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Methanol poisoning: acute MR and CT findings in nine patients

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Abstract
Introduction Methanol poisoning is an uncommon but potent central nervous system toxin. We describe here the CT and MR findings in nine patients following an outbreak of methanol poisoning.
Methods Five patients with a typical clinical presentation and elevated anion and osmolar gaps underwent conventional brain MRI with a 1.5-T Gyroscan Interna scanner. In addition nonenhanced CT was performed in another three patients with more severe toxicity.
Results Bilateral hemorrhagic or nonhemorrhagic necrosis of the putamina, diffuse white matter necrosis, and subarachnoid hemorrhage were among the radiological findings. Various patterns of enhancement of basal ganglial lesions were found including no enhancement, strong enhancement and rim enhancement.
Conclusion A good knowledge of the radiological findings in methanol poisoning seems to be necessary for radiologists. The present study is unique in that it enables us to include in a single report most of the radiological findings that have been reported previously.

Keywords Methanol · Poisoning · CT · MRI

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Introduction

Methanol is a component of varnishes, paint removers, perfumes, antifreeze, copy machine fluid and gasoline mixtures, and may be ingested accidentally or intentionally. It is a CNS depressant that is potentially toxic after ingestion, inhalation or transdermal exposure [1, 2], and it also crosses the placenta [3]. Without treatment, ingestion of 30 ml pure methanol usually results in death and as little as 4 ml can result in blindness [1, 2, 4]. Blood methanol levels above 200 mg/l are considered toxic and levels above 1500 mg/l are potentially fatal [1, 2]

The peculiarity of methanol poisoning is the latency period between its ingestion and the appearance of manifestations. The latency may be related to the concomitant ingestion of ethanol which affects the metabolism of methanol [5]. Although the latency in symptom onset is variable, symptom progression may be rapid [6]. Early symptoms of methanol poisoning, except for visual disturbances, are nonspecific and include nausea, vomiting and abdominal pain. Late manifestations are due to acidosis secondary to the accumulation of formic acid and lactic acid [7]. The terminal event is often respiratory arrest and the fatal period is from 6 to 36 hours.

Current treatment of methanol poisoning consists of gastric lavage, ethanol therapy, alcohol dehydrogenase enzyme blockade by means of fomepizole, dialysis, alkalinization, and the use of cofactors such as folate [8, 9]. In spite of improved treatment, mortality in methanol poisoning remains high, mainly because of an often difficult and therefore delayed diagnosis.

Various imaging techniques have enabled a better understanding of the clinical manifestations of methanol toxicity. Modern neuroimaging techniques also lead to much earlier diagnosis of CNS damage by focusing attention on the lesions that are radiologically detectable. A good knowledge of the target areas in methanol poisoning can lead the radiologist to suspect methanol poisoning in a relevant clinical setting, and guide early treatment.

As far as we are aware, the imaging findings of methanol poisoning have been described in only a few small case series in the literature, the largest one including five patients [10]. We describe here the radiological manifestations of methanol poisoning in nine patients following an outbreak of methanol poisoning.

Materials and methods

The outbreak occurred in Shiraz, the southern city of Iran. There are severe restrictions on the production and consumption of alcoholic beverages in Iran for religious reasons, and the liquor causing the present outbreak of methanol poisoning was produced illegally in the country, and was sold in bottles looking much like original bottles. Most of the patients were admitted to two main university hospitals: Namazee Hospital and Faghighi Hospital. The diagnosis of methanol poisoning was based on the typical clinical presentation, elevated anion and osmolar gaps, the presence of methanol which was measured as part of an alcohol screen, and the absence of other toxins in a toxicology screen. All patients were treated with intravenous alcohol, hemodialysis, folate therapy and alkalinization.

