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CASE REPORT

Targeted Temperature Management After Cardiovascular Collapse Secondary to Contrast Agent Anaphylaxis

Kontrast Madde Anafilaksisine Sekonder Gelişen Kardiyovasküler Kollaps Sonrası Hedeflenmiş Sıcaklık Yönetimi

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Abstract

Anaphylaxis is the most critical type of contrast agent (CA)–related hypersensitivity. In this report, we present a case of a 26-year-old female who experienced anaphylaxis during contrast-enhanced computerized tomography scanning. After cardiovascular collapse with asystole on the electrocardiogram, the patient was resuscitated. She received standard post-resuscitation care and an additional targeted temperature management (TTM) for clinical and radiological diagnoses of cerebral ischemia after the return of spontaneous circulation. After 19 days of treatment in the intensive care unit, she regained full consciousness. TTM can be kept in mind as an additional mode of therapy, irrespective of initial arrest rhythm in the treatment of comatose patients after cardiac arrest and cerebral hypoxia resulting from CM anaphylaxis.

Keywords: Anaphylaxis; Cardiovascular collapse; Targeted temperature management

Anaphylaxis is a severe, life-threatening allergic reaction.[1] Globally, a huge number of radiological imaging with the use of contrast agent (CA) are conducted yearly.[2] With the increase in administration of low-osmolality nonionic CAs instead of high-osmolality ionic agents, the incidence of critical acute CA hypersensitivity diminished remarkably from 0.1–0.4% to 0.01–0.04%. Nonetheless, fatality that results from anaphylaxis still can be observed in 1–3 per 100,000–1,000,000 administrations irrespective of ionicity.[1]

A case of anaphylaxis caused by CM sensitivity with resultant cardiovascular collapse is presented in this report. The patient experienced hypoxic encephalopathy and was treated by TTM as an additional mode of treatment and recovered without any neurological sequels.

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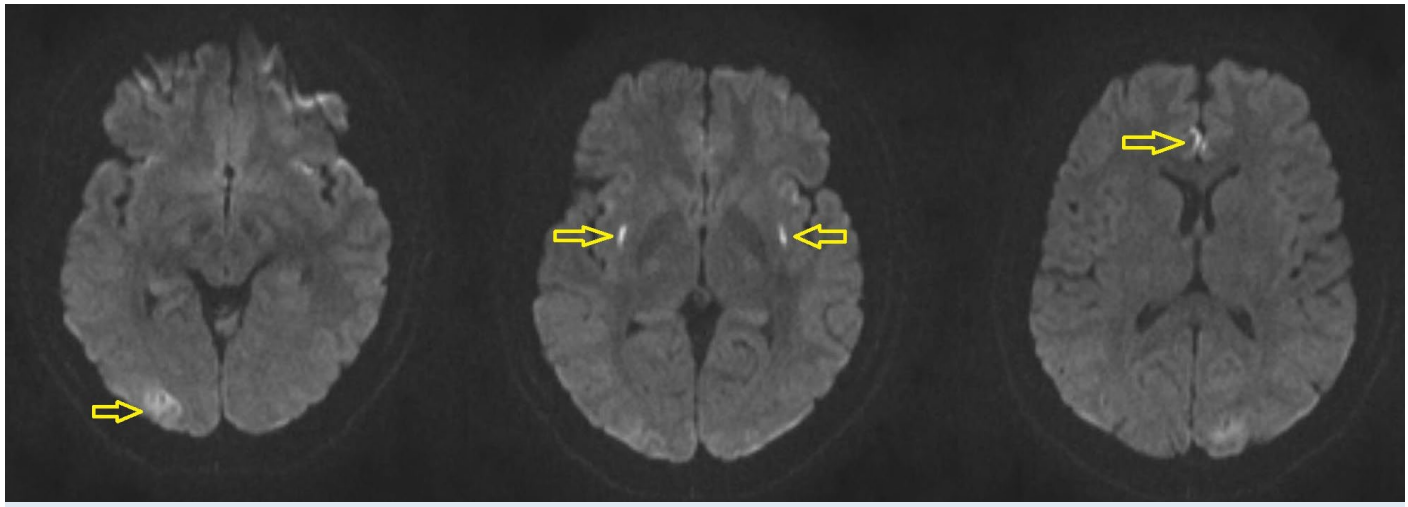

Figure 1. Diffusion MRI showing bilateral focal ischemia on the medial, frontal, parietooccipital, and perisylvian cortex.

