyzed using IBM-SPSS version 26.0, and significance was set at $p<0.05$. **Results:** A total of 200 patients with acute benzodiazepine overdose were analyzed. There were 50.5% patients who were male, with an overall median age of 24 years (IQR: 20–38). In most cases, 75.5% presented within two hours of ingestion, and 81.5% had taken ten or fewer tablets. Hypertension and diabetes were present in 4.5% and 4.0% of patients, respectively. The most common presenting features were altered consciousness (25.0%) and non-reactive pupils (27.5%). Intubation was required in 2 patients (1.0%), and flumazenil was given to 3 (1.5%). **Conclusions:** This study documents current clinical practice in managing benzodiazepine overdose, including adjunctive Kahwa administration. The results are descriptive and do not indicate the therapeutic effect of Kahwa specifically, as no causal interpretation is possible.

INTRODUCTION

Data from the developed world have highlighted an increased ratio of deaths in the last two decades due to benzodiazepine (BZD) overdose [1, 2]. A study involving 38 states of the US documented a drastic increase of over 500% in mortality secondary to BZD overdose, in just a duration of one year (2019 to 2020) [1]. Benzodiazepine overdose, most commonly involving agents such as diazepam, lorazepam, clonazepam, or alprazolam, typically causes central nervous system depression manifesting as drowsiness, ataxia, confusion, and, in severe cases, respiratory depression or coma [1, 2]. To date, flumazenil has been the mainstay antidote of BZD overdose as it antagonizes BZD activity by binding competitively to the extracellular surface of GABAA receptors [3]. However, Flumazenil should not be used regularly in cases of mixed overdose, prolonged QRS interval, epilepsy, and BZD tolerance, which warrant the trials of better and safer alternatives. Recently, an increasing trend of using black tea (also known as Kahwa) in many diseases has been noticed due to its numerous health benefits with less to no side effects and easy availability [4-6]. According to a recent report, 75-78% of the total worldwide tea

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production and consumption is of black tea [7, 8]. It is predominantly consumed in Western and Asian countries while various constituents such as flavonoids (catechins, TFs and TRs), methylxanthines (caffeine), phenolic acids (CGA, CA, GA and cauramic acid), lipids, proteins, β-carotene, amino acids (theanine), carbohydrates, volatile compounds-fluoride, folate and traces of vitamins C, K, A are available [8-10]. There have been a few reports highlighting the cytoprotective and anti-apoptotic effects of polyphenols, which are found in black tea [11, 12]. It is also known to have been beneficial in the prevention and treatment of hypercholesterolemia and heart disease [10]. Taking all these mechanisms of action and benefits into account, black tea usage in cases of acute BZD overdose is in trial phases. There are no side effects of using Kahwa.

While black tea (Kahwa) has demonstrated cytoprotective and anti-apoptotic effects in preliminary studies, its potential role in the supportive management of acute BZD overdose remains largely unexplored. Limited evidence exists on its safety, efficacy, and impact on short-term clinical outcomes in this context. Therefore, this study aims to describe the clinical characteristics, management practices, and short-term outcomes of patients with acute benzodiazepine overdose who received Kahwa as part of supportive care.