Nonenhanced CT of the brain was the initial imaging study in all patients, and was repeated in some patients during the hospital course according to neurological manifestations. Conventional brain MRI with a 1.5-T Gyroscan Interna scanner was also performed in all patients except those who were severely ill who were admitted to

Table 1 Demographic and clinical characteristics of nine patients with methanol toxicity, and their imaging findings

Patient no.	Age (years)	Sex	Mental status at presentation	Methanol level (mg/dl)	Visual acuity	Arterial pH	Outcome[a]	Radiological findings
1	27	Male	Awake	42.7	2–5/10	7.1	Lost to follow-up	Bilateral hemorrhagic necrosis of the putamen and globus pallidus with more severe changes in the former. Whole lentiform nucleus enhanced strongly on contrast-enhanced images. Normal CT findings on the day of admission
2	19	Male	Confusion	13.2	4–5/10	7.0	Lost to follow-up	Bilateral putaminal necrosis. No enhancement on contrast-enhanced images
3[b]	29	Female	Awake	–	3–5/10	–	Parkinsonian symptoms at 1 month	Bilateral lentiform nucleus necrosis. Contrast-enhanced images revealed peripheral enhancement of putaminal lesions
4	26	Male	Confusion	49.5	Finger count	7.1	Lost to follow-up	Bilateral hemorrhagic lentiform nucleus necrosis. Normal initial CT findings
5	24	Male	Awake	14	10/10	7.43	Lost to follow-up	Normal CT and MRI findings
6	40	Male	Awake	12.4	2–4/10	7.37	Lost to follow-up	Normal CT and MRI findings
7	42	Male	Awake	13	1–3/10	7.06	Lost to follow-up	Normal CT and MRI findings
8	26	Male	Comatose	14.6	–	6.87	Death	Global cerebral hypodensity; no intraparenchymal bleed; generalized subarachnoid hemorrhage
9	24	Male	Comatose	35.6	–	7.08	Death	Bilateral basal ganglia especially putaminal necrosis, diffuse white matter hypodensity and bilateral occipital necrosis more prominent on the left side

[a] Since the drinking of alcoholic beverages is banned in Iran, almost all patients refused to attend for follow-up.
[b] Late presentation at 1 month.

