

# Clinical Impact of Associated Volatile Compounds in Acute Ethanol Poisoning

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## Abstract

Adults across the globe in majority of the nations consume alcohol in different ratios. Usage of alcohol is linked with many adverse medical and psychological results for both the drinker and the community in general apart from resulting in considerable issues for majority of the drinkers. The focus of the current study is to assess the clinical influence of a set of majority of identified dangerous compound substances including acetone, methanol and isopropanol, in habitual alcoholics symbolized by severe ethanol alcoholism at the emergency divisions. The extant research employed an associate reviewing investigation of electronic medical record (EMR) evaluative assessment of patients currently suffering from severe ethanol harmfulness leading the habitual alcoholic ranking. When the patient was admitted to the ER division, the average intensity of ethanol, acetone, isopropanol and methanol in assumed ethanol harmfulness with other dangerous compounds were (131.06 mg/dl, 60.5 mg/dl, 9.2 mg/L and 26.6 mg/L). The intense intensities of blood acetone, isopropanol and methanol surpassing their endogenous proportions were linked uncharacteristically to be "ache in the stomach- 62%, anxiety- 68% and acetone breath smell- 31.25%" and an overstated severe ethanol harmfulness appearance as "puking -56% and Haematemsis 25%". Considering the above, it can be inferred that the intensities of blood acetone, isopropanol and methanol surpassing their endogenous propositions can be employed as clinical signs of uncharacteristic and/or overstated severe ethanol poisonous appearance.

**Keywords:** Ethanol toxicity; Toxic alcoholic metabolites; Acetone; Volatiles compounds

## Introduction

Adults across the globe in majority of the nations consume alcohol in different ratios. Usage of alcohol is linked with many adverse medical and psychological results for both the drinker and the community in general apart from resulting in considerable issues for majority of the drinkers. Alcohol is responsible for 3.2% of all deaths or 1.8 million deaths on an annual basis across the world. It is thus responsible for 4.0% of the illness problems [1].

Intense confusion, erratic conduct, lethargy, sudden mistakes, puking, seizures, bradyapoenia, pale, bluish, cold and sweaty skin are the consequence of severe ethanol inebriation. Habitual ethanol inebriation however is typified by weakening in decision making, memory, response time, motor co-ordination, rise in sexual need but with loss in sexual performance, thermoregulatory troubles with either hyper or hypothermia, vasomotor center deficiencies which result in hypotension and peripheral vasodilatation [2].

It is regular to see the existence of metabolic compounds including acetone, methanol and isopropanol in the blood of human beings when their bodies are in an ordinary physiological state. The intensity may differ from 0.01 to 10 mg/l [3,4]. Such endogenous metabolic compounds are developed when usual physiological processes occur: for instance: the existence of acetone is the output of breakdown of fats. The generation of methanol has been completely described as the outcome of several aspects since some ingredients leave an impact (specifically fruits which comprise of high proportions of pectins which have the ability to hydrolyze to methanol), chiefly as the result of the endogenic digestion by bacteria in the intestine [5].

Furthermore, post the intake of alcoholic drinks, the intensity of volatile compound rises, as virtually all alcoholic beverages comprise of acetone, methanol, and other alcohols as adulterations but at a very low intensity [6].

Dependence on alcohol results to their build-up in the body.

Inferences made in few researches related to the purging of methanol, acetone, and other compounds in alcoholics have been printed but the link between the metabolism of ethanol, the clinical influence in severe harmfulness is not yet evident [5,7,8].

The emphasis of the current article is to put forth a clinical link amongst ethanol and other volatile compounds substances including acetone, methanol and isopropanol, in habitual poisoned alcoholics characterized with severe alcoholism inebriation at the emergency division located in the Dammam Medical Complex. The researchers have made an attempt to set their link amongst the poisonous intensity of these compounds and their clinical signs in severe alcoholic inebriation.

## Materials and Methods

### Study setting

The current research adopted a surveying observational plan. Clinical presentations accounts for severe alcoholic inebriated were documented for all earlier habitual alcoholic inebriated 68 (all being males) patients, during a time frame ranging from 1 April 2012 to 30 September 2012, and all those fulfilling the suitability benchmarks.