Case Report

A 26-year-old female presenting a symptom of hirsutism was admitted to the obstetrics and gynecology clinic of our hospital. A computed tomography scan of the surrenal glands was ordered. She did not have a history of previous CM exposure. During the procedure, the patient experienced nausea and vomiting immediately after intravenous administration of 70 ml of CM (Iopromide; Ultravist® 300, Bayer Healthcare, Brussels, Belgium). Immediately, she experienced cardiopulmonary arrest. An emergent endotracheal intubation was conducted, which was followed by positive-pressure ventilation. Simultaneously, cardiac resuscitation was started, and repeated doses of 1-mg epinephrine with a total dose of 9 mg were administrated. Her initial rhythm was asystole. For the treatment of anaphylaxis, 45.5-mg pheniramine and 250-mg methylprednisolone were given intravenously. Fluid administration was carried out with lactated ringer solution via intravenous route. After the first 20 minutes of CPR, the patient had a return of spontaneous circulation (ROSC) with stable hemodynamic status under dopamine infusion. She was then admitted to the intensive care unit (ICU) following an emergent diffusion magnetic resonance imaging (MRI) was conducted. The MRI revealed bilateral focal ischemia on the medial, frontal, parietooccipital, and perisylvian cortex most probably due to hypoxia (Fig. 1). In the first hour of the ICU stay, the patient experienced generalized tonic–clonic seizures. She was in a deep coma with a Glasgow coma scale (GCS) of 3. Hypoxic brain injury was suggested as an underlying cause of her coma and an esophageal temperature probe to measure and titrate the core temperature was inserted; hence, TTM was planned as an additional treatment. It was applied by using active cooling pads that were pasted on the thoracoabdominal region and lower extremities (ArcticSun, Medivance, Inc., Louisville, Colorado). A targeted core temperature of 32°C–34°C was obtained in the second hour and continued for 24 hours. Shivering was prevented, and seizures were controlled via intravenous administration of thiopental sodium and neuromuscular blocking agents. Total serum IgE levels were within normal limits. After the completion of hypothermia, rewarming was carried out with slow increments in temperature for 8 hours. She was extubated on the third day of her ICU stay. The diffusion MRI on the eighth day showed acute-subacute infarcts with restriction of diffusion at the splenium region of the corpus callosum and both fronto-temporooccipital cortex and subcortical white substance. After the termination of the TTM, she remained unconscious for 19 days with a GCS of 8 and a cerebral performance score of 3. Neurological examination presented spontaneous movement on her left side, and she was hemiparetic on her right side. Due to the early awakening of the patient with a GCS of 13 and a cerebral performance score of 2 on the 19th day of the ICU stay, percutaneous endoscopic gastrostomy was canceled. Thereafter, she was discharged from the ICU and admitted to the neurology department, where she would receive a physiotherapy and rehabilitation program for 2 months due to her balance and walking difficulties.

Finally, 60 days after the event, she was discharged from the hospital without any cognitive or motor sequels with a cerebral performance score of 1. The control diffusion MRI in the eighth month revealed normal intracranial findings (Fig. 2).