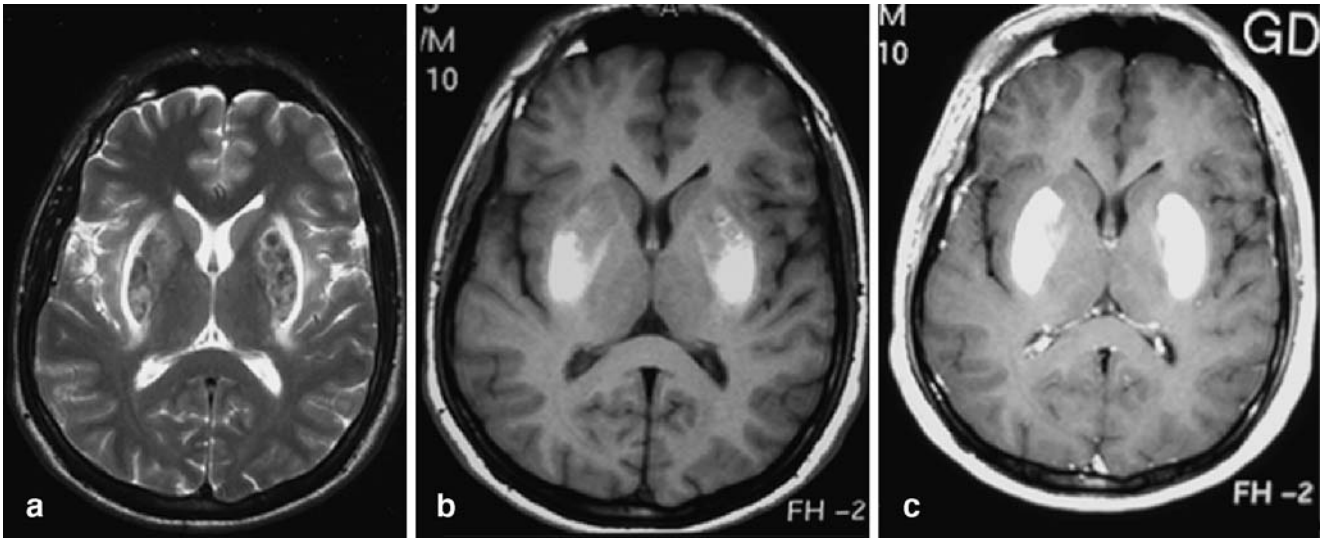

Fig. 1 MRI shows bilateral hemorrhagic necrosis of the putamen and globus pallidus as shown by mixed isointense and hypointense areas on the T2-weighted image (**a**), hyperintensity on the T1-weighted image (**b**), and strong enhancement on the contrast-enhanced image (**c**)

the intensive care unit for close observation and monitoring. MRI was also performed in a patient who was referred after 1 month and claimed to have drunk liquor from the same common source that had caused the outbreak.

This study was approved by the bioethical committee of Shiraz University of Medical Sciences.

Results

The demographic and clinical characteristics of the patient and the radiological findings are presented in Table 1. Nine patients (eight male and one female) with a mean age of 28.5 years (range 19–42 years) with documented methanol poisoning were investigated in this study. The mean methanol level in these patients was 24.3 mg/dl (range 13–49.5 mg/dl). Of the eight patients who were admitted to the hospital, seven (87%) presented with visual disturbances and six (75%) with gastrointestinal symptoms, and two (25%) were comatose. One patient presented with respiratory arrest. A 29-year-old woman also was presented 1 month after the outbreak with parkinsonian symptoms. There was no significant relationship between severity of symptoms at presentation, clinical outcome or extent of brain injury and methanol levels ($P>0.05$).

Regarding outcome, only one patient recovered completely and was discharged from hospital without sequelae. Five patients recovered partially and were discharged with sequelae and the remaining two patients did not respond to the therapeutic measures and died.

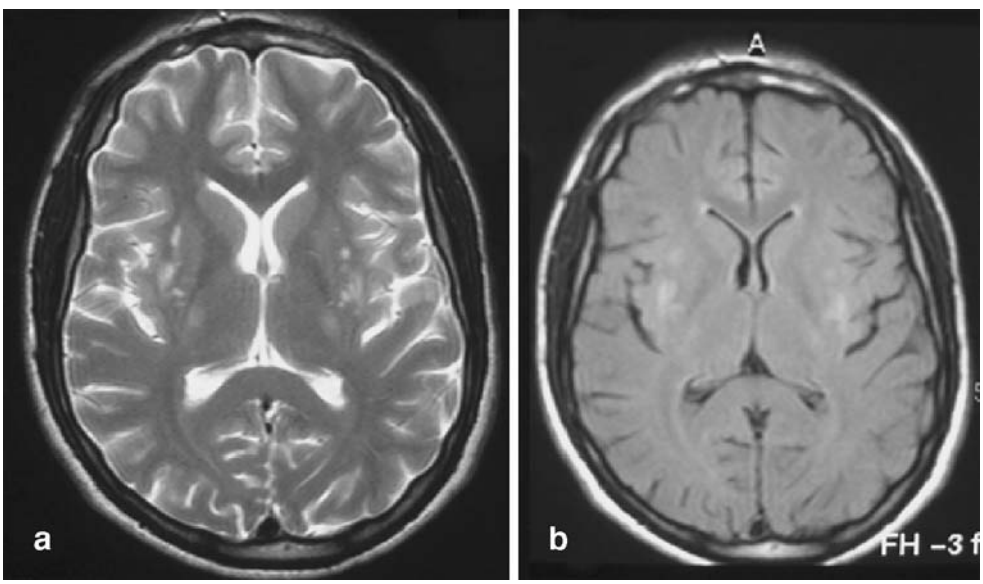

Fig. 2 MRI shows bilateral necrosis of the putamen as shown by small linear and plaque-like hyperintensities with extension to the lateral and superior thalami on the T2-weighted image (**a**) and corresponding increased signal intensity on the FLAIR image (**b**)