### Inclusion and exclusion criteria

Those benchmarks employed in the current research were described to be inclusion benchmarks which were employed in

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identification for habitual alcoholic inebriation as the following: “extreme or repetitive consumption of alcohol for a minimum period of six months and one of the subsequent conditions: (a) signs of withdrawal; (b) proof of organic or psychological sicknesses as a result of alcohol harmfulness; (c) pathological configurations of employment; (d) hindrance with usual functions (social associations, performance at work, legal responsibilities)”, for people who are more than 18 years old and who have a clinical existence and laboratory record of intense alcoholic inebriation. Patients who are alcoholically inebriated who did not consume alcohol for over 6 months, or those who experienced metabolic or endocrinal mistake illnesses including (diabetes mellitus), and for patients who are less than 18 years old, those suffering from intense hepatic damage, intense renal damage; patients displaying one or more of the above stated conditions were not part of the current sample.

### Study population parameters

The data for the current research was gathered from patients who had received treatment for severe ethanol inebriation in addition to habitual alcoholism and their electronic records were gathered from the Online Toxicology Analytical Request and Result (OTARR) electronic system at the Dammam Regional Poison Control Center DRPCC-KSA. The data procured from the patients electronic medical records comprised of their personal history data, extant indicators of severe noxiousness, period of alcohol misuse, blood ethanol, acetone, Isopropanol and methanol concentration, random blood glucose level, serum electrolytes Na/K, blood gases and pH and blood bicarbonate level.

### Assay procedure

The blood samples for the toxicological investigation were obtained in straight tubes post the admission of the patient to the emergency division. The ethanol intensities in a runs of whole blood were calculated with the employment of head-space gas chromatography (Perkin Elmer, AutoSystem XL with HS 40 autosampler). Blood (0.2 ml) was mixed with 1.8 ml of 0.02 g/l of 2-methyl-2-propanol (tert-butyl alcohol), which was employed as an internal standard (IS). The samples were incubated in the autosampler for 22 min at 60°C. Separation was achieved on a Carbowax 1500 column under isothermal conditions (at 100°C). The temperature of the flame ionization detector was 200°C.

The same technique was employed to ascertain the intensities of other compounds (methanol, acetone and Isopropanol). The chromatograms were documented and measurements were undertaken

by employing the Turbochrom computer program. The researchers prepared six-point calibration curves [AUC compound/AUCIS=f (compound concentration)], with the intensities of the analytes in average solutions varying between 0.1 to 5 g/l for ethanol and from 0.1 to 50 mg/l for other compounds.

All the investigations undertaken in the current research were part of the typical diagnostic procedure for severely poisoned alcoholics and were ratified by the Ethical Committee of Dammam Regional Poison Control Center. The Helsinki Declaration of 1975 /Fifth revision (2000) was followed while conducting the experiments.

### Statistical analysis

A latest SPSS statistical package Version 19 was employed to evaluate the data statistically. The data was put forth as Mean ± Standard Deviation (S.D.). The T-test was employed to contrast the two groups and the p-values were regarded to statistically important if ≤ 0.05.

### Grouping of the studied patients

Based on the existence or absence of poisonous metabolic final products of ethanol metabolism were employed to segregate the investigated samples into two groups:

**Group I:** Ethanol Intoxicated Group by laboratory detection without toxic concentration of other organic compounds as “Acetone, Isopropanol, and Methanol”.

**Group II:** Ethanol Intoxicated Group by laboratory detection with toxic concentration of other organic compounds as “Acetone, Isopropanol, and Methanol”.

### Results

The extant study included a sample size of 68 patients (all males with an average age ± SD: 36.3 ± 11.2 years). In all 68 samples were tested for ethanol, acetone, isopropanol, methanol, random blood glucose level, blood gases, pH and bicarbonate level in the study period spanning across 6 months (the entire sample populace had been admitted in the ER after reporting severe ethanol inebriation in addition to habitual alcoholism).

The varied attributes of the patients is depicted in Table 1. Different yet crucial criteria including age, sex, and admission status were used to investigate and evaluate the patients. In both the groups that were analyzed the table 1 also depicted the time frame of habitual alcoholism and the extent of poisoning intensity score.

