G19 Globus Pallidus Necrosis Unrelated to Carbon Monoxide Poisoning: Retrospective Analysis of 24 Cases of Basal Ganglia Necrosis

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After attending this presentation, attendees will understand that, in contrast to textbook descriptions of globus pallidus necrosis, this lesion is rarely associated with and certainly not specific for Carbon Monoxide (CO) poisoning.

This presentation will impact the forensic science community by critically examining the assertion that globus pallidus necrosis is a typical finding of CO poisoning and will contribute to the understanding of causes and mechanisms of globus pallidus injury.

It is well demonstrated that CO poisoning can cause central nervous system injury in a variety of locations, including the basal ganglia, cerebral cortex, hippocampus, and cerebellum, with delayed CO exposure more associated with globus pallidus, basal ganglia, and white matter injury (e.g., Grinker's myelinopathy). Globus pallidus necrosis, especially when seen bilaterally, is described in forensic textbooks as a lesion characteristic of, or classically seen in, delayed CO poisoning. However, this description conflicts with a number of published reports. A literature search revealed that globus pallidus necrosis has been observed in a wide variety of cases, including different types of drug overdose, post-anesthesia, in children after cardiac surgery, prolonged coma, cerebral arteriosclerosis, hemolytic uremic syndrome, acute renal failure, metabolic disorders, viral infections, and wasp stings.

To test the hypothesis that globus pallidus necrosis is characteristic of CO poisoning, this study examined autopsy cases from the King County Medical Examiner's Office between 1994 and 2013 in which globus pallidus or basal ganglia necrosis was photographically documented. There were a total of 39 cases, in which 15 showed unilateral hemorrhagic lesions of the basal ganglia which were subsequently eliminated from analysis, since they were either due to hypertensive stroke or trauma and since none were related to carbon monoxide exposure. This left 24 cases with necrotic or cystic lesions of the globus pallidus or basal ganglia, 14 of which had bilateral lesions. For comparison, two cases of delayed CO poisoning were identified.

The 24 cases of necrotic basal ganglia lesions were examined for the associated or causative disease or injury with the following results: nine were drug-related deaths or had significant drug abuse history; six were due to hypertensive and atherosclerotic cardiovascular disease; two were due to Huntington's-like disorders; and two were due to asphyxia (one infant with positional asphyxia and one adult with compressional asphyxia). Of the remaining five cases, there was one case of each of the following: remote trauma; delayed drowning; rheumatic heart disease; chronic ethanolism; and cerebral artery gas embolism in a scuba diver. Seven of the 24 cases had a clinical diagnosis of anoxic encephalopathy. No cases were identified with basal ganglia or globus pallidus necrosis associated with CO poisoning, and the single case dying after prolonged coma following CO monoxide poisoning had no evidence, gross or microscopically, of globus pallidus or basal ganglia necrosis.

The results of this study indicate that globus pallidus and basal ganglia necrosis is a consequence of cerebral hypoxia from a variety of causes and is not specific for any type of injury. However, selective hypoxic injury of the basal ganglia is not a common feature of global cerebral hypoxia and is likely due to factors of cerebral blood flow that are not well understood. Finally, this study provides no support for considering globus pallidus necrosis to be characteristic of CO poisoning.

Carbon Monoxide Poisoning, Cerebral Hypoxia, Globus Pallidus Necrosis

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