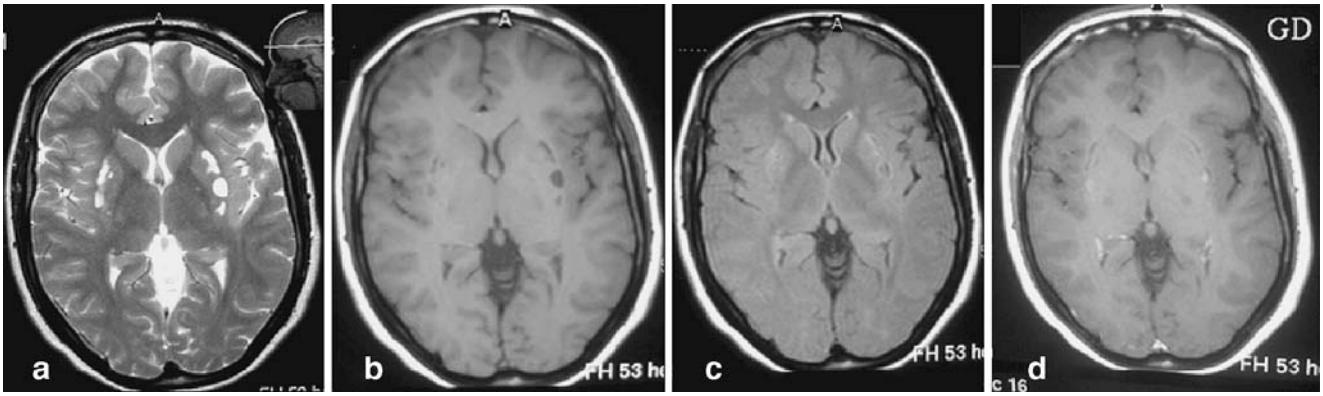

Fig. 3 MRI shows bilateral lentiform nucleus necrosis as shown by hyperintense putaminal lesions, less prominent globus pallidus involvement on the T2-weighted image (**a**), with reciprocal hypointensities on the T1-weighted image (**b**). Hypointense putaminal lesions with a hyperintense rim are evident on the FLAIR image (**c**). Slight peripheral enhancement is seen on the contrast-enhanced image (**d**)

MRI was performed in five patients during their hospital course and in another patient who presented 1 month after the outbreak; however MRI could not be performed in the other three patients who were too ill.

Regarding imaging findings, four patients had hemorrhagic or nonhemorrhagic necrosis of the basal ganglia (Figs. 1, 2, 3 and 4). One patient showed subarachnoid hemorrhage accompanied by global cerebral hypodensity (Fig. 5) and another patient revealed bilateral basal ganglia especially putaminal necrosis accompanied by diffuse white matter hypodensity and bilateral occipital necrosis more prominent on the left side (Fig. 6). The remaining three patients had no abnormal findings on brain CT or MRI.

