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Speech therapy and hyperbaric oxygen for aphasia after carbon monoxide intoxication



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ABSTRACT

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Acute carbon monoxide (CO) intoxication may result in delayed neurological sequelae, which can include amnesia, ataxia, aphasia, emotional lability, disorientation, dysphagia, and other manifestations. A 27-year-old man reported symptoms of aphasia with agraphia and alexia in a review after CO intoxication. The patient received outpatient speech therapy, as well as repeated sessions of hyperbaric oxygen for 15 days, interspersing speech therapy with hyperbaric oxygen therapy for two months. After this period of combined treatment the aphasic symptomatology remitted, and oral and written language was normal. The complete disappearance of aphasia with agraphia and alexia confirms the efficacy of the combined intervention. More data from large clinical studies are needed to assess the outcomes of hyperbaric oxygen treatment in patients with delayed neurological sequelae after CO intoxication, but this case suggests it may be a good therapeutic option in combination with specific speech therapy.

KEYWORDS: aphasia; carbon monoxide; delayed neurological sequelae; hyperbaric oxygen; speech therapy

INTRODUCTION

Speech therapy should be individualized according to the specific needs of the patient. Problems such as those reported here, in which speech therapy was used in combination with hyperbaric oxygen therapy to treat aphasia caused by carbon monoxide (CO), are uncommon. CO is a colorless, odorless gas that does not irritate the airway and is generated by the incomplete combustion of materials containing carbon [1]. Acute CO intoxication is rare and sometimes underdiagnosed in Spanish hospital emergency services [2]. Current knowledge of the clinical manifestations induced by CO inhalation and the quantification of carboxyhemoglobin (COHb) in blood or by pulse co-oximetry are fundamental elements in the detection and adequate treatment of this condition.

The annual number of CO intoxications in Spain is difficult to assess, as there are little data and/ or robust epidemiological approximations. It has been suggested that there may be more than 2,500 cases per year, of which 100 would be fatal, along with overdoses from drugs of abuse as the main causes of toxicological deaths [3]. Our group reported one of the largest series of CO intoxications in Spain, with data collected between January 1996 and June 1997 [4].

On arrival at the emergency room, CO patients generally report headache (90% of cases), nausea, vomiting, diarrhea, severe asthenia, and other non-specific symptoms, which may lead to a misdiagnosis of influenza in winter or gastroenteritis in summer. In more severe acute cases, patients present with obnubilation, muscular impotence, syncope, lipothymia, or varying degrees of coma, which may lead to death. Some patients may have arrhythmias or acute coronary syndrome. The physical examination may be normal in mild intoxications or show a coma without neurological focus, or with hypertonia, hyperreflexia, or pyramidal syndrome in severe cases [1].

After acute intoxication and discharge from the emergency room some patients may present delayed neuropsychiatric sequelae (DNS) when, after apparent recovery from the acute episode, any of the following late symptoms may appear: memory loss, confusion, ataxia, aphasia, seizures, emotional lability, disorientation, hallucinations, Parkinson's, mutism, cortical blindness, psychosis, dysphagia and other motor manifestations; some patients can improve with hyperbaric oxygen (HBO₂) treatment [5,6]. The latency period after acute intoxication for the appearance of DNS is usually between two and 40 days, although the reported incidence varies between 2% and 47% of patients. Structural brain lesions have been found to justify the injury only in some cases [7].

We report the first case of aphasia with transient agraphia and alexia described in Spain, with DNS due to CO treated by speech therapy and repeated sessions of hyperbaric oxygen, which were alternated in order to seek greater effectiveness of the overall treatment plan.

CASE REPORT

A 27-year-old man with no previous medical history of interest, went to the emergency room reporting headache, nausea, discomfort, and prostration secondary to accidental CO inhalation due to the poor combustion of a water heater. The physical examination was normal, and tests showed a carboxyhemoglobin (COHb) level of 21%, which was measured at 1% eight hours later. Initially the patient was treated with 100% oxygen using a reservoir mask and discharged to home totally asymptomatic at 12 hours, with a diagnosis of acute CO intoxication. At a scheduled outpatient review after 15 days, the patient reported symptoms compatible with aphasia, which had started two days before, with a normal motor neurological examination. Urgent magnetic resonance imaging excluded space-occupying lesions and demyelinating phenomena. The internal medicine and neurology departments were consulted, and an assessment was made by a speech therapist.

Tests included a blood count, biochemistry with electrolytes, liver, kidney and thyroid function tests, C-reactive protein, vitamin B12, folic acid, angiotensin-converting enzyme, and serology for *Borrelia* and *Treponema pallidum*, all of which were negative or normal. Cerebrospinal fluid (CSF) analysis showed normal proteins and glucose, with normal culture and cytology, as well as antineuronal antibodies in blood and CSF. Thoracoabdominal computed tomography and cerebral positron emission tomography revealed no abnormalities. The electroencephalogram showed normal central activity. For these reasons, space-occupying lesions and metabolic or degenerative diseases were excluded.

Language was evaluated through an initial structured interview and the Boston Naming Test [8]. In the initial interview by the speech therapist, speech was examined in a contextualized manner, demonstrating that the patient presented acceptable oral expression with errors. His attention span and concentration were slightly impaired, and he spent more time than usual performing tasks. Natural and conversational gestures showed no apraxia. The orolingual and facial muscles showed slowed movement. There was good control of the respiratory mechanics, with a lower middle thoracic pattern. Oral expression was relatively fluid, with discrete alterations in articulation such as omissions, substitutions, and distortion of phonemes in spontaneous and repeated language. Paraphasia was observed which, in some cases, were so striking that they originated neologisms. The patient demonstrated difficulty in naming, which improved substantially with phonetic help and in repeated language. There were no alterations in prosody, with adequate control of the pauses of the spoken chain, with adequate handling of vocal inflections and their intensity. Sentences were simple and short, with few elements, and conversational turns were respected.