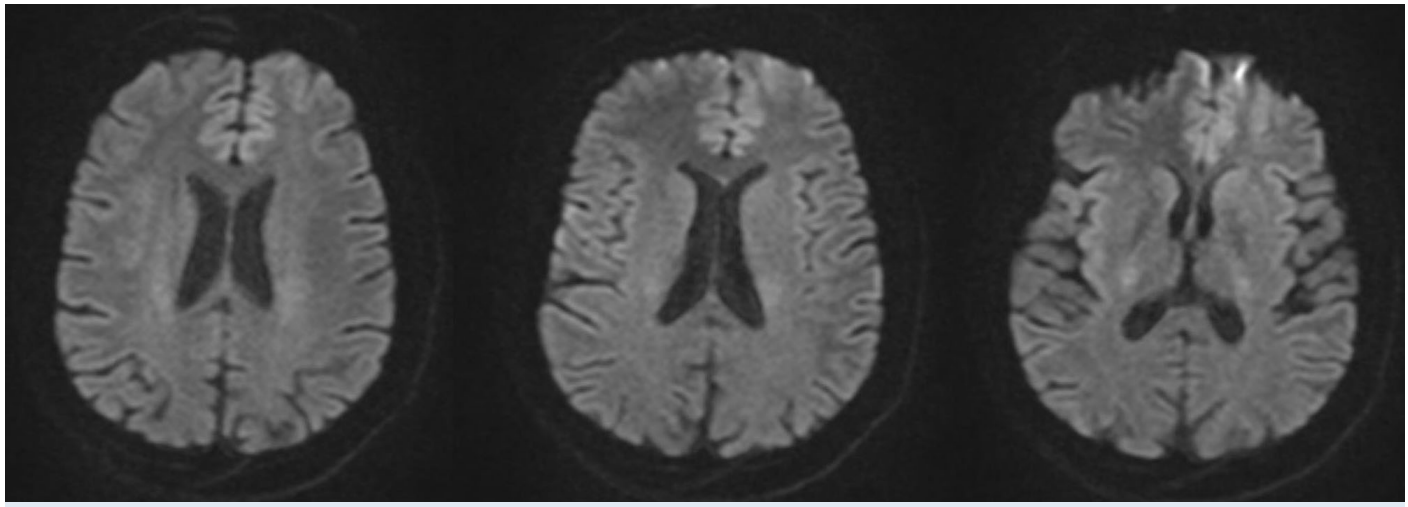

Figure 2. Control diffusion MRI in the eighth month showing no ischemia in the regions revealing diffusion defects in the previous MRI.

Discussion

Adverse reactions to contrast media, although uncommon, are often high-acuity events and potentially life-threatening.[3] Although their frequency is low, allergic adverse reactions to CAs are associated with morbidity.[4] The mechanisms of rapid and late reactions to CA have been speculated for years. It is generally agreed that radio-CAs may result in both allergic and nonallergic reactions.[2] Anaphylaxis is a systemic acute reaction with a large spectrum of mechanisms, clinical presentations, and severity caused by rapid, systemic release of mediators from basophils and mast cells. Mast cells are the primary effector cells of acute hypersensitivity reactions, and β-tryptase is the main protease in mast cells, which also presents in basophils in trace amounts. Release of B-tryptase and many other mediators from cytoplasmic granules may be induced by immune reactions. These reactions can be IgE-dependent or IgE-independent.[4] However, it has been agreed that immunologic, IgE-mediated mechanisms are seen particularly in cases of severe and mortal reactions.[2] In our case, serum total IgE levels were normal despite that it was a life-threatening anaphylaxis. However, reportedly, even the relatively high levels of IgE, specific for a received allergen, may not result in serum total IgE elevations.[4]

Risk factors for CA hypersensitivity, which is severe, include a history of previous CA hypersensitivity, previous allergic reactions that require medical treatment, asthma, beta-adrenergic blocker use, female gender, Mediterranean and Indian ethnicities, CA type, dose and route of administration, and associated malignant tumor, cardiovascular, renal, hematologic, autoimmune, and metabolic diseases.[1,2] Kim et al.[1] reported that the risk factors for anaphylactic shock are older age, previous exposures to CA more than once, and use of iopromide. Even so, Palmiere et al.[2] in the light of the data obtained from the literature, reviewed that there had been previously received contrast compounds in only a small number of the mortal cases, whereas most severe anaphylaxis cases were the patients who immediately reacted on first exposure. Recent literature shows that more than half of the cases had no adverse drug reactions in past ICM administration.[5] Our patient did not have a history of previous CA exposure and she has some of the risk factors including Mediterranean ethnicity, female gender, and metabolic disorders.

