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Poboljšanje terapijskog odgovora s dodatkom loratadina u tretmanu subseroznog eozinofilnog gastroenteritisa

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SAŽETAK

Opisan je slučaj muškarca od 45 godina, koji se inicijalno prezentirao sa simptomima akutne intestinalne obstrukcije. Dodatni dijagnostički testovi otkrili su postojanje izraženog eozinofilnog ascitesa s naglašenom eozinofilijom u periferiji, kao i postojanje zadebljanja zida želuca i crijeva, uz biopsijski verificiranu infiltraciju mukozе želuca i duodenuma eozinofilima. Nalazi su bili u skladu sa subseroznim tipom eozinofilnog gastroenteritisa, te je pacijentu ordinirana kombinacija parenteralnog metilprednizolona i oralnog loratadina. Nakon 5 dana tretmana dolazi do potpunog kliničkog, biohemijskog i radiološkog odgovora.

Ključne riječi: eozinofili, antihistaminici, ultrazvuk, tretman

CASE REPORT

Pseudo-subarachnoid hemorrhage and death after a bee sting

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ABSTRACT

We report a case of a 33-year-old woman who developed severe brain edema and pseudo-subarachnoid hemorrhage (SAH) at 36-hour follow-up after successful cardiopulmonary resuscitation for anaphylactic shock as a result of a bee sting. The patient died on the sixth day of the follow-up due to multiple organ failure and brain herniation. Our case suggests that the SAH-like findings on computed tomography scanning were not a new complication ("real" SAH) arising from the bee sting; rather, it was a pseudo-SAH related to prolonged cardiopulmonary resuscitation).

Key words: pseudo-subarachnoid hemorrhage, bee sting, resuscitation

INTRODUCTION

Throughout the world, 9.3–28.5% of sensitization cases are known to be due to hymenoptera venoms (1). In this regard, the hyperalgesic and edematogenic effects of venoms of neotropical social wasps (*Polybia paulista* and *Protonectarina sylveirae*) and honeybees (*Apis mellifera*) were reported in a recent trial (1). Unusual and more serious complications may also be seen, such as anaphylactic shock, acute myocardial infarction, acute renal failure, acute pulmonary hemorrhage, acute hemorrhagic pancreatitis, and atrial fibrillation (2). Possible neurological complications

after bee stings include optic neuritis, brachial plexus block, exacerbation of multiple sclerosis, cerebral hemorrhage, hypoxic brain injury with motor apraxia, encephalomyelopolyradiculoneuritis, and hemorrhagic vasculitis (3).

We report the case of a 33-year-old woman who developed severe brain edema and pseudo-subarachnoid hemorrhage (SAH) following successful cardiopulmonary resuscitation (CPR) for anaphylactic shock due to bee sting.

CASE REPORT

A 33-year-old female patient was transferred to our hospital from another medical center. Before the transportation, CPR was performed on the patient due to anaphylactic shock induced cardiac arrest, which was attributed to a single sting from a honeybee (*A. mellifera*). Such a clinical picture developed 15 minutes after the sting. The patient had a history of allergy and asthma. At the time of admission to our clinic, she was intubated and exhibited tonic-clonic convulsions. Her initial vital signs and laboratory values were as follows: arterial blood pressure (ABP) 124/98 mmHg; heart rate 99/min; respiratory rate 12/min; body temperature 36.8°C; white blood cell count 21.5 K/uL; aspartate aminotransferase (AST) 144 U/L; alanine aminotransferase (ALT) 114 U/L; and C-reactive protein 33.5 mg/L. Her score on the Glasgow Coma Scale was 7.

Cranial computed tomography (CT) images were obtained in the transverse plane parallel to the orbitomeatal line, without intravenous contrast

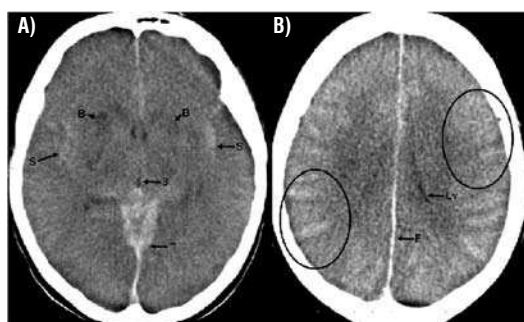


Figure 1. A) Brain CT of a 33-year-old woman on the third day after resuscitation showing low attenuation of basal ganglia (B), with obliteration of bilateral Sylvian valliculae (S) and narrowed third ventricle (3). High density areas within the bilateral Sylvian valliculae (S) and tentorium cerebelli (T) were noted; B) Brain CT section at the level of the centrum semiovale shows obliteration of the bilateral cortical sulci (elliptic regions), the slit-like appearance of the lateral ventricle (Lv), and a high-density area along the falx cerebri (Tekelioglu U, 2012)

