

Images

Neurological damage caused by carbon monoxide intoxication

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A patient 27 years of age rescued by firemen from a fire in public premises with immediate attention from the mobile ICU team. Glasgow 8 with non-reactive and mydriatic pupils. Oro-tracheal intubation (OTI) could only be performed with a number 7 tube, given that tube of a higher number was unable to pass through the glottis. The aspirations of respiratory secretions were full of soot. Cyanokit (5 grammes) was also administered. The patient was subsequently taken to our hospital, where hyperhydration was initiated via a central line in the right subclavian vein; steroids, antibiotics and antitetanus immunization were administered. The patient presented with second degree surface and deep burns in upper limbs and face (18%). Patient was stable, with adequate sedoanalgesia and mechanical ventilation and thorax and abdomen normal on physical examination.

Three hours later, nonreactive left anisocoria, was observed leading to cranial computerized tomography scan (CT) (figure 1), showing significant cerebral oedema and base node infarction.

Given the persistence of neurological deterioration after 8 hours cranial CT scan was repeated (figure 2). In addition to the abovementioned lesions, images showed bilateral hypodense lesions in the interphase of vascular cerebral areas of the middle and posterior cerebral artery.

Seventy-two hours later the patient evolved to brain death.

It is well known that carbon monoxide causes neurological damage; however, this has rarely been reflected in complementary imaging tests.

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Figure 1.



Figure 2.