Cerebral edema is a difficult condition for doctors to diagnose without proper testing. Your diagnosis will depend on your symptoms and the underlying cause.

Some common procedures doctors use to diagnose brain swelling include: physical exam to detect pain, discomfort, or abnormalities, CT scan to identify the location of the swelling, head MRI to identify the location of the swelling, blood tests to determine the cause of brain swelling.

Brain swelling can become a life-threatening condition. It should be treated immediately. Treatment options are meant to restore blood flow and oxygen to the brain while reducing the swelling.

It's also important to treat the underlying cause to prevent any further damage.

There are six common treatment options.

1. Medication.

Depending on the severity of your condition and the underlying cause, doctors may prescribe you medication to help reduce swelling and prevent blood clots.

2. Osmotherapy.

When your brain swells, it accumulates excess fluid. Osmotherapy is a technique meant to draw water out of the brain. This is done using osmotic agents such as mannitol, or high-salt saline. Osmotic therapy also helps improve blood circulation. This will help reduce swelling and ICP in the skull.

3. Hyperventilation.

Some doctors may perform a controlled hyperventilation to help lower your ICP. Hyperventilation causes you to exhale more than you inhale, lowering the amount of carbon dioxide in your bloodstream. Proper blood flow in your brain is dependent upon carbon dioxide. Controlling this process lowers the blood flow in your brain and reduces ICP.

4. Hypothermia.

Another treatment method includes inducing hypothermia. Lowering the body temperature decreases metabolism in the brain and can also reduce swelling.

Though there've been some success stories with this method, controlled hypothermia is still being researched.

5. Ventriculostomy.

This is a more invasive procedure that involves draining fluid from the brain. A doctor will make a small incision in the skull and insert a tube as a drain. This method will relieve ICP pressure.

6. Surgery.

In more severe cases of cerebral edema, you may need surgery to relieve ICP. This surgery could mean removing part of the skull or removing the source of the swelling, such as in the case of a tumor.

7. Prognosis.

Brain swelling is a serious condition that can cause long-term damage to your memory and ability to think. It may also be fatal if treated too late. If you begin to experience side effects after a fall, accident, or while fighting off an infection, visit a doctor immediately.

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TRANSIENT REVERSAL OF ANOXIC BRAIN INJURY-RELATED MINIMALLY CONSCIOUS STATE AFTER ZOLPIDEM ADMINISTRATION

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Introduction

Zolpidem is a unique non-benzodiazepine sedative hypnotic drug that selectively binds to omega-1-aminobutyric acid receptors in the brain. Although used for years in Israel and

abroad for insomnia, there have been periodic reports of unusual or remarkable neurologic effects in patients with various brain pathologies. Here, we report on a 50-year-old woman 18 months after severe anoxic brain injury in a minimally conscious state. Residual deficits included mutism, athetoid movements of the extremities, and complete dependence for all personal care. After the administration of 5 to 10 mg of zolpidem, within 45 minutes, the patient's condition improved markedly, including the cessation of athetoid movements, regained speaking ability, and ability to perform various tasks including self-feeding. These effects lasted 3 to 4 hours, after which the patient returned to her former state. This effect was repeatable on a daily basis. Existing evidence and possible mechanisms to explain zolpidem's effects in brain injury are described.