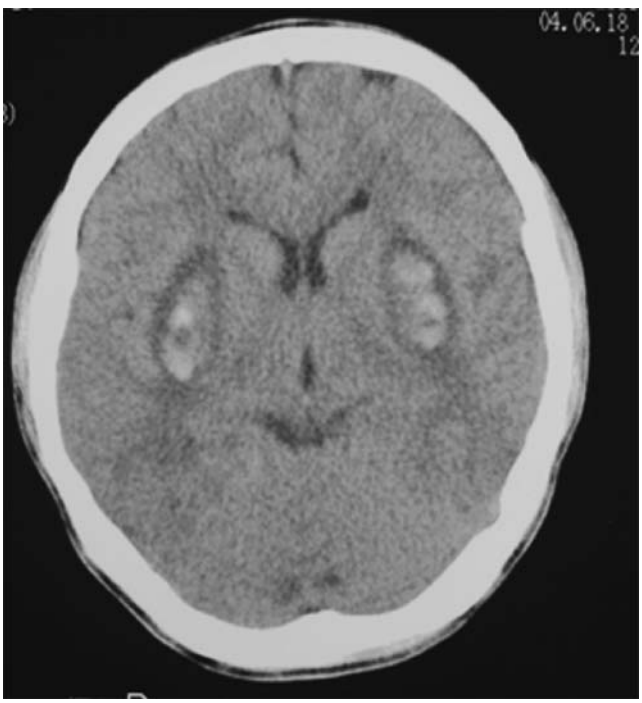

Fig. 4 CT scan shows bilateral hemorrhagic nucleus necrosis

Discussion

Radiological manifestations of methanol poisoning have been frequently described previously. Bilateral necrosis of the basal ganglia is accepted as the most characteristic radiological feature of methanol poisoning [9, 11–15]. Both hemorrhagic and nonhemorrhagic damage of the putamen can occur [9, 11]. Both CT and MRI give similar results showing bilateral areas of putaminal necrosis; however, MRI gives better anatomical detail and may reveal small hemorrhagic lesions [14]. If the patient survives the acute phase of the illness, resorption of the infarcted hemorrhagic putamen tissue occurs with formation of cystic cavities within the nucleus [16]. Other brain lesions occasionally described include edema, necrosis of subcortical white and gray matter, cerebellar cortical lesions, bilateral intracerebral hemorrhage, bilateral tegmental necrosis and diffuse cerebral edema [9, 11, 17–20]. Table 2 illustrates the main radiological features of methanol poisoning reported in previous series. As expected bilateral basal ganglia lesions were also the predominant pattern in our patients.

The mechanism responsible for selective putaminal necrosis is unknown. It has been postulated that the necrosis results from decreased blood flow through the basal veins of Rosenthal secondary to hypotension [30]. However, many cases described in the literature did not experience hypotension during their hospital course, thus making decreased venous flow as the cause of putaminal necrosis less likely [31]. Another possibility is that the necrosis occurs as a direct toxic effect of formic acid with higher concentrations of formic acid accumulating in the putamen than in other areas of the brain [31] or due to varying sensitivity of striatal neurons to toxic metabolites of methanol [32]. Finally the association of bilateral putaminal necrosis with Leigh disease which is characterized by congenital lactic acidosis may suggest that the putamen may indeed be more sensitive to an acidic environment than other areas of the brain [33, 34].

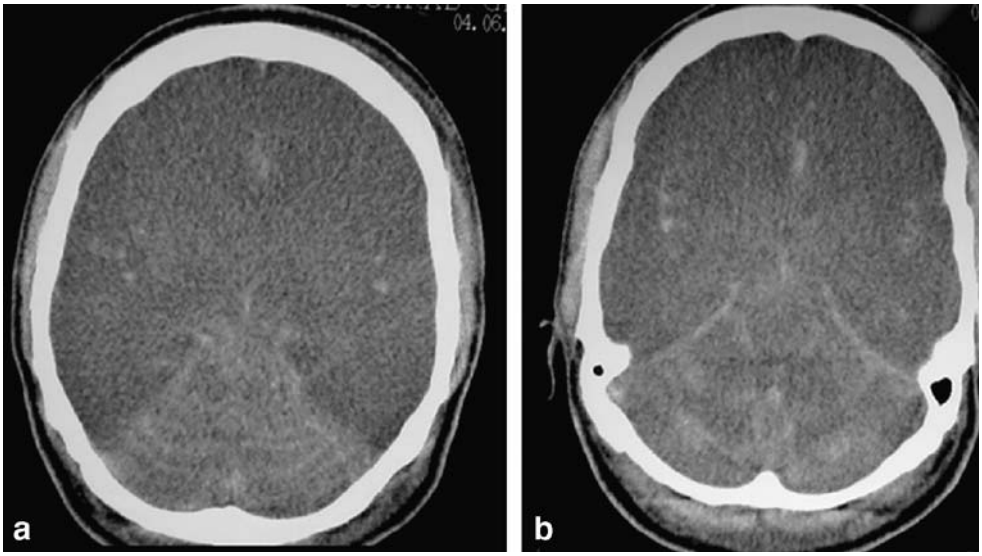

Fig. 5 CT scan shows global cerebral hypointensity, and generalized subarachnoid hemorrhage

