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Neuropsychological Sequelae of Out-Of Hospital Cardiac Arrest Case Study

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Abstract
This paper presents a case of a 63-year old patient after OHCA (out-of--hospital cardiac arrest), who was admitted for rehabilitation in the neurology department. About half of out-of-hospital cardiac arrest survivors experience secondary anoxic brain damage. Neurological outcome can be influenced by rehabilitative treatment approaches, but the nature and severity of persistent disabilities remain unclear. The aim of the study was to explore neuropsychological symptoms, general functioning and life situation of this lady. Function was investigated by clinical rating scales, neuropsychological standard tests, and clinical psychological inventories. Family members were asked about the quality of life before and after OHCA. We presented predictors of outcome and frequency of neuropsychological sequelae.

Key words
cardiac arrest, neuropsychology, cognitive and behavior impairments, neurorehabilitation

Introduction

Out-of-hospital cardiac arrest (OHCA) is an important cause of death in developed countries, but the advances in emergency healthcare have increased the percentage of cardiac arrest survivors [18]. In Europe the incidence of sudden cardiac arrest in 1980-2004 was estimated to be 38 per 100,00 person/year with a survival rate of 10.7% [1,7]. In Poland, the reported incidence rate of OHCA is 55.6/100,00 inhabitants/year [25]. About 80% of cardiac arrests occur at home, for which the rate of death is at least 90% [1,25]. More than a half of the survivors have permanent brain damage of varying degrees [31]. The use of an implantable cardioverter defibrillator in selected patients and the availability of public-access defibrillators have improved survival with an acceptable quality of life [1,7,39]. In-hospital arrests have slightly better outcomes than those that occur outside the hospital, with restoration of circulation in 44% of patients and survival to discharge in 17% of patients [2]. Two randomized, controlled trials showed a positive effect of hypothermia on mortality and morbidity after cardiac arrest [39].

The human brain has a constant requirement for oxygen to meet energy demands. When oxygen delivery is disrupted, a series of cerebral homeostatic and vascular mechanisms is activated, directed toward maintaining oxygen supply [1,5]. This autoregulation maintains cerebral blood flow against abrupt blood pressure change within a certain range (about 50 – 150 mm Hg) and maintains delivery and utilization of oxygen despite fluctuations in the supply [5,23]. Therefore, even though the brain does not store oxygen, oxygen deprivation must be sustained in order to render cerebral autoregulatory mechanisms effectual and for the brain not to become injured. A period longer than 4 to 8 min is said usually to produce cerebral infarction and disseminated cell death [5,18] The mechanisms which cause anoxic damage are both complex and diverse. They comprise of a cascade of time-dependent alterations in neuronal function, metabolism and morphology [5,11,13]. The first concerns the distribution and disposition of cerebral blood vessels so that those portions of the supply territory that lie in the fringes of supply of the major cerebral arteries are the first to experience reductions in blood flow [5,13]. Second, the specific metabolic and biochemical properties of the involved structures are such that the higher the metabolic demand of a given area the more likely it is to be affected by oxygen deprivation [5,11,31].

Predictors of Outcome

Most studies that have assessed predictors of outcome in patients with anoxic ischemic encephalopathy have the reliable prediction of an outcome no better than a vegetative state or severe disability with total dependency at 3 to 6 months after arrest as their prime objective. They are shown below [12,13,39].

1. The absence of a pupillary reaction to light suggests a poor prognosis but has unclear specificity when assessed early after a cardiac arrest. All patients whose pupillary reactions were absent at day 3 after cardiac arrest had poor outcomes.
2. The motor response to noxious stimuli also provides useful prognostic information. Prospective studies showed that a motor response to noxious stimuli that was no better than extensor posturing (i.e., a decerebrate response or no response) at 72 hours was associated with a poor outcome.
3. The corneal reflex is tested by touching the cornea with a gauze or cotton swab and looking for contraction of the orbicularis oculi on either side. In two prospective studies, the absence of a corneal reflex at 72 hours was associated with a poor outcome.
4. The absence of eye movements in response to irrigation of the ear canals with ice water at 72 hours is another indicator of poor outcome. However, the caloric response can be blunted by many sedative drugs. To avoid the confounding effects of cumulative or large doses of sedative, anesthetic, and analgesic drugs, it is best to put off testing until there is ample evidence that they have been cleared from the circulation.
5. Myoclonic status epilepticus (bilaterally synchronous twitches of limb, trunk, or facial muscles) is likewise a marker of a poor outcome.
6. Electrophysiological Signs and the measurement of somatosensory evoked potentials (SSEPs), especially the response from the primary somatosensory cortex (assessed 20 msec after electrical stimulation of the median nerve at the wrist), has emerged as the most accurate predictor of a poor outcome in patients with anoxic ischemic encephalopathy.
7. Neuroimaging computed tomographic (CT) images are usually normal immediately after a cardiac arrest, but by day 3 they often

show brain swelling and inversion of the gray-white densities (with the use of quantitative measures) in patients with a poor outcome. Further study is needed to assess the clinical use of these findings in establishing a prognosis.