METHODS

This retrospective descriptive study was based on a review of patient medical records, Emergency Department case files, and hospital admission registers maintained at the National Poison Control Center, Jinnah Postgraduate Medical Center (JPMC) Karachi, Pakistan. The study analyzed data collected between August 2023 to April 2024. Approval was obtained from the Institutional Review Board of Jinnah Postgraduate Medical Center (JPMC) (letter no. F.2-81/2023-GENL/98/JPMC). All data were obtained exclusively from existing hospital documentation, including Emergency Department triage sheets, physician admission notes, nursing charts, toxicology assessment forms, medication administration records, and laboratory reports maintained in the medical files of each patient at the National Poison Control Center, JPMC. No new data were collected directly from patients or attendants, consistent with the retrospective nature of the study. The Poison Control Center is a tertiary care facility specializing in the management of acute toxicological emergencies, serving a diverse population from urban and semi-urban areas of Karachi and neighbouring districts. As this was a retrospective review of existing clinical records with no direct patient interaction, the requirement for informed consent was waived by the IRB, in accordance with institutional policy and relevant national ethical guidelines. The sample size of 200 patients represented all eligible cases of acute benzodiazepine overdose admitted during the study period. As this was a retrospective descriptive study, no prospective sample size calculation was performed. However, this sample provided sufficient precision to describe key clinical patterns and allowed post-hoc verification showing >80% statistical power for detecting medium effect sizes (Cohen's $d \approx 0.4$) at $\alpha = 0.05$, using G*Power sample size calculator. Inclusion criteria were patients of either gender, aged 13 to 70 years, and a clinical history consistent with acute benzodiazepine ingestion, confirmed either by self-report, reliable informant history, or supporting laboratory findings where available. Patients were excluded if they had a pre-existing diagnosis of neurodegenerative or age-related brain diseases (such as Alzheimer's or Parkinson's disease), known malignancies, chronic systemic illnesses (such as chronic liver, kidney, or heart disease), genetically determined disorders, or any documented familial diseases that could confound neurological assessment or recovery patterns. Cases involving polypharmacy overdose where other central nervous system depressants or stimulants were involved were also excluded to ensure study homogeneity. A non-probability consecutive sampling technique was employed. All eligible patient records meeting the predefined criteria within the study window were included. Clinical details at presentation were recorded, including the duration since time of overdose (in hours), quantity of pills ingested, and presenting complaints (such as altered consciousness, respiratory depression, or seizures). The presence of any positive danger signs, such as unresponsiveness, respiratory failure, or shock, was specifically noted. The Glasgow Coma Scale (GCS) score at admission was documented, along with an assessment of vital stability upon arrival (including blood pressure, heart rate, and respiratory rate)[13]. As per the clinical protocols at the Poison Control Center, patients diagnosed with acute benzodiazepine overdose received standard supportive care, which included airway protection, monitoring of vital signs, intravenous fluids as necessary, and the administration of activated charcoal where indicated. The diagnosis of acute benzodiazepine overdose was established based on a combination of clinical history, presentation, and, where available, toxicological confirmation. In most cases, ingestion was confirmed through patient self-report or reliable informant history, supported by circumstantial evidence such as empty medication strips or prescription records. Laboratory confirmation of benzodiazepine ingestion was available for 62 (31.0%) patients, performed using urine immunoassay screening at the JPMC toxicology laboratory. The remaining cases were classified as clinically consistent with benzodiazepine overdose based

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on characteristic symptoms (e.g., CNS depression, hyporeflexia, slurred speech) and exclusion of other toxicological causes. In addition to standard management, patients received Kahwa administered enterally via nasogastric tube (NG) as part of the supportive treatment regimen. Kahwa was prepared according to a standardized in-hospital recipe traditionally used at the center as a stimulant beverage. The preparation consisted of green tea leaves (Camellia sinensis, approximately 2–3 g per 150 mL serving) brewed in hot water (90–95 °C) for 5–7 minutes, with optional additions of cardamom pods (1–2) and cinnamon (a small stick) for flavor. No sugar or milk was added. Each serving contained an estimated caffeine content of 25–35 mg per cup, comparable to mild green tea [14]. Patients were given Kahwa orally once they were clinically stable and able to swallow safely, typically one to two cups within the first 6 hours of admission. All patients received standard supportive management according to institutional protocols. Airway protection and gastric decontamination were prioritized upon arrival. Activated charcoal (50 g single dose) was administered via nasogastric (NG) tube in patients presenting within two hours of ingestion, provided they had an intact or protected airway and no contraindications such as altered mental status without intubation [15]. Patients who were fully conscious and able to swallow safely received charcoal orally (PO) instead. The timing and route of administration were documented in clinical charts. Activated charcoal was not given beyond two hours post-ingestion. Supportive care also included intravenous fluids, monitoring of vital signs, and oxygen supplementation when indicated. No gastric lavage or additional pharmacologic interventions were routinely performed other than flumazenil in selected cases. Flumazenil was administered only in selected cases presenting with marked central nervous system depression (GCS ≤ 8) or respiratory compromise, in accordance with institutional toxicology protocols. It was avoided in patients with a history of chronic benzodiazepine use, seizure disorder, or suspected mixed-drug ingestion. Administration was carried out before any adjunctive therapy, including Kahwa, to ensure standard care was not delayed. In all cases, Kahwa was administered only after airway stabilization and completion of essential interventions, including gastric decontamination and, where indicated, flumazenil administration. The total duration of hospital stay (in days) and the final clinical outcome were recorded. The study did not assess the safety or efficacy of Kahwa as a pharmacological intervention. All patients received standard supportive care, and Kahwa was administered as a traditional institutional adjunct. No causal inference or pharmacodynamic interaction analysis was undertaken due to the retrospective, descriptive design. A special proforma was designed to record all relevant study data. Data analysis was performed using IBM-SPSS Statistics, version 26.0. Normality of quantitative variables was assessed using the Shapiro–Wilk test, and homogeneity of variances was evaluated using Levene's test. For normally distributed variables with equal variances, the independent t-test or one-way ANOVA was applied as appropriate. When assumptions were not met, the Mann–Whitney U or Kruskal–Wallis tests were used. Effect sizes (Cohen's d for t-tests and η^2 for ANOVA) were calculated and interpreted qualitatively as small (<0.2), medium (0.2–0.5), or large (>0.5). A Bonferroni correction was used to adjust for multiple comparisons due to its conservative control of type-I error for small subgroup analyses. P-value<0.05 was taken as significant.