	Group I	Group II
	Ethanol with low volatile compounds level group (n=52)	Ethanol with high volatile compounds level group (n=16)
Age (Years) (Mean ± SD)	38.3 ± 9.7	32.6 ± 7.4
Sex	All of the studied cases were male	
BMI (Kg/m2) (Mean ± SD)	26.5 ± 3.5	28.1 ± 3.8
Alcoholic dependency duration (Years)	Range (1-10) / Mean (4 ± 2.2)	Range (1-10) / Mean (6.5 ± 2.8)
Admission/Non-admission Status	42/10	16/0
Hospital stay duration (Days) (Mean ± SD)	1.2 ± 0.5	1.9 ± 0.7
Poisoning Severity Score		
0 Non	0 (0%)	0 (0%)
1 Minor	13 (25%)	0 (0%)
2 Moderate	22 (42.2%)	5 (31.3%)
3 Severe	11 (21.3%)	7 (43.7%)
4 Fatal	6 (11.5%)	4 (25%)

**Table 1:** Age, sex, duration of abuse, admission status, alcoholic dependence duration and poisoning severity score in the studied acute alcohol poisoning on top of chronic alcoholism.

All the investigated instances in group II of the current research were admitted while in 10 instances patients from group I were discharged after being given 1<sup>st</sup> aid management in the ER department. For levels 3 and 4 in the investigated instances of group II, a poisoning severity score of 25% and 43.7% were documented while on the other facet a low percentage of 21.3% and 11.55% were reported for the same ranking.

The variable averages and levels of ethanol and its toxic metabolites (acetone, methanol and isopropanol) in both the investigated groups were depicted in table 2. The study reported an extremely crucial increase in the average acetone in the investigated group of ethanol mixed with toxic metabolites when contrasted to the intense ethanol noxiousness group without toxic metabolites (60.5 ± 62.4) (2.19 ± 1.6) mg/l respectively. A crucial dip in the average of the calculated pH and bicarbonate level in the investigated group II was noticed in contrast to the group I whose p value stood at <0.05.

About 44% of the studied cases in the toxic ethanol metabolite group II were presentenced with Glasgow coma scale less than 8. The general ratio of the worried conscious level in the investigated severe ethanol noxiousness without toxic metabolites association linkages stood at 32.7%.

The indicators of severe ethanol noxiousness in addition to habitual alcoholism and linked ECH modifications were depicted in table 3. Alcoholic breath smell, puking and dizziness were the usual indications in the severe ethanol inebriation without poisonous toxic metabolites generation group (42.3%, 38.4% and 32.6%) to be specific. However, on the other hand, the serious indicators and symptoms were rise in the severe ethanol noxiousness linked to the intensely poisonous metabolites groups particularly in hemeatemesis, hallucination, and seizures (25%, 31.25% and 6.25%) respectively.

In context to ECG indicators: out of all the investigated instances in group II with sinus tachycardia, sinus tachycardia was seen in around

44% of the cases. On the contrary facet, around three-fourth (75%) of the investigated instances in group I documented a standard ECG pattern.

In context to deducing the varied acetone, isopropanol and methanol toxic metabolites intensities laboratory tests were undertaken. These were depicted in table 4. Patients from group (1) were tested for non-poisonous blood intensity ratios for the acetone, methanol and isopropanol. 12 cases from group II however on the other hand, were found to have a high blood acetone level (>10 mg/dl), isopropanol level (>1 mg/L) and methanol level (>5 mg/L). High blood acetone and methanol levels were seen in two patients in spite of non-poisonous isopropanol intensity. It was only in one instance that a patient showed signs of only noxious acetone intensity and one instance depicted a mixed acetone and isopropanol intensity.

The scatted spot graphs amongst blood Ethanol intensity and noxious metabolites acetone, isopropanol and methanol for a non-crucial link (p value; 0.117, 0.241 and 0.651) correspondingly are depicted in figures 1, 2 and 3.

## Discussion

68 patients (all males with an average age of ± SD: 36.3 ± 11.2 years) were part of the sample for the current research. The researcher noticed a rise in the BMI of the investigated group I with an average length of exploitation of 4 years and the group II showed a higher rise in their BMI (ethanol with high toxic ethanol metabolites) with a protracted length of abusing alcohol (6.5 years) in the current research. The inferences made in the current research were as per the inferences depicted by from Stefan et al. [9], according to which a high BMI was seen in 54 males who were alcoholic (29.1 kg/m<sup>2</sup>). The rise in BMI was considered to be the result of a depression in the frontal lobe and thalamic neurotransmitter (N-acetylaspartate and GM choline).

