Prognostic Factors Including Clinical Manifestation and Paraclinic Finding in Sever Methanol Toxicity

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**Abstract**

**Objective:** Ingested methanol (a toxic alcohol) undergoes enzymatic oxidation to toxic formic acid, resulting in acidosis and formaldehyde which resulted in neurotoxicity specifically optic neuritis and blindness and death in severe poisoning. According to Iranian law, selling, buying, and consumption of alcoholic beverages is a punishable crime and people who wish to drink alcohol use industrial or homemade ethanol. Due to depressant effects of methanol on the central nervous system, the objective of this study was to assess the pathologic findings on brain CT scan in methanol poisoned patients beside of their clinical information.

**Material and method:** This prospective study was from Jan 2013 to May 2013. All 20 methanol poisoned patients examined and questionnaires were filled by physician. Brain CT scans without contrast medium were obtained.

**Results:** All of cases were male; their mean age was 33.1 ± 9.2 years. The mortality rate was 15%. Hemodialysis was performed in 85% cases. Mean primary Methanol Level was 22.4 ± 10.5 mg/dL and after hemodialysis was 7.3 ± 6.1. Totally serum methanol levels pre and post hemodialysis were 22.4 and 7.3, respectively. Brain death was recorded in 2 patients. The most common findings at admission were loss of consciousness (n=11) and blurred vision (n=8). The early pathologic brain CT findings were bilateral hypo density lesion in putamen, low attenuation in sub cortical, white matter bilateral hemorrhagic necrosis in putamen and bilateral hypo density in globus pallidus, respectively.

**Conclusion:** In conclusion, it seems CT finding are important as Methanol concentration before any other Para clinic findings and even clinical manifestations.

**Key words:** Methanol; Toxicity; Brain CT scan

**Introduction**

Methanol as a toxic alcohol exists in many solvents, antifreeze solutions, glass cleaner, and paint remover. Also its contamination may occur along with Home Ethanol production in some countries. Ingested methanol undergoes enzymatic oxidation to toxic formic acid, resulting in acidosis, neurotoxicity and death in severe poisoning. High mortality rate from massive methanol ingestion has been recorded annually. In poisoned methanol patients with delay admission severe metabolic acidosis for toxic metabolites has developed. Treatment is based on antidote administration such as fomepizole or ethanol. These are for antagonizing methanol oxidation and also folic acid to facilitate the catabolism of formic acid, correction of acidosis and dialysis for methanol elimination [1]. According to Iranian law, selling, buying and consumption of alcoholic beverages is a punishable crime. People who wish to drink alcohol use industrial or homemade ethanol that sometimes are a mixture consisting of methanol and ethanol [2]. Both fear of punishment and delayed onset of symptomatic poisoning cause late presentation and are associated with a high mortality rate. This occurs even though patient confidentiality is maintained. Rapid diagnosis and treatment are necessary to prevent death and to minimize the neurologic sequelae. The objective of this study was to assess the pathologic findings on brain CT scan in methanol poisoned patients beside of their clinical information.

**Material and Method**

This was a prospective cross-sectional study which was carried at Loghman poison ward in Tehran, Iran. The Loghman toxicology unit serves a population excess of 12 million and normally sees 28 000 emergency ward presentations due to poisoning each year of which 12.5 thousand are admitted. This is the only tertiary hospital for poisoned patient in the capital city and is the largest in the nation. According to the best of our knowledge our inpatient complex seems to be the biggest clinical toxicology department in the Middle East. The study period was from Jan 2013 to May 2013. In that period of time all 20 methanol poisoned patients examined and questionnaires were filled by physician. In this study, patients with ethanol toxicity were excluded. Descriptive data were include age, gender, time elapsed consumption, blood pH, level of consciousness, laboratory profile include methanol level, presenting symptoms and physical examinations on date of admission. All cases reviewed were assigned to one of the following four categories based on their outcomes: (1) complete recovery, (2) blindness and other neurological morbidities, (3) death and (4) discharge by their own consent. Blindness was confirmed by an ophthalmologist. In another research which was done in our hospital and TRC by Dr. Hassanian et al. CT finding in sever Methanol intoxication [2,3]. Brain CT scans without contrast medium were obtained.

Treatment was given according to the available standard protocols, and in accordance with the Helsinki Declaration. Analyses were


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performed from blood samples already drawn for treatment purposes. Time interval between methanol ingestion and ED arrival time was traceable in all patients. Data was reported as mean ± SD, frequency and relative frequency for quantitative data. Chi-square and fisher’s exact test were used. All data was analyzed with SPSS software, version 16.