ACQUIRED BRAIN INJURY is a major cause of disability worldwide, from traumatic and non-traumatic causes .Common forms of acquired brain injury include ischemic and hemorrhagic stroke, traumatic brain injury, and anoxic brain injury. Anoxic injuries are considered to be particularly devastating, given the relative young age of victims, the breadth of the neurologic damage, and the relatively poor long-term prognosis of survivors. Anoxic brain injury causes widespread cellular injury and cellular death, causing severe neurologic deficits that may leave a profoundly disabled patient. Deficits may include impairment of consciousness, severe motor and sensory deficits, and cognitive impairment. Treatment options are very limited, with early spontaneous improvement probably being the most significant factor in recovery. Rehabilitation may be offered to selected patients in an attempt to minimize impairments, with variable results. There is no proven effective drug treatment for anoxic brain damage. However, given the profound socioeconomic and personal costs, patients, families, and treatment providers may highly value even minor changes that contribute positively to their quality of life .Various pharmacologic and other interventions have been used for the wide variety of neurocognitive deficits in acquired brain injury. These include neurostimulants, amantadine, anticonvulsants, and antidepressants. Generally, results are unpredictable, and, if improvement occurs, it is usually subtle. The overall contribution of these pharmacologic interventions to the ultimate functional outcome of these patients is unknown. Zolpidem is a non-benzodiazepine sedative in the imidazopiridine class and is chemically distinct from other sedatives. Although the benzodiazepines nonselectively bind to and stimulate 3 different — aminobutyric acid (GABA) receptors, zolpidem is a selective omega-1 GABA agonist. The GABA receptor complex is thought to be the mediator in these drugs' sedative, anxiolytic, and anticonvulsant properties. Zolpidem has been approved for use worldwide for insomnia and is considered preferable to the benzodiazepines because of its short half-life (2.4h) and low potential for drug dependence. Here, we present the first report in Israel of a noncomatose but minimally functional patient who responded to zolpidem with significant, albeit transient, functional improvements.

Aim

To study the treatment of comatose zolpidem according to literary sources.

Materials and methods

A theoretical analysis of literary sources and a synthesis of scientific literature for 2015–2018 was used.

Results

The patient is a 50-year-old married woman, mother of 3, with known coronary artery insufficiency and previous myocardial infarction. Eighteen months before presentation at our clinic, she suffered ventricular fibrillation on 2 occasions, one in her home and again during her subsequent hospitalization .Resuscitation was successful, but unfortunately a severe anoxic brain insult occurred. For about 3 weeks, she was comatose and, after beginning to respond, was transferred to a rehabilitation facility. There, she continued to regain consciousness. Be-

cause of persistent severe deficits of attention, lack of communication, and lack of motor control, rehabilitation was stopped, and she was transferred to a long-term care facility. A CT scan of the brain revealed mild ventricular dilatation but no other acute abnormalities. Electroencephalographic testing revealed no seizure-like electric activity. Several months after admission to the nursing facility, the patient's family elected to care for her at home, and she was discharged. At home, she continued to receive rehabilitative treatments in an attempt to improve her motor and cognitive status. However, because of her lack of ability to cooperate, these efforts were ineffective. She remained nonverbal, incontinent, and totally dependent for all personal care. Of note was that the family observed that occasionally but unpredictably the patient would become more alert and begin to speak to her family. These fluctuations occurred about once every month or 2. Because of these episodes, the family remained hopeful that recovery potential remained and consulted us. On initial examination, the patient was wheelchair-bound, awake, and distractible, and she made eye contact. There were persistent athetoid movements of the trunk, upper extremities, and head. The patient postured her left arm in forward flexion, hand suspended in the air, making mostly non purposeful movements. The right arm was held adducted at her side. She was nonverbal, except for an occasional Hebrew «yes» mostly out of context. She was only occasionally able to raise her left hand higher on command. She responded to visual and sound stimulation via eye contact only. In summary, the patient had findings consistent with a minimally conscious state (MCS) as defined by Giacino. Because of the previously reported cases of response to zolpidem, zolpidem was discussed with the patient's family, including her husband who is her legal guardian. After consultation with the institutional pharmacist and after the family consented, 10mg of zolpidem was prescribed to be administered during the day. The following day, the family reported dramatic improvement in the patient's functional status within 30 minutes of administration. This effect lasted for about 3 hours and was repeated for several days in a row after giving the drug. The effect could be produced by a 5-mg dose as well but not consistently. The following week that patient returned to the clinic and was reexamined before and after being given zolpidem. Before drug administration, the patient's condition was unchanged compared with the examination 1 week prior, being essentially MCS. Ten milligrams of zolpidem was administered in the office, and the patient was observed. Thirty minutes later, she began to become more alert with more verbalizations and a decrease in athetoid movements. At 45 minutes, her condition dramatically improved, emerging from MCS and revealing the following findings. She was alert, oriented to self and place only. All athetoid movements had stopped. She was able to answer simple questions in short sentences, including identifying her husband and daughter present in the room and other relatives not present. She was able to read Hebrew and English sentences, give short written and verbal responses to the written questions, and perform simple calculations. She laughed appropriately and expressed affection with her family members. She had profound anterograde, short-term amnesia, being unable to remember any events relating to either her MCS or prior periods of alertness. She was able to recall basic information about the family's remote past. A motor examination revealed full voluntary movements of all 4 extremities, with a minimal residual slow tremor in the right hand. She was able to grasp objects and feed herself with a spoon. She was able to stand and take a few steps with moderate assistance from 2 people. The cognitive level on the Rancho Los Amigos score increased from III (localized response) to VI (confused appropriate). The patient left the clinic 2 hours after drug administration in this alert state, and, according to her family, she returned to her baseline condition 4 hours later.