The extant research reported that all instances linked to severe

Parameters	Group I	Group II
	Ethanol with low volatile compounds level group (n=52)	Ethanol with high volatile compounds level group (n=16)
<b>Toxicological Parameters: (Mean ± SD / Range)</b>		
Blood Ethanol Level (mg/dl)	187.06 ± 116.4 / (72-512)	131.06 ± 67.3 / (104-354)
Blood Acetone level (mg/L)	2.19 ± 1.6 / (0.1-8.3)	60.5 ± 62.4** / (10-192)
Blood Isopropanol Level (mg/L)	0.25 ± 0.19 / (0.1 ± 0.97)	9.2 ± 6.27* / (1.5-24.0)
Blood Methanol Level (mg/L)	2.2 ± 0.19 / (0.01 ± 9.4)	26.6 ± 10.5* / (17.7-38.3)
<b>Non-toxicological Parameters: (Mean ± SD)</b>		
pH	7.34 ± 0.13	7.28 ± 0.15*
PaO <sub>2</sub>	88.7 ± 23.4	82.5 ± 25.4
PaCO <sub>2</sub>	37.2 ± 15.6	31.3 ± 14.1
HCO <sub>3</sub>	21.6 ± 5.7	18.4 ± 3.7*
RBG (Random Blood Glucose) (mg%)	135.1 ± 52.8	107.3 ± 48.2
<b>Vital Signs: (Mean ± SD)</b>		
Pulse (beat/min)	88.3 ± 19.1	104.2 ± 12.7
Systolic Blood Pressure (mmHg)	129.2 ± 22.1	110.1 ± 12
Diastolic Blood Pressure (mmHg)	76.7 ± 12.5	64.3 ± 8.4*
Temperature (°C)	36.9 ± 0.5	36.6 ± 0.3
Respiratory Rate (Cycle/min)	18.3 ± 6.8	23.2 ± 4.3
<b>Conscious level: Glasgow Coma Scale (GCS) n(%)</b>		
GCS<8	10 (19.2%)	7 (43.75%)
GCS 9-12	7 (13.5%)	6 (37.5%)
GCS>13	35 (67.3%)	3 (18.75%)

\*P<0.05

\*\*P<0.01

Table 2: Laboratory and clinical variables in the different studied groups.

	Group I	Group II
	Ethanol with low volatile compounds level group (n=52)	Ethanol with high volatile compounds level group (n=16)
<b>Symptoms &amp; Signs</b>		
Nausea	17 (32.6%)	12 (75%)
Vomiting	20 (38.4%)	9 (56.25%)*
Hematemesis	8 (15.3%)	4 (25%)
Abdominal Pain	14 (26.9%)	10 (62.5%)*
Dizziness	17 (32.6%)	12 (75%)
Confusion	14 (26.9%)	10 (62.5%)
Agitation	15 (28.8%)	11 (68.75%)*
Hallucination	8 (15.3%)	5 (31.25%)
Seizure	0 (0%)	1 (6.25%)
Aggressiveness	5 (9.6%)	7 (43.75%)*
Depressed	14 (26.9%)	3 (18.75%)
Dyspnea	12 (23.1%)	6 (37.5%)
Alcoholic breath odour	22 (42.3%)	4 (25%)
Acetone breath odour	0 (0%)	5 (31.25%)
Dry red skin	1 (1.9%)	0 (0%)
Pale cold clammy skin	2 (3.8%)	3 (18.75%)
<b>ECG Changes</b>		
Normal ECG Pattern	39 (75%)	7 (43.75%)
Sinus Bradycardia (<50 beats/min)	2 (3.8%)	1 (6.25%)
Sinus Tachycardia	10 (19.2%)	7 (43.75%)*
Sinus arrest	1 (1.9%)	1 (6.25%)
Hear block	1 (1.9%)	0 (0%)

Table 3: Variable signs, symptoms and ECG changes in the studied groups.

Acetone	Methanol			
	More than 5 mg/L		Less than 5 mg/L	
More than 10 mg/L	2	1	1	12
Less than 10 mg/L	0	0	52	0
	Less than 1 mg/L	More than 1 mg/L	Less than 1 mg/L	More than 1 mg/L
	Isopropanol			

Table 4: Classification of the studied cases on the basis of methanol, acetone and isopropanol concentration.