RESULTS

Out of these, 101 (50.5%) were male, and 99 (49.5%) were female, indicating a nearly equal gender distribution. The median age of the study population was 24.0 years ([IQR: 20.0–38.0 years), with the youngest patient aged 13 and the oldest 70 years. About comorbidities, hypertension was present in 9 (4.5%) and diabetes mellitus in 8 (4.0%) patients. There were 151 (75.5%) presented within two hours of benzodiazepine ingestion. The median duration since overdose was 2.0 hours (IQR: 2.0–2.0), ranging from 1 to 72 hours. There were 163 (81.5%) patients who reported ingestion of ten or fewer tablets, with a median pill quantity of 10 (IQR: 10–10), while 37 (18.5%) had consumed more than ten tablets. The number of pills ingested ranged from 3 to 50. The Glasgow Coma Scale (GCS) at presentation was less than 12 in 34 (17.0%) patients (Table 1).

Table 1: Demographic and Clinical Characteristics of Patients (n=200)

Characteristics		Frequency (%)
Gender	Male	101 (50.5%)
	Female	99 (49.5%)
Age Groups (Years)	13-20	58 (29.0%)
	21-30	71 (35.5%)
	31-40	34 (17.0%)
	41-50	12 (6.0%)
	51-60	15 (7.5%)
	61-70	10 (5.0%)
Comorbidities	Hypertension	9 (4.5%)
	Diabetes Mellitus	8 (4.0%)
Drug Addict	–	8 (4.0%)
Duration Since Time of Overdose (Hours)	≤2	151 (75.5%)
	>2	49 (24.5%)
Quantity of Benzodiazepine Pills	≤10	163 (81.5%)
	>10	37 (18.5%)

DOI: https://doi.org/10.54393/pjhs.v7i1.3481

Glasgow Coma Scale	<12	34 (17.0%)
	≥12	166 (83.0%)
Vitally Stable at the Time of Reception	Yes	147 (73.5%)
	No	53 (26.5%)

Among danger signs, the most common finding was the presence of mid-dilated, non-reactive pupils, observed in 55 (27.5%) cases. Regarding presenting complaints, altered level of consciousness was the most frequent, reported in 50 (25.0%) patients, followed by disorientation or confusion in 36 (18.0%), vomiting in 34 (17.0%), and stupor in another 34 (17.0%). A detailed breakdown of danger signs and clinical presentations is depicted (Figure 1).

