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# FORENSIC MEDICAL DIAGNOSIS OF THANATOGENESIS OF COMBINED VINEGAR AND ALCOHOL POISONING: CLINICAL MANIFESTATIONS AND MORPHOLOGICAL CHANGES.

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**Abstract**

Acute poisoning with concentrated acetic acid (vinegar essence, 70–90%) is a severe chemical trauma causing massive intravascular hemolysis, corrosive gastrointestinal burns, and rapid multi-organ dysfunction. Concomitant alcohol intoxication alters the toxicodynamics, creating a dual effect: partial mucosal protection versus accelerated systemic toxicity. Despite high lethality, chronological patterns of clinical-morphological manifestations remain underexplored in forensic literature, particularly regarding alcohol's modifying role.

**Keywords:** acetic acid poisoning, vinegar essence, alcohol comorbidity, thanatogenesis chronology, forensic toxicology, hemolysis, survival modeling, combined chemical trauma

**Objective:** To delineate the temporal sequence of toxic effects in isolated versus alcohol-complicated acetic acid poisoning, quantify ethanol's impact on thanatogenesis timing, and propose forensic criteria for severity assessment and combined intoxication diagnosis.

**Materials and Methods:** Retrospective forensic analysis of 127 fatal cases (group I: isolated acetic acid poisoning, n=68; group II: combined with ethanol 2.5–3.5‰, n=59). All exhibited severe hemolysis (plasma free hemoglobin >CH50). Symptom registration times (±SE) were compared using Student's t-test

(p<0.05 significance). Survival modeling employed probabilistic graphs based on modified Kass algorithms.

**Results:** Alcohol exerted bidirectional modulation. It delayed initial corrosive damage and exotoxic shock but markedly accelerated life-threatening systemic complications (Table 1). Key divergences included earlier bloody vomiting (13.4±2.6 vs 25.6±3.2 h, p<0.001), cerebral edema (22.4±6.2 vs 38.4±7.8 h, p<0.001), and hepatorenal failure (162.4±40.6 vs 232.4±56.4 h, p<0.001) in combined cases. Higher ethanol levels further compressed inter-symptom intervals, increasing toxic effect density per time unit.

**Table 1.** Comparative Chronology of Key Toxic Effects in Acute Acetic Acid Poisoning (Mean Registration Time, hours ± SE)

| **Sign/Manifestation** | **Isolated (Group I)** | **Combined with Alcohol (Group II)** | **p-value** |
|---|---|---|---|
| Esophageal burn | 12.4 ± 0.82 | 18.4 ± 1.2 | <0.05 |
| Gastric burn | 20.4 ± 4.7 | 28.6 ± 6.7 | <0.05 |
| Exotoxic shock | 10.4 ± 2.32 | 18.8 ± 3.46 | <0.001 |
| Bloody vomiting | 25.6 ± 3.2 | 13.4 ± 2.6 | <0.001 |
| Toxic psychosis | 34.8 ± 4.5 | 28.4 ± 2.6 | <0.05 |
| Agitation | 36.6 ± 5.2 | 22.4 ± 6.23 | <0.001 |
| Cerebral edema | 38.4 ± 7.8 | 22.4 ± 6.2 | <0.001 |
| Pulmonary edema | 62.4 ± 14.7 | 50.4 ± 7.6 | <0.05 |
| Fatty liver degeneration | 54.6 ± 8.6 | 42.4 ± 6.6 | <0.001 |
| Acute cardiovascular failure | 96.4 ± 14.4 | 74.6 ± 12.2 | <0.001 |
| Acute hepatorenal failure | 232.4 ± 56.4 | 162.4 ± 40.6 | <0.001 |

Survival analysis revealed distinct patterns (Figure 1). Isolated poisoning caused abrupt viability loss within the toxigenic phase (<72 h), whereas moderate

alcohol shifted mortality to the somatogenic phase (7–10 days) via secondary complications, though severe ethanol (>3.5‰) reverted to early lethality.

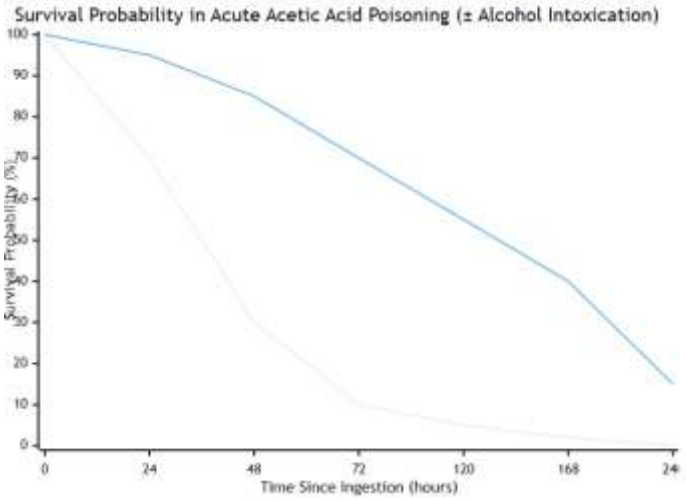


Figure 1. Probabilistic survival curves (modified Kass model). Isolated cases show rapid decline in toxigenic phase; alcohol-complicated cases exhibit initial plateau followed by delayed attrition from infections/multi-organ failure.

**Conclusion:** Moderate-severe alcohol intoxication accelerates thanatogenesis in acetic acid poisoning by intensifying hemolysis-driven systemic effects while transiently mitigating corrosive injury. Temporal deviation from isolated poisoning timelines, coupled with toxic effect density, provides objective forensic markers for diagnosing combined intoxication, estimating ingestion recency (<10 days), and grading trauma severity. These criteria enhance autopsy interpretation and medicolegal causation analysis.

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