Carbon monoxide

General information

Carbon monoxide (CO) is the largest source of death from poisoning in the U.S.A.

The normal cellular function requires ≈ 5 ml O2/100 ml blood. Blood normally contains ≈ 20 ml O2/ 100 ml.

CO binds to hemoglobin (Hb) with an affinity ≈ 250 times that of O2, and it causes a left shift of the Hb/O2 dissociation curve. It also binds to intracellular myoglobin. Only ≈ 6% of patients show the classic “cherry-red” color of blood.

Clinical findings

Diagnostic studies

EKG changes are common, usually non-specific ST-T wave changes.

In cases of severe intoxication, CT may show symmetrical low attenuation in the globus pallidus.

Complications

Approximately 40% of patients exposed to significant levels of CO die. 30–40% have transient symptoms but make a full recovery. 10–30% have persistent neurological sequelae including CO-encephalopathy (may be delayed in onset) – impaired memory, irritability, parietal lobe symptoms including various agnosias.

Brain lesions:

1. white matter lesions:

a) multifocal small necrotic lesions in deep hemispheres

b) extensive necrotic zones along lateral ventricles

c) Grinker’s myelinopathy (not necrosis)

2. gray matter lesions:

a) bilateral necrosis of globus pallidus

b) lesions of hippocampal formation and focal cortical necrosis

In previous studies, carbon monoxide (CO) poisoning showed an imbalance between cerebral perfusion and brain metabolism in the acute phase and the brain temperature (BT) in these patients remained abnormally high from the acute to the subacute phase. As observed in chronic ischemic patients, BT can continuously remain high depending on impairments of cerebral blood flow and metabolism; this is because heat removal and production system in the brain may mainly be maintained by the balance of these two factors; thus, cerebral white matter damage (WMD) affecting normal metabolism may affect the BT in patients with CO poisoning. Here, we investigated whether the BT correlates with the degree of WMD in patients with subacute CO-poisoning. In 16 patients with subacute CO-poisoning, the BT and degree of WMD were quantitatively measured by using magnetic resonance spectroscopy and the fractional anisotropy (FA) value from diffusion tensor imaging dataset. Consequently, the BT significantly correlated with the degree of WMD. In particular, BT observed in patients with delayed neuropsychiatric sequelae, a crucial symptom with sudden-onset in the chronic phase after CO exposure, might indicate cerebral hypo-metabolism and abnormal hemodynamics like “matched perfusion,” in which the reduced perfusion matches the reduced metabolism [1)].

1)
Fujiwara S, Yoshioka Y, Matsuda T, Nishimoto H, Ogawa A, Ogasawara K, Beppu T. Relation between brain temperature and white matter damage in subacute carbon monoxide poisoning. Sci Rep. 2016 Nov 7;6:36523. doi: 10.1038/srep36523. PubMed PMID: 27819312.