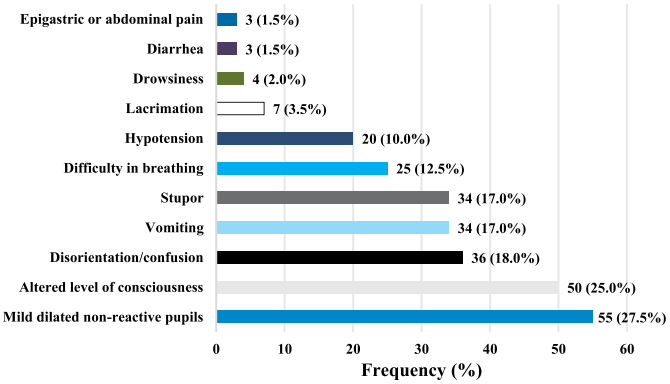

Figure 1: Frequency of Danger Signs and Presenting Complaints/Features (n=200)

Intubation was required in only 2 (1.0%) patients due to respiratory compromise. Flumazenil administration was required in 3 (1.5%) patients. Serial GCS monitoring and precise time-to-awakening data were not routinely documented in patient charts. However, the majority of patients demonstrated rapid clinical improvement, as reflected by a short mean hospital stay (1.36 ± 0.68 days), ranging 1-4 days, and the absence of mortality or severe neurological sequelae (any persistent neurological impairment observed during hospitalization or at discharge, including prolonged unresponsiveness, post-hypoxic encephalopathy, cognitive dysfunction, or new-onset seizures). Most regained full consciousness within 24 hours of admission based on clinical progress notes. There were no deaths reported among the studied subjects. The mean duration of hospital stay was significantly longer in males compared to females (1.5 ± 0.8 vs. 1.2 ± 0.5 days, p=0.001). Increasing age was associated with a progressive rise in hospital stay, with those aged 51–60 years experiencing the longest admissions (2.0 ± 0.8 days, p<0.001). Patients with diabetes mellitus had a significantly longer mean hospital stay than those without diabetes (1.9 ± 0.8 vs. 1.3 ± 0.7 days, p=0.027). A lower GCS score (<12) at presentation was associated with prolonged hospitalization (1.9 ± 0.9 vs. 1.2 ± 0.5 days, p<0.001). Patients who were vitally unstable at presentation had significantly longer duration of hospitalization compared to those who were vitally stable (1.6 ± 0.8 vs. 1.3 ± 0.6 days, p=0.004) (Table 2).

Table 2: Association of Demographic and Clinical Characteristics with Duration of Hospitalization (n=200)

Characteristics		Duration of Hospital Stay (Days)	Effect Size	p-value
Gender	Male	1.5 ± 0.8	d=0.42	0.001
	Female	1.2 ± 0.5		
Age Groups (Years)	13-20	1.2 ± 0.5	n^2=0.14	<0.001
	21-30	1.2 ± 0.4		
	31-40	1.5 ± 0.8		
	41-50	1.8 ± 0.9		
	51-60	2.0 ± 0.8		
	61-70	1.9 ± 0.9		
Hypertension	Yes	1.8 ± 0.9	d=0.61	0.056
	No	1.3 ± 0.5		
Diabetes Mellitus	Yes	1.9 ± 0.8	d=0.75	0.027
	No	1.3 ± 0.7		
Drug Addict	Yes	1.5 ± 0.8	d=0.14	0.539
	No	1.4 ± 0.7		
Duration Since Time of Overdose (Hours)	≤2	1.4 ± 0.7	d=0.14	0.413
	>2	1.3 ± 0.7		
Quantity of Pills	≤10	1.4 ± 0.7	d=0.32	0.196
	>10	1.2 ± 0.5		
Glasgow Coma Scale	<12	1.9 ± 0.9	d=0.97	<0.001
	≥12	1.2 ± 0.5		
Vitally Stable at the Time of Reception	Yes	1.3 ± 0.6	d=0.41	0.004
	No	1.6 ± 0.8		

d = Cohen's d; n^2=Eta-squared

All patients received supportive therapy as the mainstay of management, consistent with standard practice in toxin ingestions. Only 3 patients (1.5%) in this series were administered flumazenil, the benzodiazepine receptor antagonist, and notably, this was done after an initial trial of Kahwa. While most patients recovered with supportive management and adjunctive Kahwa, the retrospective nature of this study precludes any inference of causality between Kahwa use and clinical improvement. No caffeine-related adverse effects were documented among the studied patients. There were no reports of tachycardia, arrhythmia, new-onset seizures, or agitation temporally associated with Kahwa administration.