administration, in 5 mm thicknesses for the infratentorial sections and 10 mm thicknesses for the supratentorial sections. On the second day after resuscitation, the brain CT showed diffuse low attenuation of brain parenchyma with the occlusion of cisterns and cerebral sulci, as well as narrowed ventricles (Figure 1). We graded the brain edema as severe in our patient due to obscuration of the gray-white matter and almost complete obliteration of the cortical sulci (4). High-density areas (HDAs) mimicking SAH were noted along the bilateral Sylvian valliculae and tentorium cerebelli. We confirmed that there was no intracranial aneurysm on CT angiography (data not shown). The CT values of the HDAs of the Sylvian valliculae and low-density areas of the adjacent brain parenchyma were 42.4 and 33.8 HU, respectively (Figure 2).

Mannitol and dexamethasone therapy was started. We closely monitored the patient's fluid management via central venous pressure. The patient's blood electrolytes (Na, K, Ca, Cl) were in the normal range in the first four days of her hospital stay. Her daily volume balance was well arranged. Inotropic agents were started for excessive hypotension. Severe electrolyte and laboratory (ALT, AST, urea, creatine) imbalance developed on the fifth day. The patient died due to multiple organ failure and brain herniation.

Four different possibilities were considered in the differential diagnosis of the appearance

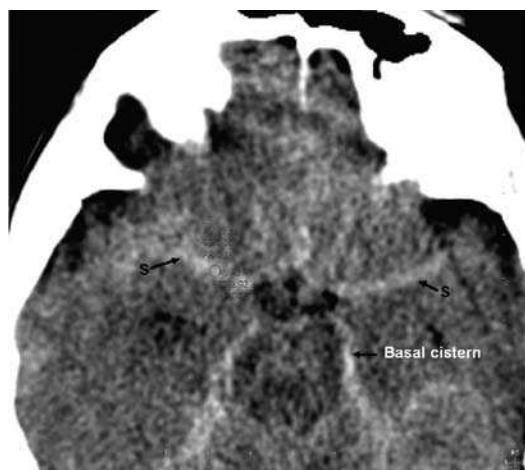


Figure 2. Brain CT section at the level of the Sylvian valliculae shows HDAs accompanying the obliteration of the bilateral Sylvian sulci (S) and basal cisterns. An oval region of interest is defined on the HDA of the Sylvian vallicula shows that the CT number is 42.4 HU, and a round or oval region of interest that is defined in the brain parenchyma just ventral to the Sylvian vallicula shows that the CT number is 33.8 HU (Tekelioglu U, 2012)

of non-contrast brain CT and underlying cause of the clinical condition in our case: blood brain barrier (BBB) was disrupted by toxins or a toxin-related autoimmune reaction causing SAH, the BBB was disrupted by ischemic intolerance during the cardiopulmonary arrest (CPA) and caused reperfusion injury, and as a result, the BBB was disrupted resulting in SAH (5,6); a silent intracranial aneurysm ruptured due to bee toxin or resuscitation-related medication (3); it was not a true SAH, but rather pseudo-SAH (4,7).

At the global level, 9.3–28.5% of sensitization cases are due to hymenoptera venoms (1). The stings cause severe pain, local inflammation, sensitization, local and systemic alterations, and occasionally even death in allergic patients (1). The possible neurological complications after bee stings include optic neuritis, brachial plexus block, exacerbation of multiple sclerosis, cerebral hemorrhage, hypoxic brain injury with motor apraxia, encephalomyelopolyradiculoneuritis, and hemorrhagic vasculitis (3).

