

Refractory Epilepsy in Adults after CVI

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Abstract

Annual incidence of status epilepticus puts it in the second place among neurological disorders (acute stroke, being the first), with high mortality risk. Occurrences of RSE have been mostly associated with acute, severe and potentially fatal underlying etiologies such as, encephalitis, massive stroke; or rapidly progressive brain tumors. This may be severe impairment of consciousness. In non-convulsive status epilepticus, brain is either continuously seizing or seizing so frequently that the patient never has a chance to recover from the period of extreme confusion that normally follows a seizure. This recovery period is called the post-ictal state. Often, people in non-convulsive status epilepticus look like many other patients, who are unresponsive due to an encephalopathy. Firstly, we have to determine whether a patient really is seizing. Part of the reason is that NCSE is a relatively newly described occurrence, and by its nature it isn't dramatically obvious as a convulsive status epilepticus. Treatment of NCSE is most commonly based either on benzodiazepine or an anesthetic agent.

Keywords: Status Epilepticus; Cerebrovascular Insult; Refractory Status Epilepticus

The annual incidence rate of status epilepticus (SE) comes in second among neurological disorders (the first one being the acute cerebrovascular insult – CVI). SE has a high risk of mortality. Refractory status epilepticus (RSA) can occur together with an acute massive CVI (an infarction or hemorrhage), encephalitis or fast-growing brain tumors. This paper examines the non-convulsive status epilepticus, in which the status of the brain does not change and it does not have any prospects of recovery. The period of potential recovery occurs in the postictal phase. During the seizure itself, patients often seem as any other intensive care patients – they are in an altered state of consciousness, placed on a mechanical ventilator and suffering from encephalopathy of various etiologies. The seizure first needs to be determined and identified. The non-convulsive status epilepticus is not as dramatic as the convulsive one. The treatment includes benzodiazepines and anesthetics, such as midazolam, pentobarbital, propofol and ketamine.

Case Report

A 73-year old female was admitted to the intensive-care unit due to a sudden left-sided weakness and a state of altered consciousness, accompanied by generalized tonic-clonic seizures (GTC). The findings of diagnostic neuroradiology verified the occurrence of acute ischemia involving the right temporo-occipital (TO) region and the right cerebellum, frontal lacunar ischemia, periventricularly on both sides, in the corona radiata and centrum semiovale, together with the signs of an onset of ischemic leukoencephalopathy. The patient was carefully monitored by a neurologist, an internist and an anesthesiologist and her status was checked by performing electrocardiograms, CT scans and radiographs. Patient's vital signs were continuously checked and her biohumoral status was corrected in accordance with the results of laboratory tests. The patient's state suddenly deteriorated and she experienced paroxysmal tachypneic episodes with abdominal breathing. However, there was no change in the patient's state of consciousness and no progression of neurological deficits. In addition, no severe changes were detected in electrocardiograms or biohumoral status. The results of cardiac enzyme tests also remained

unchanged. An electroencephalogram was done and the ictal EEG recorded irritated waves above the frontal and up to temporal regions of the brain during the attack of respiratory vegetative seizures. Upon intravenous administration of midazolam, the vegetative-respiratory seizures stopped and the overall activity was normalized. The seizures repeated twice or three times a day. The last seizure led to the loss of consciousness, coma, hypotension, overall hypotonia and hyporeflexia, bradycardia, ventricular extrasystole (VES), undetected blood pressure and a fatal outcome.

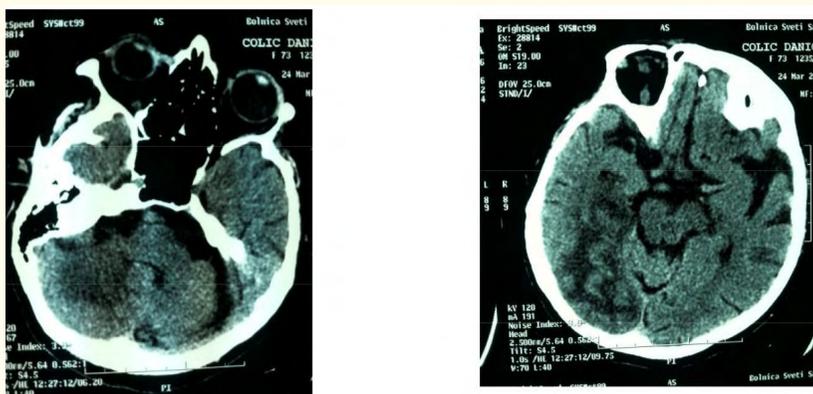


Figure 1.