Spasms in smooth muscles of the respiratory and gastrointestinal tracts, escalated vascular permeability, and vasodilation are the problems encountered after anaphylaxis. Several clinical manifestations of anaphylaxis include urticaria, itching, bronchospasm hypotension, angio-edema, syncope, and shock.[6] If sudden hypotension after exposure to a familiar allergen happens, diagnosis of anaphylaxis and risk of development of cardiovascular collapse must be considered.[1] Pan-arterial anaphylaxis affecting cerebral arteries and causing changes that resemble those encountered in the coronary arteries may take part in the pathophysiology of loss of consciousness as well as circulatory arrest followed by hypoxic damage to the nervous tissue.[7] Moreover, increased vascular permeability and intracranial pressure (ICP) develop after hypoxic brain damage.[8] The treatment modalities after anaphylaxis aim at all pathophysiological components. In our case, we added TTM to decrease the brain damage followed by it.

Currently, hypothermia is extensively utilized as a part of intensive therapy for correction of ICP in patients with traumatic brain edema.[8] Hypothermia has shown to be an effective method for cerebral protection after cardiac arrest.[9] It reduces the metabolic rate to enhance the supply and demand of O_2. Moreover, it causes excitotoxicity reduction, prevention of ATP depletion, inhibition of inflammation, and reduction of free radical production and intracellular calcium shift to prevent apoptosis. In the injured brain, it acts through reduced inflammation, reduced permeability of the blood–brain barrier, decrease in cerebral edema, and thus decrease in ICP.[9]

Clinical studies have shown that hypothermia in neurologic injuries, including stroke, subarachnoid hemorrhage, and traumatic brain injury, could be beneficial.[6] Although TTM is very beneficial after cardiac arrest with shockable rhythms, the International Liaison Committee on Resuscitation and the American Heart Association suggest TTM for adults who experience in-hospital cardiac arrest with any initial rhythm and who do not follow commands after ROSC.[9,10] Thus, we carried out TTM although the patient was arrested in hospital with an initial rhythm of asystole. As mentioned previously, cerebral arteries are affected after pan-arterial anaphylaxis, and in our case, TTM may have added a therapeutical effect on cerebral hypoxia and thus a positive outcome. Different methods include application of gel-adhesive pads, rapid cold fluid administration, application of ice packs, or endovascular cooling to maintain targeted temperature. A recent meta-analysis reported that endovascular cooling did not optimize survival or hospital discharge rates.[11] We used the gel-adhesive pad application, which is a noninvasive method to maintain a targeted temperature.

Duration of the TTM remains controversial. Some studies found no difference between cooling for 24 or 48 hours. The recommendation on the duration of TTM in international guidelines is at least 24 hours.[11] We carried out TTM for 24 hours based on the recommendations.

Fugate et al.[12] concluded that awakening after therapeutic hypothermia is generally observed within 3 days of cardiac arrest, and they did not detect any delay, in comparison with nonhypothermia cases. However, complete awakening of our patient occurred on the 19th day of the event.

Conclusion

To conclude, physicians who are using CM must recognize and be highly knowledgeable of the proper treatment of anaphylactic reactions. Thus, radiology departments must also have the essential equipment for resuscitation.[6] Furthermore, TTM may be considered as an additional mode of treatment irrespective of initial arrest rhythm in the treatment of comatose patients with ROSC after cardiac arrest that results from CM anaphylaxis.

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Informed Consent: Written informed consent was obtained from patients who participated in this study.

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