Putaminal changes on conventional MRI and CT scans are not specific to methanol intoxication and have been described in Wilson's disease, Kearns-Sayre syndrome, Leigh disease, and various other neurodegenerative disorders [34–37]. In addition there are pathological similarities between methanol poisoning and carbon monoxide inhalation [38, 39], hypoxic–ischemic injury [40, 41] hemolytic–uremic syndrome [42], hydrogen sulfide poisoning [43], and (in the rare patient who survives) acute cyanide intoxication [44]. A common industrial solvent, 1,1,1-trichloroethane (methyl chloroform), which is found in typewriter correction fluid and is also used as a recreational drug, has also been shown to produce lesions in the basal ganglia similar to those observed in patients with methanol poisoning [45].

In patients with putaminal necrosis who survive the initial insult, extrapyramidal symptoms and signs including rigidity, tremors, mask-like facies, and monotonous speech may develop. Imaging has the potential to play a role in the diagnosis of acute methanol poisoning weeks to months later in a patient presenting with dystonia and features of parkinsonism [46, 47]. Extrapyramidal symptoms are usually permanent, but improvement may occur following treatment with levodopa [48–50] although not in all patients [16]. We also encountered parkinsonian symptoms in one of our patients, but evaluation of this aspect in the other patients was impossible because they were lost to follow-up.

There is no general agreement between the extent of radiological abnormalities and clinical outcome in methanol poisoning. Patankar et al. noted that the severity and extent of necrosis of the lentiform nuclei do not necessarily correlate with the clinical outcome [19]; however, Aquilonius et al. stated that a clear relationship exists between the degree of necrotic change within the putamen and clinical outcome [20]. The presence of injury to both the subcortical white matter (with relative sparing of

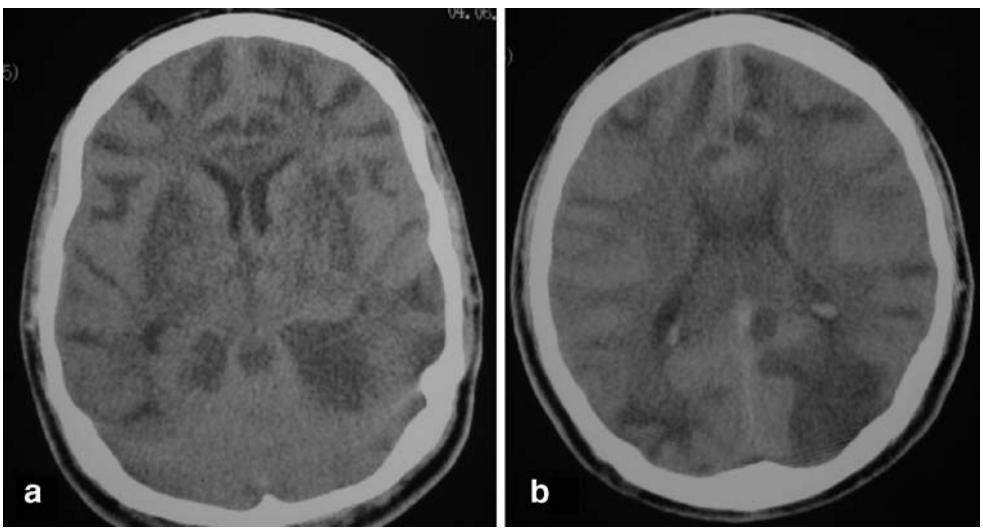

Fig. 6 CT scan shows (**a**) bilateral basal ganglia especially putaminal necrosis, and (**b**) diffuse white matter hypodensity and bilateral occipital necrosis more prominent on the left side

