Neurosciences Quiz

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A young women who couldn't see or move

Case Presentation

A 17-year-old girl was found comatose after a fire in her house. She had no external burns or injuries. She was intubated and mechanically ventilated. She received appropriate care in the intensive care unit (ICU). After extubation, she was recognized as having 2 neurological problems. An MRI of the brain is shown (**Figures 1 & 2**).

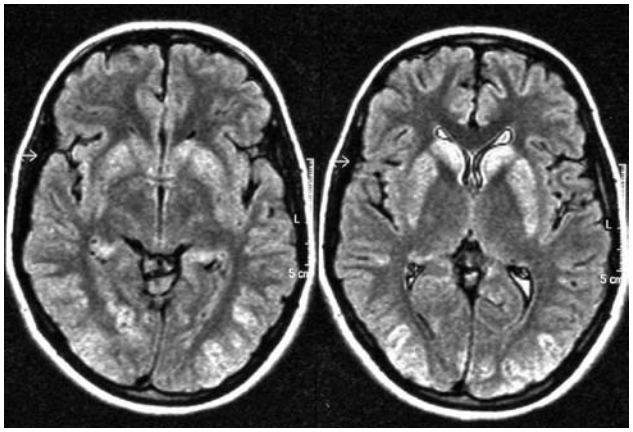

Figure 1 - Axial FLAIR MRI images.

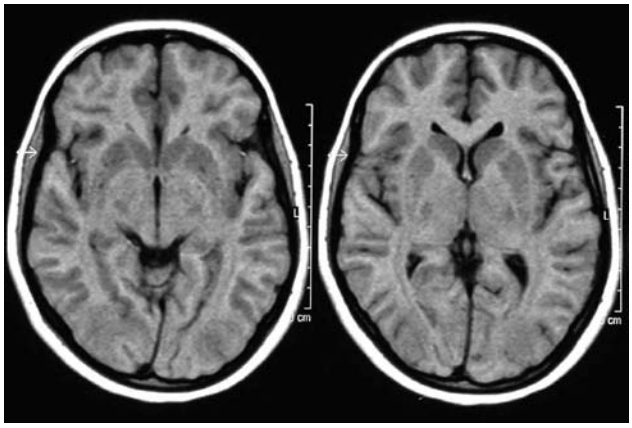

Figure 2 - Axial T1-weighted MRI images.

Questions

1. What are the 2 principal abnormalities? What is the possible underlying cause?

2. What are the expected neurological deficits in this patient?

3. What are the mechanisms of nervous system damage?

4. What is the treatment?

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Answers & Discussion

1. There are bilateral symmetrical changes in the basal ganglia, which are of high signal intensity on fluid attenuation inversion recovery (FLAIR) images (**Figure 1**) and of low signal intensities on T1 weighted-images (**Figure 2**). Also, there are bilateral occipital changes in the cortex, which are relatively symmetrical. These changes are compatible with carbon monoxide (CO) poisoning with secondary hypoxic damage.

2. Such patients might present with visual impairment (occipital lobe involvement) and movement disorder (basal ganglia involvement). Our patient developed cortical blindness with tremor, rigidity, bradykinesia, and difficulty walking without support. She was started on Sinemet and aggressive physiotherapy. She developed severe neuropsychiatric manifestation with irritability, agitation, crying spells, and so forth. She was started on a small dose selective serotonin reuptake inhibitor (escitalopram 10 mg daily). She gradually improved, including her vision. Six months later, she was completely off medications and her vision was much better. She was only left with difficulty reading.

3. a. Carbon dioxide can induce tissue hypoxia, which may be followed by a reoxygenation injury to the CNS through the production of partially reduced oxygen species. This in turn can oxidize essential proteins and nucleic acids, resulting in typical reperfusion injury.[1]
   b. Carbon monoxide (CO) exposure has been shown to cause degradation of unsaturated fatty acids (lipid peroxygenation), leading to reversible demyelination of the CNS.
   c. Carbon monoxide exposure also creates substantial oxidative stress on cells, with production of oxygen radicals resulting from the conversion of xanthine dehydrogenase to xanthine oxidase.
   d. Carbon monoxide also interferes with peripheral oxygen utilization by inactivating cytochrome oxidase in a manner similar to, but clinically less important than, cyanide. Carbon monoxide and cyanide poisoning can occur simultaneously in patients following smoke inhalation, and their combined effects on oxygen transport and utilization appear to be synergistic.[2]

4. Based on chemical and pathophysiological data, oxygen is the "natural antidote" and the half-life of carboxy hemoglobin (COHb) is inversely proportional to the pressure and concentration of oxygen in the inspired air.[3] The half-life is approximately 5 hours with ordinary room air, 90 minutes with 100% O_2 at one atmospheric pressure, and 20 minutes for 100% O_2 at 2 atmospheric pressure. Since the clinical signs and symptoms of CO toxicity are nonspecific, all suspected victims should be treated with 100% O_2 inhalation immediately after blood is drawn for COHb content. Hyperbaric oxygen therapy (HBO) should be given to patients with severe poisoning (patients in coma, neurologic deficit, or COHb >40%) regardless of COHb level, pregnant women irrespective of signs and symptoms, patients with signs of cardiac ischemia or arrhythmias, and patients with symptoms that do not resolve with normobaric oxygen after 4-6 hours.[4]

References

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