Results

During 5 month period (Jan 2013–May 1013), a total of 20 eligible methanol poisoned patients from 180 alcohol consumption cases was selected. All of them were male; their mean age was 33.1 ± 9.2 years. Of these, 3 patients died, 6 cases were Discharge by their own consent and the others were alive, whereas three of them had sequelae such as blindness. Meandose of methanol consumption and delay in admission time in four groups (survivors without/with sequelae, dead and the discharged) cases were showed in Table 1.

Hemodialysis was performed in 17 (85%) cases for methanol toxicity. Totally serum methanol levels pre and post hemodialysis were 22.4 and 7.3, respectively. Concomitant usage of other drugs was unknown. Brain death was recorded in 2 patients.

The mean serum pH for patients who survived without sequelae, Survived with sequelae, discharged and dead patients were 7.2 ± 0.17, 7.1 ± 0.03, 7.2 ± 0.14 and 6.8 ± 0.22 respectively. The early pathologic brain CT findings were bilateral hypodensity lesion in putamen, low attenuation in subcortical, white matter bilateral hemorrhagic necrosis in putamen and bilateral hypodensity in globuspallidus, respectively.

Discussion

The purpose of this study was to assess the pathologic findings on brain CT scan in methanol poisoned patients beside of their clinical information.

We detected hypodensity lesion in putamen as the most common pathologic brain CT findings, and low attenuation in subcortical, white matter bilateral hemorrhagic necrosis in putamen and bilateral hypodensity in globuspallidus, respectively.

Likewise 2 other studies hypodensity lesion in putamen were reported as the most common neuropathological finding. It was due to the location of the putamina in the boundary zones of vascular perfusion and higher concentration of formic acid accumulation [3,4].

According to the several investigation, most of the patients had normal CT scan in the acute period of methanol intoxication, and only survived cases for more than 24 h usually showed characteristics CT findings of bilateral low attenuation lesions in the putamina and cerebral deep white matter [4-7].

Our study shows that there is close relation between mean serum pH and mean time elapsed since methanol consumption (P<0.005).

<table>
<thead>
<tr>
<th>Dose of methanol consumption(CC)</th>
<th>Survived without sequelae mean</th>
<th>Survived with sequelae mean</th>
<th>Dead mean</th>
<th>Discharged patient by their own consent mean</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delay in admission time (hours)</td>
<td>33</td>
<td>18</td>
<td>38</td>
<td>28</td>
<td>30</td>
</tr>
</tbody>
</table>

Table 1: Mean dose of methanol consumption and delay in admission time in the different groups.

We found that mean methanol level in our cases is lower (22.4 ± 10.5 mg/dL versus 165, 60 and 196) than other studies [8-10]. One possible explanation for this discrepancy is that in untreated or delayed cases of methanol poisoning is reasonable to suppose that eventually all of the methanol will be metabolized and the severity of methanol poisoning is reflected by the magnitude of metabolic acidosis, which is caused by formic acid accumulation [11,12]. Toxicity is due to toxic metabolite and not from methanol itself. It seems amount of methanol consumption, the time from intake to admission and concomitant ethanol intake would be a better prognostic element. Although it may be for another metabolic acidosis of unknown origin [8-10]. In Hassanian et al. study showed that all cases who had ethanol blood level survived [2]. This finding is in agreement with the other studies which showed patients who ingested both methanol and ethanol were more likely to survive than methanol ingestion alone, while we excluded patients with ethanol toxicity in this study [1,8,13,14].

The mortality rate in our study was 15%, This was in accordance with previous reports [9,14,15]. On the contrary Hassanian et al. study, the mortality rate was 48%. In the present study more than 50% patients had loss of consciousness. The number of comatose patients and prolong time from intake to admission beside the illegality of alcohol and fear of punishment keep the patients from seeking help. According to existence of low methanol level in spite of severe metabolic acidosis in some of our cases it is logical to suppose long period from intake to admission is long enough to metabolized and developed severe toxicity and it's results, if we suppose that hemodialysis should be continued until elimination of toxic metabolites and metabolic acidosis [16-18].

In addition, blindness was the irreversible sequel of this poisoning that was occurred in 3 cases and near 50% case had blurred vision. Likewise in the Hassanian et al. study blurred vision was the most common finding (60%) [2].

Conclusion

In conclusion, it seems CT findings are important as Methanol concentration before any other Paraclinic findings and even clinical manifestations. We recommend further research in a larger group for finding more correlation of methanol pH and brain CT finding.

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References