Table 2 Main radiological features of methanol poisoning reported in previous series

Reference	No. of patients	Radiological findings
21	1	Bilateral putaminal hemorrhagic necrosis and subcortical white matter lesions with peripheral contrast enhancement
22	2	FLAIR and T2-W images (patient 1): bilateral increased signal in lentiform nuclei, especially the putamina, but also the corona radiata, centrum semiovale, and subcortical white matter FLAIR and T2-W images (patient 2): putaminal hyperintensity Diffusion-weighted images (patient 1): decreased diffusion in the corresponding areas
23	1	Fast spin echo, T2-W and FLAIR images: putaminal hyperintensity FLAIR images: diffuse hyperintensity in the subarachnoid space DWI: putaminal hyperintensity; decreased ADC values
9	1	MRI: hemorrhagic necrosis of the putamina and bilaterally increased signal on T2-W images in the corona radiata, centrum semiovale, deep white matter, and midbrain consistent with acute toxic edema
10	5	T2-W images: hyperintense putaminal lesions in three patients
24	4	T2-W images: hyperintense putaminal lesions and symmetric lesions in the medial parieto-occipital and occipital lobes
25	1	Acute: hemorrhagic necrosis of the corpus striatum; infarcts in the anterior and middle cerebral artery territories Subacute: Atrophy of the optic chiasm and prechiasmatic optic nerves; cerebral infarcts
11	1	Enhancing lesions in the caudate nuclei, putamina, hypothalamus and subcortical white matter
26	1	MRI on day 4: typical putaminal lesions and peripheral white matter lesions that spared a thin rim of subcortical white matter
17	1	Bilateral hemorrhagic necrosis of the putamen and caudate nuclei; extensive subcortical necrosis and symmetric bilateral necrosis of the pontine tegmentum and optic nerves
27	1	Bilateral putaminal hemorrhage
12	1	Bilateral putaminal hemorrhagic necrosis and increased signal throughout the cerebral white matter most prominently in the frontal and parietal regions
28	1	CT day 3: hemorrhagic necrosis of bilateral putamina and the cerebral cortex CT day 22: further changes including necrosis of bilateral subcortical white matter; following administration of contrast agent gyral enhancement in the otherwise normal looking cerebral cortex
29	1	Extensive subarachnoid hemorrhage accompanied by left caudate and pontine hemorrhage
13	1	Putaminal necrosis
18	1	Bilateral putaminal necrosis and bilateral cerebellar cortical lesions
14	1	Bilateral putaminal necrosis
15	1	Basal ganglia lesions

the centrum semiovale) and the putamen has also been claimed to be indicative of severe methanol toxicity [9]. In our patients there was a direct relationship between the degree of radiological abnormalities and clinical outcome, and the two patients with most severe radiological abnormalities succumbed to methanol poisoning.

Various studies have shown that there is no statistically significant association between methanol level and prognosis [51]. This lack of association is logical since severely poisoned patients may have a low methanol level because most of the ingested methanol is metabolized to formic acid [52]. Also repeated intake of the liquor over time could explain high methanol concentrations because ethanol is also ingested preventing methanol metabolism. Instead of methanol level, severe metabolic acidosis, coma or seizure at presentation and increased pCO_2 have been shown to be associated with poor prognosis [53–55]. As expected we also found no significant relationship between methanol level and prognosis.

Methanol intoxication produces classic neuropathological changes. Optic neuropathy related to loss of myelin in the optic nerves is perhaps the best known neuropathological change after methanol poisoning [11, 56]. Post-mortem studies of poisoned patients who survive for a period of days or weeks have shown a distinctive pattern of brain injury characterized by bilateral putaminal necrosis and white matter hemorrhagic necrosis, especially affecting subcortical regions [16, 30]. These lesions spare the most peripheral white matter, the subcortical association fibers [11, 26]. Unfortunately, permission for autopsy could not be obtained in either of our two deceased patients.