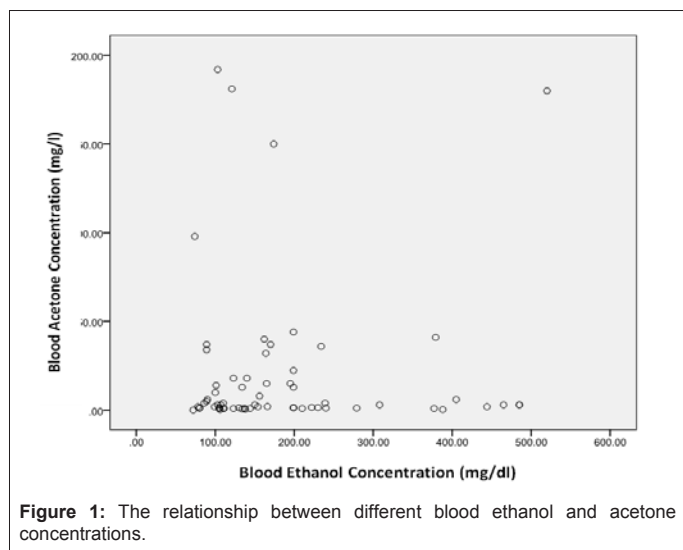


Figure 1: The relationship between different blood ethanol and acetone concentrations.

ethanol inebriation with high poisonous metabolites intensity (acetone, isopropanol and methanol) were admitted and stayed in the hospital

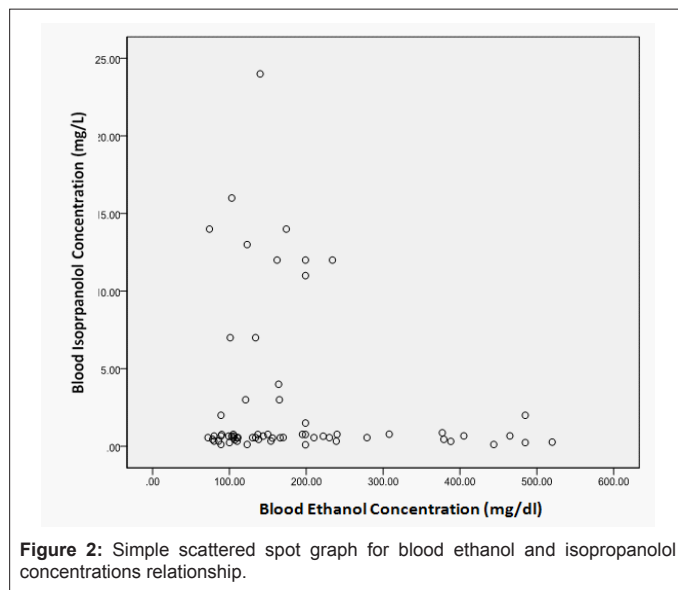


Figure 2: Simple scattered spot graph for blood ethanol and isopropanol concentrations relationship.

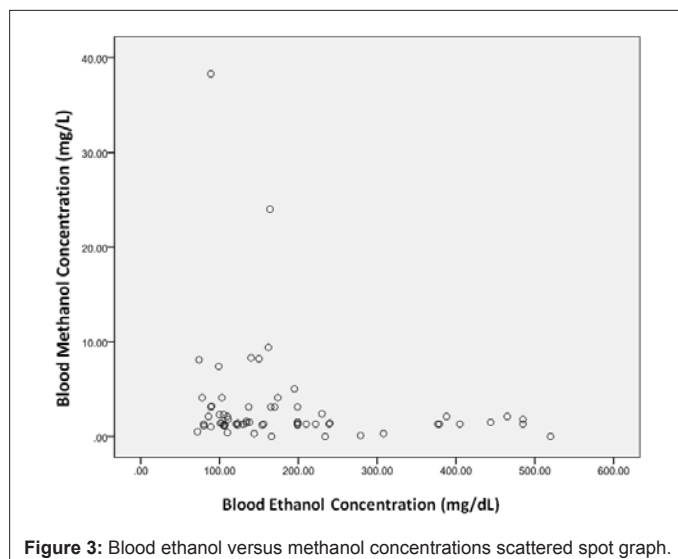


Figure 3: Blood ethanol versus methanol concentrations scattered spot graph.

for around  $1.8 \pm 0.6$  day when compared to the other group of intense ethanol noxiousness without poisonous metabolites; their discharge rate was 24%. Thus in all the cases (100%) the admission rate in instances of severe poisonous metabolites intensities displays the acuteness of the clinical scenario and the dangerous impact of their poisonous metabolites levels. As per the extant scenario, a research conducted in Ireland verified that the hospital admission linked to severe alcohol abuse represented around 10% of the entire emergency department admissions and around three-fourth (75%) from all alcoholic noxious ER visits had a mean hospital stay for around 2.7 day [10].