Hypoxic or ischemic encephalopathy is the most frequent neurological complication of CPR. Additional complications include infarcts, brain swelling, and mild intracerebral hemorrhage (6). During total circulatory arrest and the post-ischemic-anoxic phase, many complex physiological and chemical disturbances take place. This is called *cerebral post resuscitation syndrome*. It may cause late heterogeneous cerebral hypoperfusion, excitotoxicity, or free radical-induced lipid membrane damage in the brain (8,9). Therefore, in etiopathogenesis of SAH due to bleeding diathesis, there may be ischemia-reperfusion injury, which can trigger subsequent events. It has also been reported that SAH might complicate the course of patients following prolonged CPR (6). We consider that the authors of this report may not have been aware that such complications could represent a case of pseudo-SAH.

Spontaneous SAH was reported after intravenous epinephrine use in a patient whose hypotension was caused by bee sting. It was explained that intravenous epinephrine infusion coupled with hypersensitivity to a bee sting may cause the rupture of a cerebral artery aneurysm (3). Epinephrine was used in our case, but we

checked the possibility of intracranial aneurysm using CT angiography, and no intracranial aneurysm was detected. We consider that the authors of this report were also not aware that their results may have represented a case of pseudo-SAH. The brain CT image in their report did not show intraventricular high-attenuation lesions, and CT value measurement was not carried out in this study.

A pseudo-SAH finding occurs when a pseudo-lesion is found on brain CT that simulates the observation of SAH. In CT without contrast agent, the cisterns and cerebral sulci are seen as hyperattenuated compared to brain parenchyma. Enlarged superficial vessels due to elevated intracranial pressure and excessive brain edema both cause this appearance via hypoattenuated manifestation of the parenchyma. In about 20% of CPA survivors, symptoms of pseudo-SAH may develop within three days of resuscitation. Assessment of the CT value may be very useful in such cases. Specifically, the values for SAH and pseudo-SAH are very different, at least on the first day, and this may help in differential diagnosis. The absence of intraventricular high-attenuation lesions also helps to differentiate the two. In our case, the first brain CT was obtained 36 hours after the onset of CPA. The brain CT showed diffuse low attenuation of the brain parenchyma with obliteration of cisterns and cerebral sulci, as well as narrowed ventricles. It was graded as severe brain edema (4). An HDA mimicking SAH within the Sylvian vallecula measured 42.4 HU. We could not confirm the pseudo-SAH with lumbar puncture or postmortem study, but this was not necessary for a certain diagnosis, following Yuzawa et al., because the measured CT levels barely overlapped. There was no intraventricular high-attenuation lesion in our case. Yuzawa et al. demonstrated the significant difference between pseudo-SAH and SAH CT values. They showed that the CT values of HDAs in the pseudo-SAH group were significantly lower than those in the SAH group (30–42 HU versus 41–67 HU). There was very little overlap in these values (4).

Our case suggests that SAH-like findings on CT are not a new complication due to bee stings, and that these are related to prolonged CPR. In conclusion, as in our case, the CT measure-

ment of HDAs of the Sylvian valliculae must be carried out in patients following prolonged CPR, regardless of the underlying cause, for the differentiation of SAH from pseudo-SAH. This is especially necessary in patients with diffuse low attenuation of the brain parenchyma with obliteration of the cisterns and cerebral sulci, along with narrowed ventricles evident on non-contrast brain CT.

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TRANSPARENCY DECLARATIONS

Competing interests: none to declare.

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CASE REPORT

Antrochoanal polyp of unusual size

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ABSTRACT

This paper presents antrochoanal polyp of unusually large size (8x5 cm), which we removed in a 34-year old patient by the antral portion by the Caldwell-Luc approach and the portion from the epipharynx through the oral cavity with skew pliers for the pharynx biopsy.

Key words: difficulty breathing through the nose, outgrowth in the nose, nasopharynx, oropharynx

INTRODUCTION

The antrochoanal polyp represents the benign lesion which starts to grow from the mucosis of the maxillary sinus and through the sinus cavity it enters into the nasal cavity, then through choans it moves through to nasopharynx. It was first described by professor Killian in 1906 (1). It appears in 3 to 6 % of cases of sinus nasal polyposis (1).

Etiology is not clearly defined, but it is considered that an infection is one of the causes, since the chronic infection is present in 25 % of patients (2). It is usually found in nonatopic patients (2). It most frequently appears in adults between the third and fifth decade of life. They are more frequent in male than in female population (2). Pathohistologically, they are identical to other inflammatory polyps, but they have the narrow pedicle (3). The histological polyp is wrapped by the respiratory epithelium (3). Due to the narrow pedicle and compromised vascularization, bleeding can be noticed, as well as the or-