Hemorrhage has been reported in up to 14% of patients with methanol poisoning [12, 46] and has been suggested to indicate a poor prognosis [17, 20]. In some studies hemorrhage was confined to the putamina [9, 12], while in others hemorrhagic necrosis occurred both in the putamina and subcortical white matter [22, 30]. In this series some type of hemorrhage (hemorrhagic putaminal

necrosis in two patients and subarachnoid hemorrhage in one) occurred in one-third of our patients. To our knowledge following an extensive literature search, there is only one previous report of subarachnoid hemorrhage in methanol poisoning [29].

Although cerebral hemorrhage related to methanol poisoning is usually bilateral and not expansive [9, 21, 57], as in our patients, extensive hemorrhage with extension in to the ventricular system [46, 58] and massive hemorrhagic transformation of cerebral ischemic lesions [59] have also been reported.

Some believe that heparinization during dialysis may play some role in the development of brain hemorrhage [27, 46]. However, hemorrhage also is seen prior to and irrespective of dialysis [17, 19] or after hemodialysis without systemic heparin [59]. On the other hand, dialyzed patients are those most severely poisoned and therefore most likely to suffer from such hemorrhagic complications. Hemorrhagic basal ganglial necrosis was present in two of our patients who were dialyzed, but in patient 8 (Table 1), subarachnoid hemorrhage was evident on the CT scan before hemodialysis. It seems that although the direct role of hemodialysis has not been proved, avoidance of heparin in patients with methanol intoxication seems logical.

There are only a few studies in which diffusion-weighted imaging has been used in methanol poisoning. Deniz et al. [23] reported bilateral putaminal hyperintensity on diffusion-weighted images with decreased apparent diffusion coefficient (ADC) values, while Server et al. [22] reported high signal intensity and low ADC values in the subcortical white matter, basal ganglia, and right hippocampus. Takao et al. also reported bilateral putaminal lesions with restricted diffusion with development of new lesions in the subcortical white matter on the third day after admission [60]. These findings of restricted diffusion have been interpreted as cytotoxic edema which is less likely to be reversible and indicates nonviable tissue [22]. A possible explanation for diffusion abnormalities is that the accumulation of formic acid leads to metabolic acidosis and inhibition of cytochrome oxidase which causes anoxia [61] and the subsequent failure of the Na^+/K^+ adenosine triphosphatase pump which transports Na^+ and K^+ ions across the membrane. Failure of this pump leads to loss of ionic gradients and a flux of water from the extracellular to the intracellular space [62, 63] and reduction in ADC values [23]. Diffusion MR imaging has also been used during the early stages of acute carbon monoxide poisoning to show the early reversible white matter lesions [64]. Unfortunately, diffusion-weighted imaging was not performed in our patients due to some technical problems.

Contrast enhancement of brain lesions can be observed after methanol poisoning. Anderson et al. reported a 47-year-old man with significant methanol intoxication who had enhancing lesions in the caudate nuclei, putamina, hypothalamus, and subcortical white matter on MRI [11]. In another patient tiny foci of enhancement were found in the frontal region [22]. Meningeal and gyral enhancement of the cerebral cortex has also been reported on contrast-enhanced CT scans [28] as has peripheral contrast enhancement of subcortical white matter lesions [21]. Metabolic dysfunction and consequent endothelial cell injury may cause disruption of the blood–brain barrier which may be represented by contrast enhancement [11]. In our patients almost all the patterns, including nonenhancement, strong enhancement and rim enhancement of basal ganglial lesions, occurred.

Conclusion

With the increasing use of modern neuroimaging techniques in the evaluation of unconscious patients, a knowledge of the radiological findings in methanol poisoning seems to be necessary for radiologists. The radiological manifestations include a wide variety of abnormalities involving many areas of the brain. The present study is unique in that it enables us to include in a single report most of the radiological findings that have been reported previously.

Conflict of interest statement We declare that we have no conflict of interest.

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