The authors in the extant research evidently observed that poisonous metabolites of ethanol increase the poisoning seriousness score considerably to be 25% with fatal degree, 43.7% with severe degree and the remainder percentage depicted moderate degree. Thus, the radical impact of poisonous metabolites acetone, isopropanol and acetone in the worsening the clinical state of the ethanol noxiousness status is clearly observed.

The general average of the blood ethanol level in the investigated instances with poisonous metabolites denotes a non-crucial reduction



when compared to the ethanol level of group II. Despite the low blood ethanol level there was a high clinical dangerous influence for the linked poisonous metabolites (acetone, isopropanol and methanol). One research deduced the same inference related to the typical dangerous impact of poisonous metabolites, and explained their crucial variance when a contrast was made amongst the intense and habitual alcoholic people [11].

Many aspects including 1) high intake of alcoholic drinks, as nearly all alcoholic beverage have acetone, methanol, and other alcohols as impurities [6]; (2) being addicted to alcohol results in their build-up in the body. Consequently, the metabolism of ethanol to the other volatile organic compounds particularly in heavy drinkers may be responsible for the high level of poisonous metabolites in the group II that was investigated [12,13].

In the extant research 16 instances of severe ethanol poisonous with high levels of acetone were investigated; methanol and isopropanol were suffered from a considerable reduction in blood pH and bicarbonate level when compared with 52 acute ethanol toxicity personnel with normal earlier stated poisonous metabolites. The researchers in the current investigation may ascribe this radical variation due to the neurological inhibitory and metabolic production action of acetone, methanol and isopropanol. The processes of earlier impacts were also documented in several literatures [14-16].

The haemodynamic impact of ethanol toxicities in moderate 44.5 mg/dl after 50 minutes from when the ethanol was in taken was investigated by Stott et al. 1991. They discovered normal systolic and diastolic blood pressures without crucial modifications in the sitting or standing position. Contradictorily, Watson et al., documented a typical hypotensive impact acetone, isopropanol and methanol in varied documented instances of yearly records of the American Association of poison Control Centers Toxic exposures Surveillance System 2004 [17]. A similar inference was made in the current research, that the group of only ethanol toxicity indicated more less normal ranges in the crucial symptoms, while contradictorily the group of mixed ethanol and high poisonous metabolites intensities depicted abnormal low pressure up to crucial reduction in the diastolic blood pressure ( $p < 0.05$ ).

Majority of the typical symptoms in the intense mixed ethanol/volatiles compounds inebriations were aches in the stomach (62.55%), anxiety (68.7%) and acetone breath smell (31.25%). All the signs were the consequence of mixed ethanol, acetone, isopropanol noxiousness. Jeffrey and Kurtz [18] discussed the varied types of noxiousness and listed a typical sign of every volatile compound separately as acetone represented mainly irritable gastric symptomatology and typical acetone smell and mouth odour, isopropanol chiefly linked with neurological irritability and hypotensive impact effects and ultimately methanol chiefly linked with metabolic acidosis and neurological depressive action.

The authors also observed a noticeable overstatement of routine indicators and signs of severe ethanol inebriation that mixed with other organic poisons even with low ethanol intensity (131 mg/dl) when contrasted with only severe ethanol inebriation (187 mg/dl) as nausea (75%), vomiting (56.25%), Haematemesis (25%) and finally pale cold clammy sweating (18.75%). Thus, from the particulars stated earlier it can be explained that the existence of severe ethanol noxiousness scenario with signs that surpass the characteristic indicators of severe ethanol noxiousness; a rise in the portability of diagnosis mixed ethanol noxiousness particularly high volatile compounds that may be the outcome of metabolic pathways of ethanol "endogenous source" or adulteration of alcoholic drinks with other organic compounds as isopropanol, acetone and methanol.

## Summary and Conclusion

The outputs of the current research prove that the existence of acetone, isopropanol, and methanol in the blood of patients who are alcoholic symbolized with severe ethanol toxicities is usual. It is recommended by the writers that the intensity of blood acetone, isopropanol and methanol surpassing their endogenous quantities can be employed as a clinical sign of a characteristic and/or hyperbolic severe ethanol poisonousness presence. Additionally, the researchers also propose investigations to identify the source of connections between dangerous compounds with severe ethanol drunkenness from endogenous generation of ethanol metabolism or exogenous source of unhygienic alcoholic drinks.

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