In relation to comprehension, the patient discriminated body parts, objects of habitual use, colors, letters, and numbers, and simple words belonging to different semantic categories (food, animals, and others). Oral comprehension was preserved for simple, five- to six-element phrases and a simple lexicon, but was lost in longer and more elaborate sentences. There was difficulty in making three or more serial orders and in following conversations with several interlocutors. In written language, paragraphia was observed in copying and dictation and in spontaneous writing, without alterations in the writing stroke. Reading was slow and hesitant, with more striking errors in sentences and texts than in isolated words or pseudowords. Reading comprehension was altered in simple texts, with difficulty in extracting the main ideas. The ability to calculate was not affected. After an assessment by the multidisciplinary team, the patient was diagnosed with mixed aphasia with motor predominance with alexia and agraphia in relation to a DNS due to CO.

The patient received outpatient speech therapy and 15 sessions of HBO₂ at 2.5 atmospheres absolute for 90 minutes in the hyperbaric chamber of a hospital in northern Spain, to which he was transferred on the corresponding day by ambulance. It was decided to alternate the speech therapy sessions (three days a week) with the hyperbaric therapy sessions (three days a week, including Saturday). The combined treatment lasted five weeks and was continued until the eighth week with speech therapy alone.

The main goal of speech therapy was optimum recovery of communication skills and improvements in all areas of emotional, social, family and work life in individual 45-minute sessions for three days a week for two months, interspersed with HBO₂. In the last two weeks of treatment, there were five group sessions with other aphasic patients. Two months after the combined treatment, the evolution was very positive, with aphasic symptoms remitting. The patient presented oral and written language within normal parameters at discharge. Table 1 summarizes the findings in the speech therapy assessment before and after treatment.

DISCUSSION

As stated, 15 days after acute CO intoxication DNS was assessed in this patient by referral to a specialized outpatient clinic with a specific DNS evaluation and detection protocol [4]. This may be fundamental for the detection of DNS, and we suggest it should form part of standard procedures for CO intoxication. In our experience 93% of patients invited to a review of CO intoxication attend, which shows the concern generated by acute CO intoxications; 17% of cases have symptoms compatible with DNS, the most frequent of which are memory loss, irritability, and difficulties in concentration [4]. However, in the 25 years of experience of our clinical toxicology outpatient clinic, we have seen hardly any cases of mixed aphasia such as that described here and none with the specific features described.

Although aphasia due to DNS of CO intoxication has been described, the incidence is very low and, until fairly recently, there were doubts that it was due to the neurological damage induced by CO [9]. Today we know there are cases accompanied by apraxia, dysphagia, muscle stiffness and urinary

ATTENTION AND CONCENTRATION	INITIAL EVALUATION Disperse, not maintained for more than 10 minutes	FINAL EVALUATION Sustained attention Collaborates and is involved in activities.
OROLINGUAL APRAXIA	Slowed, especially lingual muscles.	None
PHONEME ARTICULATION	Errors of omission, distortion, and substitution in spontaneous and repeated language.	All articulatory patterns acquired and consolidated
PHRASE-MAKING	Short and grammatically simple statements.	Use of elaborate phrases without difficulty.
DIFFICULTY IN NAMING	Anomie for basic category elements.	Able to denominate without errors.
LISTENING	Preserved for simple elements of different semantic categories and for simple phrases of 5-6 elements. Difficulty in placing serial orders (three or more).	Entirely preserved. Understanding and use of complex phrases, with extensive vocabulary. Carry out serial orders without difficulty.
WRITING	Paragraphia in copying, dictation, and spontaneous writing.	Entirely preserved, without errors.
READING	Slow and not very fluid, with gaps in understanding.	Good reading speed, without errors and ^o adequate understanding.

TABLE 1. Results of the speech therapy evaluation before and after combined treatment with speech therapy and hyperbaric oxygen

and fecal incontinence while, in other cases, aphasia is accompanied only by alexia [10,11]. There are various hypotheses regarding the mechanism that induces aphasia or DNS, ranging from tissue hypoxia or demyelination, through lipid peroxidation related to oxidative stress in some brain areas or a decrease in brain flow in Brodmann's areas 7,8,11 and 40 [1,5,10]. Among the limitations of the case presented here is that we cannot indicate which of these hypotheses induced aphasia. However, since a report in the New England Journal of Medicine [12] on the efficacy of HBO₂ in the prevention of DNS in acute CO intoxication which found that DNS such as headache, irritability, personality changes, confusion, and memory loss in 10 patients were resolved rapidly with HBO₂, this therapy has become accepted [10].

In summary, the experience of administering HBO_2 as an adjuvant to speech therapy in the case reported was positive, with complete disappearance of aphasia with agraphia and alexia.

CONCLUSIONS

The combination of speech therapy and HBO₂ produced very favorable results in a patient presenting with aphasia. The efficacy of HBO₂ in patients with DNS requires evaluation in large clinical studies, but this report reaffirms the suggestion that HBO₂ may be an option to combine with speech therapy to improve and shorten the symptoms of aphasia efficiently.

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