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Altered neurocognitive functions in a patient with carbon monoxide poisoning☆

Funciones neurocognitivas alteradas en paciente intoxicada por monóxido de carbono

Dear Editor:

Carbon monoxide (CO), released into the environment by the incomplete combustion of hydrocarbons, forms carboxyhaemoglobin (HbCO) when it comes into contact with haemoglobin (Hb) in the blood. HbCO reduces the formation of oxyhaemoglobin, impeding the transportation of oxygen to body cells and leading to hypoxia, anoxia, and death when the concentration increases. As CO is an invisible and highly toxic gas, death can arrive before the subject realises anything is happening (silent killer). In industrialised countries, CO is the principal cause of death by poisoning (suicides are 3.4 times more frequent than accidents). In Mexico, an estimated 233 deaths annually are due to toxic gases, of which 166 are accidental and 5 are suicides; however there is reason to believe that CO deaths are under-reported, given the non-specific nature of clinical manifestations, the lack of pathognomonic signs or symptoms, and the dearth of diagnostic equipment.[1–3] In addition to underdiagnosis, neuropsychological symptoms of CO poisoning are largely unknown, even though damage may remain for several years after the event.

Our patient was a 30-year-old woman with a bachelor's degree who was poisoned by CO emanating from a malfunctioning gas heater, which caused the death of her partner and diverse sequelae to herself. Her cardiovascular system was unscathed, but her vision was damaged and her central nervous system (CNS), especially neurocognitive function, was severely affected. She was diagnosed with upper motor neuron syndrome, right hemiparesis secondary to hypoxic—ischaemic encephalopathy, and disability in activities of daily living.

Results of the neurocognitive evaluation. INTELLIGENCE QUOTIENT: Verbal IQ = 66; performance IQ = 60; full-scale IQ = 60; verbal comprehension index = 66; perceptual organisation index = 69; processing speed index = 62; working memory index = 86. These scores are extremely low, which means these aspects of IQ were severely affected. Working memory, though less affected, was below average, indicating an inability to learn new information and communicate with others. *LURIA NEBRASKA NEUROPSYCHOLOGICAL BATTERY*: Sensory aphasia, predominantly auditory aphasia and anomia. *Abbreviated Barcelona Test*: Performance on the various tasks assessed was poor or very poor. *REY-OSTERRIETH COMPLEX FIGURE TEST*: Left hemispatial neglect and apperceptive visual agnosia; the patient was unable to draw a line of the figure. *Orientation*: severely affected in all 3 spheres. *Higher mental functions*: **Attention*: short attention spans, easily distracted; auditory attention: fluctuating; visual attention: incapable of perceiving a picture according to Gestalt laws, image segmentation. **Language comprehension*: fluctuating and severely impaired. The relationship between 'signifier' and 'signified' was not preserved. *Language fluency*: not very fluent, unspontaneous, only answered questions. The patient provided incorrect, unreal, and incoherent information but was able to complete some familiar series (days of the week, months of the year, numbers 1-10). The patient presented anomia, paraphasia, perseverations and confabulations, but not dysarthria; *grammatical structures*: she produced poorly structured sentences containing the 3 main components (subject, verb, and predicate). **Writing*: visuospatial problems, left hemispatial neglect, micrography, automatisms, with structural defects. Numbers and letters crowded together and overlapped. During the dictation test, the patient wrote only mono- and bisyllabic words, and when she was required to copy some text, she was only able to identify one consonant from a sentence. Dissociative writing: in regards to reading and writing, the patient was incapable of connecting signifiers with signifieds. **Reading* and **executive functions*: severely impaired. **Calculations*: the patient's ability to do mental and written arithmetic was severely affected. She was unable to understand numerical abstraction and had no understanding that a 1 or a 0 could be either a unit or a decimal, as in 0.01, 10, 100, or 1000. She was able to write the numbers from 1 to 10 but did not identify them as symbols or do mathematical operations with them, and could not classify one as 'more' or 'less' than another. **Memory*: The patient's episodic memory was severely impaired and she produced confabulations with unreal content. Her semantic memory was also severely impaired and characterised by anomic aphasia. **Thinking*: concrete. **Visuospatial ability*: severely impaired. Incapable of distinguishing 'right', 'left', 'in front', or 'behind', the patient was only able to identify 'up'. In addition, she could only visualise the top third of the right side of a piece of letter-size paper in a horizontal position. **Body*

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scheme: the patient only recognised a few large parts of the body and their functions; she was unable to identify any others. *Behavioural aspects*. **Attitude*: the patient had normal hearing and ordinary voice characteristics, childish behaviour, impulsiveness, and simple, automatic, incorrect, apathetic responses. The patient presented several continuous tics such as sticking out her tongue, and she answered questions by trial-and-error guessing.

She showed severe neuropsychological alterations after being found unconscious due to the effect of carboxyhaemoglobin, which led us to deduce that CO had damaged a number of brain structures. This theory was further supported by CT and MRI findings of damage to the cerebral parenchyma and multiple focal areas in the cortical grey matter and subcortical white matter of the frontoparietal lobes towards the convexity and occipital lobes (predominantly on the left side), the left parahyppocampal gyrus, and both cerebellar hemispheres, areas which have traditionally been associated with neuropsychological functions,[1,4–9] especially the semioval centre, which has been significantly correlated to cognitive sequela.[10]

It has also been reported that the presence of lesions in the globus pallidus and the reticular part of the substantia nigra after CO poisoning indicates a poor cognitive state, which is associated with extensive injury to both grey and white matter, in addition to damage to the nigrostriatal pathway.[11]

Neuropsychological alterations are linked to the presence of persistent cephalic disorders, fatigue, a reduction in visual perception, manual dexterity, a decline in memory, attention, and the abilities needed to drive, and sleep disorders, especially insomnia[7]; all these symptoms were clearly seen in our patient. A polysomnography revealed asymmetric sleep spindles, parasomnias, and no insomnia. Although neurocognitive alterations might go undetected at first, they may become evident at some point. Some patients may present serious neuropsychiatric disorders after more than 10 months of apparently normal function. This condition is called 'delayed encephalopathy' or 'delayed neuropsychiatric syndrome'.[10] From a neurospychological and bioethical point of view, a neurocognitive evaluation and long-term follow-up is necessary in these patients, as many can be rehabilitated with consequent improvement in quality of life.

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Conflicts of interest

The authors have no conflicts of interest to declare.

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