DISCUSSION

All the patients involved in this research received enteral Kahwa dose via NG as mentioned, along with the rest of the supportive treatment. The sparing use of flumazenil reflects its well-known controversial role in overdose, and hence, with the mentioned approach of including Kahwa in initial supportive therapy, we were able to limit the need for flumazenil [16, 17]. A prior Pakistani study reported zero flumazenil use in 281 benzodiazepine poisoning cases,

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likely for these reasons [18]. Patient outcomes in this study were overwhelmingly positive, as no deaths occurred. This is congruent with the known benign course of isolated benzodiazepine overdose, as fatalities are rare when benzodiazepines are the only substance involved [19]. Benzodiazepine-induced respiratory depression is usually not life-threatening unless compounded by other CNS depressants or underlying pulmonary compromise [20]. Even massive ingestions of benzodiazepines alone are often survived with supportive care, as documented in case series where patients remained sedated but ultimately recovered fully within 24–36 hours. A study in Faisalabad, Pakistan, recorded a 1.4% mortality rate (4 of 281 cases, all male) [18]. Those fatalities may have involved extremely high doses or co-ingestants (the report lacked details on the cause of death), highlighting that outcomes can vary. In this study, the absence of any deaths likely reflects the combination of timely medical intervention, the relative safety margin of benzodiazepines, and possibly the adjunctive use of Kahwa contributing to vigilance. Hospitalization stays were short, as the mean length of stay was only 1.36 ± 0.68 days (range 1–4 days), indicating that most patients required just overnight observation. The most novel aspect of this study is the deliberate use of Kahwa as part of the supportive treatment for benzodiazepine overdose. This is the first clinical study to document caffeinated beverages in managing acute sedative overdose. Kahwa is a traditional preparation in South Asia, typically consisting of green tea leaves brewed with spices like saffron, cardamom, and cinnamon [21]. Administering a caffeine-containing beverage in an overdose setting is a logical, albeit unconventional, attempt to hasten arousal [22]. The proposed mechanism by which Kahwa exerts its effect lies in its caffeine content. Caffeine acts as a central nervous system stimulant by antagonizing adenosine receptors in the brain, counteracting the sedative effects of benzodiazepines, and promoting wakefulness [23]. Unlike flumazenil, which directly displaces benzodiazepines from their receptors, caffeine provides a functional antagonism, improving consciousness while the drug remains in the system [24-26]. Since caffeine's effect is transient, repeated administration may be needed during observation. Kahwa's other components, such as green tea's theophylline and various spices, may also contribute by stimulating respiration, promoting gastric motility, and reducing nausea, thereby supporting overall recovery [27]. The combined effects may help explain the low rate of serious complications, including aspiration, observed in this study. Clinically, using Kahwa is attractive due to its simplicity and low cost, and it should be considered only in patients who can safely swallow or have a nasogastric tube in place, limiting its use to mild or moderate overdose cases [25].

While supportive care remains the standard for benzodiazepine overdose, benign adjuncts like Kahwa may be considered, especially in resource-limited settings, pending further evidence from controlled studies.

The study's retrospective design inherently carries risks of bias. This is a single-center study from a specialized poison control hospital. The patient population and management protocols may differ from other hospitals or regions. Karachi's poison center likely has experienced staff and established protocols (including the unique Kahwa intervention) that are not universally practiced. There was no control or comparison group, which limits the ability to establish causal relationships or directly attribute outcomes to the use of Kahwa. Prospective controlled studies should be conducted to evaluate the efficacy and safety of Kahwa as an adjunct in managing benzodiazepine overdose.

CONCLUSIONS

This study documents current clinical practice in managing benzodiazepine overdose, including adjunctive Kahwa administration. The results are descriptive and do not indicate the therapeutic effect of Kahwa specifically, as no causal interpretation is possible. These findings warrant further prospective studies to explore the potential supportive role of Kahwa in benzodiazepine overdose recovery.

Authors' Contribution

Conceptualization: OS
Methodology: JK, IK, MAK[1], OS, AK[2], DZ
Formal analysis: IK
Writing and drafting: JK, IK, MAK[1], DZ
Review and editing: OS, JK, IK, MAK[1], AK[2], DZ

All authors approved the final manuscript and take responsibility for the integrity of the work.

Conflicts of Interest

All the authors declare no conflict of interest.

