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Is hippocampus the answer of retrograde amnesia in glufosinate poisoning?

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Objective: Glufosinate is derived from bialaphos, a novel tripeptide herbicide discovered in the 1970s. The herbicides containing ammonium salt of glufosinate (GLA) and its low environment toxicity makes it marketed worldwide. In Taiwan, the sale of herbicide formulations containing GLA is rapidly increasing, and poisoning from incidental and suicidal ingestion increased, too. The exact mechanism of GLA poisoning in human is still uncertain, especially in the CNS system. The neurologic complications usually are characterized by loss of consciousness, convulsions, and one of the most unique symptoms is retrograde amnesia. However, only few literatures reveals brain examinations results which focus on this feature. Herein, we report a patient with GLA poisoning who showed retrograde amnesia with bilateral hippocampal areas lesion.

Case report: A 67 year-old man without systemic diseases came due to consciousness changed. According to his cohabitation girlfriend, the patient drank much alcohol-like rice wine and herbal wine for three days. This morning, he was found that conscious disturbance and urinary and stool incontinence by his girlfriend. A bottle pesticide Glufosinate ammonium was left over. He was brought to our ER for help where showed BP: 166 / 83 mmHg, HR: 122, RR: 18, BT: 37.5℃, G.C.S.: E 4M6V5. Bilateral pupils 3.0/3.0 with light reflex. CXR showed RLL alveolar pattern, suspect aspiration pneumonitis. Initial blood vein gas showed pH 7.16, pCO_2 71mmHg, pO_2 23mmHg, HCO_3 25.3 mM/dL, and ammonia level 113 was noted later. Endotracheal intubation and ventilator support was performed because of acute respiratory failure. We treated him with supportive care and the endotracheal tube removed after four days. Retrograde amnesia was then noticed because he could not remember the detail about recently one month, including GLA digestion. The brain MRI shows subtle hyperintensity in bilateral hippocampal areas, insular and cingulate cortices(picture). Otherwise, he doesn't have other long-term neurological deficits.

Conclusion: The retrograde amnesia in this patient might be related to bilateral hippocampal lesion, which is similar to one previous case report. The mechanisms of GLA-related neurotoxicity remain unclear but have been proposed to be caused by GLA itself, its metabolites or imbalance between glutamate and glutamine caused by GLA. The hippocampus plays a key role in the formation of new memories by NMDA-type glutamate receptors. It may need further surveying and research to explain the MRI finding of our patient.