

Clinical improvement of multiple sclerosis after implementation of mild therapeutic hypothermia: A case report

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Abstract

Therapeutic hypothermia is the latest, revolutionary therapy, mostly used in intensive care units after out-of-hospital, and sometimes in-hospital, cardiac arrest due to its neuroprotective effect. This therapeutic intervention has also been used to improve neurological outcome after cerebrovascular accidents and other neurological catastrophes. Multiple sclerosis is one of the main diseases that cause neurological dysfunction in young adults due to its irreversible autonomic nervous system damage. Its main symptoms are tremors, sensory loss, weakness, ataxia and diplopia, and the progression of

these end up disabling the patient permanently, sometimes even with the implementation of immunotherapy. We report a patient with a long-standing history of relapsing-remitting multiple sclerosis, with frequent attacks every 1-3 months, that underwent therapeutic hypothermia for 96 hours after in-hospital cardiac arrest; her neurological recovery was outstanding with no neurological deficits caused by the cardiac arrest, and surprisingly, when she was assessed months after the ischemic insult, it was found that she hadn't presented a relapse of multiple sclerosis since hypothermia was implemented.

Key words: Multiple sclerosis, therapeutic hypothermia, cardiac arrest, thermoregulation.

Introduction

Therapeutic hypothermia (TH) has been studied and applied for several decades to improve the neurological outcome in patients who suffer out-of-hospital cardiac arrest (OHCA) as well as in-hospital cardiac arrest. (1-3) This intervention had been attempted via the use of cooling devices to improve symptoms of multiple sclerosis (MS), but the effects of this intervention only lasted a very short period of time. (4-6) We present a case report of a patient with longstanding history of relapsing-remitting multiple sclerosis (RRMS), who, after undergoing mild TH to treat in-hospital cardiac arrest, no longer presented symptoms related to MS, or had relapses, without MS treatment.

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Case presentation

A 61-year-old woman with important medical history of RRMS, coronary artery disease (CAD), chronic lower back pain, depression and hypothyroidism was admitted to the intensive care unit (ICU) after successful resuscitation from a cardiopulmonary arrest allegedly secondary to a ventricular dysrhythmia. She had been admitted 6 days prior to the cardiac event with a large infected decubitus ulcer. She was undergoing her sixth day of antibiotic and mechanical debridement for her decubitus ulcer when she had a sudden ventricular fibrillation. Significant laboratory data obtained during the cardiac arrest revealed a pH 7.1, pCO₂ 22 torr, pO₂ 84 torr, potassium 3.4 mEq/L, magnesium 0.6 mEq/L. She received 55 minutes of manual CPR, intravenous magnesium and medications following the American Heart Association algorithms for ventricular fibrillation until she had return of spontaneous circulation.

Upon arrival to the ICU, she was comatose. Her history was that of longstanding RRMS, with multiple relapses since its diagnosis, usually an episode every 1-2 months, and had been treated with interferon beta 1-b, with an unsuccessful response, shown through the disease's failure to remit or be controlled. Seven months prior to this admission, this therapy was discontinued because of adverse reactions; since then, the patient continued receiving only symptomatic therapy and had several MS

exacerbations requiring hospitalization. The patient was on mild TH, cooled down with hydrogel cooling device, achieving a target temperature of 32 °C in 128 minutes, where she continued for a total of 96 hours without complications. Rewarming was done over a period of 48 hours. The primary reason of her cardiac arrest was assumed to be related to a very low level of magnesium on the day of the arrest (0.6 mEq/L). Within 12 hours after rewarming, the patient was awake, alert and had no obvious new neurological deficits, evaluated with Cerebral Performance Category (CPC), which was recorded as CPC 1. After 15 days of hospitalization she was transferred back to a long-term facility, where she continued to receive physical therapy, as she did in the past. For a period in excess of 6 months, the patient was closely monitored. Surprisingly, she had not had another MS relapse since TH was used, without the use of immunotherapy. The patient is currently on no MS treatment and the disease has neither progressed nor relapsed. In addition the patient is no longer bed-bound.

Discussion

There is significant clinical data on the use of TH for post-cardiac arrest care, in hopes of this revolutionary therapy being neuroprotective and improving neurological outcome. (2,3) The use of TH in MS patients to improve symptoms and functionality was first studied in the 1950's, when a woman with MS regained her vision and mobility of a paralyzed extremity when she was submerged in cold water. (6) Ever since then, body cooling as a symptomatic therapy for MS has been studied, and several cooling garments have been designed for this purpose, but as effective as this therapy may be, body cooling effects are limited to only a few hours (1.5-4 hrs). (4)

MS is a very deteriorating disease, without an established treatment that is known to work on almost every patient and markedly unpredictable. A few of the most common and disabling symptoms

are spasticity, fatigue, weakness, paralysis, bladder dysfunction (4,7) and impaired thermoregulation, leading to hyperthermia. (4,5,7) Many of these symptoms seem to be correlated to the increase in core temperature, therefore, several investigations have been made to find the best cooling device that achieves the improvement of MS symptoms. (4,5) So far, only cooling vests have been designed to improve MS symptoms for a few hours. (4,5) It is believed that by decreasing leukocyte nitric oxide production, the conduction in demyelinated axons is intensified, therefore improving the symptoms. (8,9) Ku and coworkers studied 26 MS patients whose core temperature was decreased by 0.3-0.4 °C with 3 different cooling vests, finding that symptoms were better for a few hours after the therapy had been applied. (4)

Watson reported in 1959 that a decrease of at least 1 °F was necessary for improvement of MS symptoms. (6) However, to the best of our knowledge, every time TH has been used in the context of MS, such as with cooling vests, with a drop in temperature much smaller than that of the one achieved in the case described, and for a shorter period of time, the symptoms disappear or ameliorate only for a few hours. We believe that the greater decrease in temperature is, in part, responsible for the longer-lasting results and the major improvement in functionality in our patient. We initially used TH with the goal of obtaining better neurological outcome in a victim of sudden cardiac death. We were amazed when we realized that our patient had now improved her basal neurological condition and her response to TH was much better than expected.

Conclusions

TH proved useful in our patient with MS. Her symptom resolution and improvement in neurological findings is still not totally clear. Randomized-controlled studies are necessary to better elucidate the mechanisms involved and duration of TH that these patients may require.

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