Source of Funding

The author received no financial support for the research, authorship and/or publication of this article.

REFERENCES

[1] Liu S. Trends in Nonfatal and Fatal Overdoses Involving Benzodiazepines—38 States and the District of Columbia, 2019–2020. Morbidity and Mortality Weekly Report. 2021 Aug; 70(34): 1136–1141. doi: 10.15585/mmwr.mm7034a2.

[2] Marie BJ, Witry MJ, Reist JC. Barriers to Increasing Prescription Drug Monitoring Program Use: A Multidisciplinary Perspective. Computers, Informatics, Nursing. 2023 Aug; 41(8): 556-62. doi: 10.1097/CIN.0000000000000997.

DOI: https://doi.org/10.54393/pjhs.v7i1.3481

[3] Sharbaf SN, Bistas KG, Patel P, Saadabadi A. Flumazenil. 2024 Feb. In: Stat Pearls [Internet]. Treasure Island (FL): Stat Pearls Publishing; 2025 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470180.

[4] Xiao X, Erukainure OL, Sanni O, Koorbanally NA, Islam MS. Phytochemical Properties of Black Tea (Camellia sinensis) and Rooibos Tea (Aspalathus linearis); and Their Modulatory Effects on Key Hyperglycaemic Processes and Oxidative Stress. Journal of Food Science and Technology. 2020 Dec; 57(12): 4345-54. doi: 10.1007/s13197-020-04471-w.

[5] Pan H, Le M, He C, Yang CS, Ling T. Dark Tea: A Popular Beverage with Possible Medicinal Application. Chinese Herbal Medicines. 2023 Jan; 15(1): 33-6. doi: 10.1016/j.chmed.2022.08.005.

[6] Naveed M, Bibi J, Kamboh AA, Suheryani I, Kakar I, Fazlani SA *et al.* Pharmacological Values and Therapeutic Properties of Black Tea (Camellia Sinensis): A Comprehensive Overview. Biomedicine and Pharmacotherapy. 2018 Apr; 100: 521-31. doi: 10.1016/j.biopha.2018.02.048.

[7] Aaqil M, Peng C, Kamal A, Nawaz T, Zhang F, Gong J. Tea Harvesting and Processing Techniques and Their Effect on Phytochemical Profile and Final Quality of Black Tea: A Review. Foods. 2023 Dec; 12(24): 4467. doi: 10.3390/foods12244467.

[8] Ismail A, Akhtar S, Riaz M, Gong YY, Routledge MN, Naeem I. Prevalence and Exposure Assessment of Aflatoxins Through Black Tea Consumption in the Multan City of Pakistan and the Impact of Tea Making Process on Aflatoxins. Frontiers in Microbiology. 2020 Mar; 11: 446. doi: 10.3389/fmicb.2020.00446.

[9] Aydemir ME, Takım K, Yılmaz MA. Characterization of Phenolic Components of Black Teas of Different Origins and the Effect of Brewing Duration on Quality Properties. Food Science and Nutrition. 2024 Jan; 12(1): 494-507. doi: 10.1002/fsn3.3782.

[10] Truong VL and Jeong WS. Cellular Defensive Mechanisms of Tea Polyphenols: Structure-Activity Relationship. International Journal of Molecular Sciences. 2021 Aug; 22(17): 9109. doi: 10.3390/ijms 22179109.

[11] Wnuk E. The Antioxidant Potential of Black Tea Polyphenols in Heavy Metal Toxicity: An In Vitro Perspective. International Journal of Molecular Sciences. 2025 Aug; 26(16): 7926. doi: 10.3390/ijms 26167926.

[12] Deo AS, Devi PA, Sijisha KS, Anusha R, Mishra T, Mathew S *et al.* Comparative Studies on the Antioxidant, Anticancer and Anti-Inflammatory Activities of Green Tea, Orthodox Black Tea and CTC Black Tea. Journal of Food Science and Technology. 2024 Jul; 61(7): 1315-25. doi: 10.1007/s13197-023-05900-2.

[13] Teasdale G, Knill-Jones RO, van der Sande JA. Observer Variability in Assessing Impaired Consciousness and Coma. Journal of Neurology, Neurosurgery and Psychiatry. 1978 Jul; 41(7): 603-10. doi: 10.1136/jnnp.41.7.603.

