Background: Methanol poisoning is a life-threatening condition which induces acute toxic optic neuropathy with possible long-term visual sequelae in survivors.

Aim: To study the prevalence, character, dynamics, and key determinants of chronic morphological and functional visual pathway changes during 4 years after methanol-induced optic neuropathy.

Methods: A total of 55 patients with confirmed methanol poisoning with mean age 46.7 ± 3.6 years (46 males and 9 females), and 41 controls were included in this prospective longitudinal cohort study. The patients were examined 4.9 ± 0.6, 25.0 ± 0.6, and 49.9 ± 0.5 months after discharge. The following tests were performed: visual evoked potential (VEP), optical coherence tomography with retinal nerve fiber layer (RNFL) measurement, brain magnetic resonance imaging (MRI), complete ocular examination, biochemical tests, and apolipoprotein E (ApoE) genotyping.

Results: Of 42/55 patients with all three consecutive examinations, abnormal RNFL thickness was registered in 13 (31%) and chronic axonal loss during the observation period was found in 10 (24%) patients. The risk estimate of chronic global RNFL loss for arterial blood pH<7.3 at admission was: 11.65 (1.91-71.12; 95% CI) after adjusting for age and sex. The patients with chronic axonal degeneration demonstrated further progressive visual loss in 7/10 cases. Abnormal VEP latency P1 was registered in 18/42 right eyes (OD) and 21/42 left eyes (OS), abnormal amplitude N1P1 in 10/42 OD and OS. Mean latency P1 shortening due to remyelination during the follow-up period was 15.0 ± 2.0 ms for OD and 14.9 ± 2.4 ms for OS. A further decrease of amplitude N1P1 ≥ 1.0 mcV was observed in 17 of 36 patients (47%) with measurable amplitude. ApoE4 allele carriers had lower global and temporal RNFL thickness and longer latency P1 compared to the non-carriers (all $p < 0.05$). The odds ratio for abnormal visual function was 8.92 (3.00 – 36.50; 95% CI) for ApoE4 allele carriers ($p < 0.001$). The patients with abnormal RNFL thickness had MRI signs of brain damage in 10/13 versus 8/29 cases with normal RNFL thickness ($p=0.003$). Pre-hospital ethanol administration as a “first aid” antidote and rapid acidemia correction and formic acid elimination by intermittent hemodialysis were associated with better visual outcome (both $p < 0.05$).

Conclusion: Methanol-induced toxic optic neuropathy may lead to chronic retinal axonal loss during the following years. Arterial blood pH on admission was the strongest predictor of chronic RNFL thickness decrease. Chronic retinal neurodegeneration was associated with progressive loss of visual functions and necrotic brain lesions. Improvement of optic nerve conductivity occurred in more than 80% of patients, but the amplitude of evoked potential tended to decrease during 4 years of observation. ApoE4 allele carriers demonstrated lower RNFL thickness, longer latency P1, and more frequent methanol-induced brain damage compared to the non-carriers. Pre-hospital ethanol administration as a “first aid” antidote and intermittent hemodialysis demonstrated positive preventive effect against long-term visual sequelae of methanol poisoning.