This case corroborates previous case reports of dramatic zolpidem-related improvement in patients with chronic acquired brain injury, although this is the first case reporting such effects in a patient with MCS. The mechanism of this unexpected effect is unknown, but Clauss

and Nel proposed a mechanism based on brain single-photon emission computed tomography studies. These scans, performed on «zolpidem responders», showed that after zolpidem administration, there was a marked increase in blood flow to areas of the brain adjacent to or distant from the damaged tissues, such as in the contralesional (ipsilateral) cerebral hemisphere or cerebellum. These areas are believed to be inhibited by the site of injury by a GABA-mediated mechanism, and this inhibition is modified by zolpidem. Further proof of this is offered by the fact that the zolpidem effect can be blocked by flumazenil, which blocks omega-1 receptors. The idea that functional neurologic deficits are not caused solely by brain tissue death is not new and is the basis of newer rehabilitation techniques, such as constraint-induced movement therapy. Thus, this explanation for zolpidem's action is plausible and consistent with current concepts on brain plasticity, which involves cortical reorganization and activation of previous unused or underutilized pathways. Ipsilateral motor cortex areas are believed to play a role in neurologic recovery, particularly early on, and may show an inhibitory effect on the ipsilesional (contralateral) cortex via interhemispheric inhibition.This inhibition decreases with neurologic recovery. In essence, zolpidem appears to temporarily "short circuit" the recovery and learning process, which is usually required for plasticity-related neurologic recovery. That zolpidem induces sleep in normal persons but causes arousal in brain-injured patients is a remarkable paradox. Furthermore, it is not clear why the selective action of zolpidem on the omega-1 receptor acts favorably on this inhibition in brain injury, whereas classic benzodiazepines do not. Short-interval intracortical inhibition is a GABA-mediated motor cortex inhibition that is increased by benzodiazepines but not by zolpidem. Thus, the inhibition exerted by the omega-1 units, in the absence of stimulation of the other 2 subtypes, may be related to the unexpected arousal. Some patients do not respond to zolpidem; presumably, in these cases, the neurologic deficits are caused mainly by severely damaged or dead tissue rather than from inhibitory effects.

Conclusion

Given the poor prognosis of patients with chronic acquired brain injury with disorders of consciousness, zolpidem may offer great hope for patients and their families. Clearly, more work needs to be done, both to delineate the mechanisms by which this drug works and to perform controlled trials to prove effectiveness, both short- and long-term.

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INTRACEREBRAL HEMORRHAGE NON-TRAUMATIC CAUSES AND MANAGEMENT

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Introduction

The American Heart Association/American Stroke Association has defined the term intracerebral hemorrhage (ICH) as «A focal collection of blood within the brain parenchyma or ventricular system that is caused by traumatic/other metabolic causes or non-traumatic» and also causes a life-threatening type of stroke called hemorrhagic stroke (HS).

Aim

To study the clinical picture and the manifestations of intracerebral hemorrhage non-traumatic causes according to literary sources.