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ORIGINAL ARTICLE

Spectrum of MRI findings in 58 patients with methanol intoxication: Long-term visual and neurological correlation

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Optic nerve

Abstract *Purpose:* To describe MRI and DWI spectrum of brain and optic pathway changes in cases who survived acute methanol poisoning and explore whether there is correlation between imaging features and long-term visual and neurological sequelae.
Materials and methods: We retrospectively reviewed the conventional MRI and DWI of 58 consecutive patients with methanol poisoning. All patients were examined in the chronic phase.
Results: Optic nerve enhancement and atrophy were detected in 33 cases (56.9%). Degree of optic nerve atrophy correlated well with cupping and time lag since initial exposure to methanol. Bilateral putamen necrosis was present in 45 cases (77.6%), 19 showed asymmetrical involvement, and caudate was involved in 6 cases. Asymmetrical necrosis and caudate involvement were correlated with higher grade of neurological deficit. Twenty-one cases (36.2%) showed combination of bilateral putamen necrosis and optic nerve enhancement. Subcortical white matter high SI was detected in 25 patients (43.1%). DWI clearly depicted putamen necrosis with non-restricted pattern.
Conclusion: Spectrum of residual MRI Findings in patients who survived methanol poisoning included bilateral optic nerve atrophy and enhancement, bilateral putamen and caudate necrosis as well as subcortical white matter high SI at T2WI. Diffusion WI did not have additional value in chronic stage.

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1. Introduction

Methanol intoxication appears after accidental, suicidal oral ingestion of industrial solvents, and cleaning of antifreeze liquids, or occasionally is due to fraudulent adulteration of wine or other alcoholic beverages (1).

Methanol is a highly toxic substance and acute methanol poisoning produces severe metabolic acidosis and serious neurological symptoms, including severe visual impairment, extrapyramidal signs and coma (2). Optic neuropathy and putaminal necrosis are the two main complications of methanol poisoning, generally occurring in combination after severe intoxication. Surviving patients usually show permanent sequelae (residual visual) and consist of bilateral blindness and motor dysfunction including rigidity, hypokinesia, and other Parkinsonian-like signs (3).

There are few reported specific findings for methanol intoxication on magnetic resonance imaging (MRI) of the brain and optic pathway, fewer on DWI.

The aim of this study was to describe MRI and DWI spectrum of brain and optic pathway changes in cases that survived acute methanol poisoning and explore whether there is correlation between imaging features and long-term visual and neurological sequelae.

2. Patients and methods

This is a retrospective case series of 58 consecutive patients seen at the King Khaled Eye Specialist Hospital and the King Saud University hospitals since September 2010 to November 2015. Informed consent was waived for this Health Insurance Portability and Accountability Act (HIPAA)-compliant institutional review board-approved retrospective study.

2.1. Case selection

Inclusion criteria included patients who presented with different degree of visual or neurological deterioration with past history of methanol intoxication (oral intake) at least 4 weeks prior to examination and performed MRI examination of the brain and orbit including post contrast study and DWI. Exclusion criteria included cases that did not perform complete MRI examination or lack visual field examination, cases with other medical diseases that could affect visual acuity or motor function such as glaucoma, diabetes or Parkinsonism, cases who had no clear history of methanol ingestion or there is denial from the patient or their family, and cases who have contraindications to do MRI (cardiac pacemaker, metallic FB in the eye, claustrophobic, etc.) were also excluded. None of the patients were examined during the acute phase of intoxication.

2.2. Clinical assessment

All patient charts were reviewed including gender, age, date of methanol ingestion, any persistent or newly developed signs or symptoms since date of methanol ingestion and any other concurrent medical disease. The following tests were performed: optical coherence tomography or OCT with evaluation of the retinal nerve fibers layer (RNFL), visual evoked potentials (VEP), complete ocular examination (visual acuity/field, color vision, contrast sensitivity, and fundus), neurological examinations, and biochemical tests.

Assessment motor function is evaluated by board certificated neurologist (TB) with further grading of residual motor effect into grade I (rigidity) and grade II (rigidity + tremors).

Visual acuity, visual fields, pupillary reaction, and fundus features were assessed by board certificated senior neuroophthalmologist (AG and TB) and ratio of optic disk cupping was recorded.

2.3. Imaging

MR imaging studies were performed at 3-T scanner (Magnetom Allegra 3 T; Siemens, Erlangen, Germany) with the use of a dedicated head coil. The gradient strength was 40 mT/m and the slew rate was 400 T/m/s. First, sagittal spin-echo T1-weighted MR images were obtained with TR/TE of 350–7500/9–13 ms. Transverse T2-weighted MR images were obtained with TR/TE of 2400–2800/19–96 ms, FOV of 20×22 cm, section thickness of 4 mm, interslice gap of 1–2 mm, and matrix of 320×180. Coronal T2-weighted MR images with fat suppression were obtained with TR/TE of 2400–2800/19–87 ms, FOV of 20×22 cm, section thickness of 4 mm, inter-slice gap of 1–2 mm, and matrix of 320×216.

The diffusion gradients were applied along the three orthogonal directions (x, y, and z) with the same strength. Diffusion-weighted MR images were acquired with a diffusion-weighted factor b of 0, 500, and 1000 s/mm^2, and ADC maps were generated for all images using a multi-slice spin echo planar imaging sequence. Imaging parameters were TR/TE of 3200/81 ms, FOV of 20×22 cm, section thickness of 4 mm, inter-slice gap of 1–2 mm, number of excitations of 6, matrix of 128×128, EPI factor of 128, and RF pulse and width of 1200. The data acquisition time for the diffusion weighted images was 1.33 min.

After intravenous injection of 0.1 mmol/kg gadopentetate dimeglumine (Magnevist; Schering, Berlin, Germany), axial, coronal, and sagittal T1-weighted MR images (TR/TE = 400–575/13–15 ms) with fat suppression was applied. Fat suppression was accomplished with a frequency selective pre-saturation pulse.

2.4. Image interpretation

Senior neuroradiologist (SE) who was blinded to the clinical diagnosis and the degree of visual field affection re-reviewed the MRI images of all patients. Any abnormal high SI in the basal ganglia or subcortical white matter was assessed in both conventional images, diffusion weighted images (DWI) and ADC map (to detect areas of restricted or non restricted diffusion). The caliber and enhancement of the Optic nerves (ON) were assessed visually by the senior neuroradiologist (SE) with further grading into either normal or mild, moderate and severe atrophied in coronal T2 image which provided the best cross sectional view of the ON. The slice was selected then the retrobulbar area was zoomed to 300×, and then ON diameter was measured 3 mm behind the globe using an electronic caliper. The ON diameter obtained from both sides was correlated in collaboration with the vision acuity and fundus examination as there are a lot of normal variations in the size of the optic nerve.