[14] Shannon E, Jaiswal AK, Abu-Ghannam N. Polyphenolic Content and Antioxidant Capacity of White, Green, Black, and Herbal Teas: A Kinetic Study. Food Research. 2017; 2(1): 1-11. doi: 10.26656/fr.2017. 2(1).117.

[15] Association of Poisons Centres and Clinical Toxicology E. Position Paper: Single-Dose Activated Charcoal. Clinical Toxicology. 2005; 43(2): 61-88. doi: 10.1081/CLT-51867.

[16] An H and Godwin J. Flumazenil in Benzodiazepine Overdose. Canadian Medical Association Journal. 2016 Dec; 188(17-18): E537-. doi: 10.1503/cmaj.160357.

[17] Weinbroum A, Rudick V, Sorkine P, Nevo Y, Halpern P, Geller E *et al.* Use of Flumazenil in the Treatment of Drug Overdose: A Double-Blind and Open Clinical Study in 110 Patients. Critical Care Medicine. 1996 Feb; 24(2): 199-206. doi: 10.1097/00003246-199602000-00004.

[18] Saleem U, Mahmood S, Ahmad B, Erum A, Azhar S, Ahmad B. Benzodiazepine Poisoning Cases: A Retrospective Study from Faisalabad, Pakistan. Pakistan Journal of Pharmacology. 2010; 23(1): 11-3.

[19] Giftson J, Hazra D, Chandy GM. A Two-Year Retrospective Observational Cohort Study of Benzodiazepine Overdose Cases in the Emergency Department. Indian Journal of Critical Care Medicine: Peer-Reviewed, Official Publication of Indian Society of Critical Care Medicine. 2025 Feb; 29(3): 230. doi: 10.5005/jp-journals-10071-24925.

[20] Carry E, Vasilatis A, Johnson AL, Ryan JW. Expecting Medication Misuse: A Proactive Approach to Drug Discovery to Prevent Fatal Overdose. Future Medicinal Chemistry. 2025 Mar; 17(6): 681-92. doi: 10.1080/17568919.2025.2476388.

[21] Bano S, Majumder A, Srivastava A, Nayak KB. Deciphering the Potentials of Cardamom in Cancer Prevention and Therapy: From Kitchen to Clinic. Biomolecules. 2024 Sep; 14(9): 1166. doi: 10.3390/biom14091166.

[22] Reichert CF, Deboer T, Landolt HP. Adenosine, Caffeine, and Sleep-Wake Regulation: State of the Science and Perspectives. Journal of Sleep Research. 2022 Aug; 31(4): e13597. doi: 10.1111/jsr. 13597.

DOI: https://doi.org/10.54393/pjhs.v7i1.3481

[23] Hladun O, Papaseit E, Martín S, Barriocanal AM, Poyatos L, Farré M *et al.* Interaction of Energy Drinks with Prescription Medication and Drugs of Abuse. Pharmaceutics. 2021 Sep; 13(10): 1532. doi: 10.3390/pharmaceutics13101532.

[24] Wasowski C, Marder M. Flavonoids as GABAA Receptor Ligands: The Whole Story? Journal of Experimental Pharmacology. 2012 Feb: 9-24. doi: 10.2147/JEP.S23105.

[25] Farn SS, Cheng KH, Huang YR, Lee SY, Chen JT, Chang KW. Automated Synthesis of [18F] Flumazenil Application in GABAA Receptor Neuroimaging Availability for Rat Model of Anxiety. Pharmaceuticals. 2023 Mar; 16(3): 417. doi: 10.3390/ph16030417.

[26] Elgarf AA, Siebert DC, Steudle F, Draxler A, Li G, Huang S *et al.* Different Benzodiazepines Bind with Distinct Binding Modes to GABAA Receptors. American Chemical Society Chemical Biology. 2018 May; 13(8): 2033-9. doi: 10.1021/acschembio.8b00144.

[27] Hassen G, Belete G, Carrera KG, Iriowen RO, Araya H, Alemu T *et al.* Clinical Implications of Herbal Supplements in Conventional Medical Practice: A US Perspective. Cureus. 2022 Jul; 14(7): e26893. doi: 10.7759/cureus.26893.