Discussion

Vegetative autonomic symptoms occur often during epileptic seizures, either as a dominant manifestation of the seizure itself or as phenomena accompanying the complex partial or generalized seizures. The understanding of vegetative seizures accounts for pathophysiological mechanisms of their serious complications, such as sudden unexpected death in epilepsy (SUDEP), ictal cardiac arrhythmias and neurogenic pulmonary edema. Vegetative symptoms and signs are caused by a dysfunction or activation of the central autonomic network (CAN), which consists of the insula and medial prefrontal cortex, amygdala, stria terminalis, preoptic area and hypothalamus, periaqueductal gray matter in the mesencephalon, the pontine parabrachial area, nucleus tractus solitarius and intermediate reticular zone in the medulla oblongata.

The insula and the medial prefrontal cortex maintain the highest level of autonomic control. The insula is regarded the primary visceral sensory region, which receives information from the gustatory pathways, gastric mechanoreceptors, arterial chemoreceptors and baroreceptors. Electrical stimulation of the insula affects cardiac frequency, blood pressure, respiration, piloerection, as well as the pupillary, gastrointestinal, salivary and adrenal responses. The stimulation of the right insular cortex results in tachycardia and an increase in blood pressure, whereas the stimulation of the left insular cortex causes blood pressure to drop and bradycardia. Both the amygdala and stria terminalis are responsible for the autonomic and motor responses to emotions. The preoptic area and the hypothalamus integrate autonomic, endocrine and behavioral responses which are essential for homeostasis and reproduction. The periaqueductal region of the mesencephalon, the parabrachial area of the pons, the nucleus tractus solitarius and the ventrolateral medulla oblongata contain a network made up of respiratory, cardiovagal and vasomotor neurons.

Thus far, epileptic seizures caused by the activation of the autonomic nervous system which include cardiovascular, respiratory, gastrointestinal, dermal, pupillary, genitourinary and sexual ictal symptoms have been reported in relevant literature.

There is a wide range of respiratory manifestations which take place during epileptic seizures. In most cases, hyperventilation occurs in the course of simple and complex partial seizures along with mesial temporal lobe epilepsy (MTLE). Postictal central apnea or hypoventilation has a central role in the pathogenesis of SUDEP. Central apnea is the key event which induces postictal bradycardia and cardiac arrest due to changes in cardio-respiratory reflex. The neurogenic pulmonary edema is a potentially fatal manifestation of the epileptic seizure. Its pathophysiological mechanism involves a rise in the pulmonary vascular pressure due to the effect of the sympathetic nervous system on pulmonary vasoconstriction or increased pressure in the left atrium. It has not yet been defined with certainty which regions in the brain are responsible for respiratory manifestations of epileptic seizures. The respiratory arrest involves such regions as the temporal pole, the insula, hippocampus, amygdala, frontal cingulate gyrus and the uncus. Temporal lobe epilepsy, in particular MTLE, has a consistent interictal EEG in 95% of the cases of MTLE and has a spike and sharp waves with maximum electronegativity above the frontal temporal regions. The postictal pattern shows a slow, irregular activity, which is desynchronized or repetitive spikes may be present. The most characteristic element of the ictal EEG is an occurrence of 5 Hz or fast rhythmic activity above the temporal regions within the first 30 to 40 seconds of the occurrence of the first clinical symptoms and signs.

The epileptogenic network represents a functionally and anatomically connected and bilaterally linked population of cortical and subcortical brain structures and regions in which an activity of one of its parts has an effect on the activity of all the other elements of the network. The network is a functional entity responsible for clinical and electrophysiological phenomena which are characteristic of epilepsy. Epileptic seizures may propagate in a variably extensive way and may affect any of the regions that are anatomically connected to the epileptogenic network. Seizure propagation may affect a number of regions, depending on which structures make up the epileptogenic network. Each structural disruption in the network, a modification of network activities in any of its parts resulting from electrical, biochemical or metabolic influences changes the expression of the seizure and its occurrence – it represents a basis for remission [1-33].



Figure 2.

Conclusion

Pharmacoresistant focal epilepsy (MTLE) or the limbic network comprises the hippocampus, the amygdala, the entorhinal cortex, the lateral, temporal neocortex – medial thalamus and the inferior frontal lobe.

The predomination of vegetative symptoms and signs may lead to a wrong diagnosis of a non-epileptic seizure or event. In case of an unquestionable diagnosis, the administration of antiepileptic drugs may lead to further deterioration of vegetative functions. Vegetative

manifestations during epileptic seizures must be analyzed, well interpreted and their relevance must be assessed.

An analysis of vegetative signs in the context of an increased risk of sudden death in patients with pharmaco-resistant epilepsy requires additional technological measures, which will be the subject of future research and studies.

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