PP – 05

Severe middle cerebral artery infarction following by carbon monoxide poisoning

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Objective: Carbon monoxide (CO) is a colorless, odorless, tasteless, non-irritating yet poisonous gas; it is a product due to the incomplete combustion of hydrocarbons. CO poisoning is a major cause of death following attempted suicide. It causes tissue injury by several known mechanisms, such as tissue hypoxia, tissue ischemia, cellular asphyxia, and reperfusion injury. The central nervous and cardiovascular systems are most susceptible to hypoxia associated with carbon monoxide intoxication. However, ischemic stroke following CO poisoning is rare in literature. Thus, we herein report a case of middle cerebral artery (MCA) infarction after severe CO poisoning.

Case Report: A 32 year-old female was found to be unarousable on the bed by herself, with after-flaming charcoal nearby. Her family lost contact with her for two days. When sent to the emergency department, she was noted to have hyperthermia (BT: 39.1 °C), comatous conscious (Glasgow Coma Scale score of 3), with stable blood pressure and heart rate. Pupil sizes revealed bilateral 4.0 mm with light reflex. Initial treatment included mechanical ventilation support. Laboratory test results showed elevated WBC count (18200/ul), elevated troponin I (1.9 ng/mL), lactatic acidosis, elevated CPK, and acute kidney injury (Cr: 3.8 mg/dL). Toxico-examination revealed only elevated carboxyhaemoglobin (3.9 %). Brain CT surprisingly revealed bilateral globus pallidus hypo-density and extensive hypo-density over right middle cerebral artery territory. Furthermore, diffuse brain swelling with midline shifting was also noticed. Under the impression of severe right MCA infarction, we suggested craniotomy for her. Unfortunately, she died after operation because of multiple organ failure.

Conclusion: Ischemic stroke is a rare complication of carbon monoxide poisoning. In the literature there are limited cases reported on immediate stroke coexisting with carbon monoxide poisoning. Current hypothesis are: 1) oxidative damage induced micro-vascular impairment. 2) CO exposure caused pro-coagulant activity leading to thrombosis. A cohort study before demonstrated an association between CO poisoning and an increased risk of long-term ischemic stroke. CO poisoning may act as a trigger in patients who are predisposed to stroke. Further large-scale study of the mechanism through which CO poisoning affects ischemic stroke is necessary.