Many patients who do not have the abovementioned unfavorable clinical features still have poor outcomes. The brain stem is more resistant to anoxic ischemic damage than the cerebral cortex; thus, compromise of brain-stem reflexes suggests that the cortex must be severely damaged. However, preserved brain-stem reflexes do not imply intact cortical function [5,39].

Neuropsychological Sequelae of Anoxic Episodes

OHCA survivors show no fixed pattern of cognitive and neurological consequences of brain injury [25]. Most neurologists encounter these patients during the early phases of coma and confusional states [18].The symptoms depend on the degree of brain damage, which is proportional to the duration of OHCA [18,27]. The most common cognitive disorders range from minor disability to no conscious cognitive activity due to severe arousal disturbances, limited awareness and attention, and higher-level cognitive dysfunctions. The latter commonly include memory and executive disturbances [14,18] and personality abnormalities [9, 12, 25].

An amnesic syndrome was reported in 54% cases, while memory disturbance without accompanying cognitive change was identified in 19% of them [5]. The amnesia was said to be characterized by intact registration, severely depressed free recall, less depressed recognition memory, and greater than normal susceptibility to interference. The precise nature of the memory disturbance in anoxic patients most likely depends on which brain structures are affected and to what extent. Mackenzie Ross and Hodges [5] found well preserved knowledge of famous people in the context of profound impairment of personally relevant autobiographical memory, which they suggested may be attributable to the multifocal neocortical damage sustained by this patient, who presented generalized intellectual impairment and marked deficits in frontal executive function in addition to the memory deficit [5]. An association between amnesic disorders and the structures of the mesial temporal region, in particular the hippocampus, has long been established [40]. However, the phenomenon of amnesia has not been associated exclusively with the hippo-

campus, and post-anoxic amnesia has not invariably been associated with hippocampal changes [40]. Experimental evidence has demonstrated that lesions to the anterior thalamic nuclei, the fornix and the hippocampus can all produce comparable memory deficits [5,40]. It has also been suggested that there may be a characteristic memory deficit associated with lesions of the dorsolateral prefrontal cortex and caudate nucleus [5]. Unlike amnesic syndromes associated with lesions of the hippocampus or thalamus, frontal amnesic deficits are characterized by poor recall with relative preservation of recognition abilities. Cummings [5] has argued that the reason for this is that the frontal subcortical circuits mediate memory activation and search functions rather than storage; lesions produce deficits of information retrieval with relatively preserved recognition [5]. The precise nature of the memory disturbance in anoxic patients most likely depends on which brain structures are affected and to what extent.

Changes in personality and behavior, or in some aspect of executive function, were noted in 46.2% of individual cases under review [5]. The most frequently reported change in personality was either emotional lability and impulsivity, or alternatively a lack of emotional expression. Both of these were often associated with lack of concern [5]. Reich et al. [5] reported 6 survivors of cardiac arrest who suffered from only mild cerebral impairment, manifested primarily in personality changes and behavioral symptoms. The patients were distractible, irritable, and disinhibited, with disturbances of impulse control and loss of judgment. On mental status examination they showed bland or labile affect, rigid thought patterns, and major disturbances of insight, empathy, and self-awareness. In each case the symptoms were initially mistaken for depression. [5]. Lezak described a reduced capacity for planning, initiating and carrying out activities in association with anoxia, changes in executive function have attracted little attention in this etiology. Reduced executive function in this context refers to distractibility and difficulty with multiple tracking, impaired planning, and poor abstraction [17]. Behavioral changes associated with lesions of the prefrontal region have been amply demonstrated [5]. They include changes in such capacities as the orderly planning and sequencing of complex behaviors, the ability to attend to several components simultaneously or to alter flexibly the focus of concentration, the capacity to grasp the context and gist of a complex

situation, the ability to follow multistep instructions, the inhibition of immediate but inappropriate response tendencies, and the ability to sustain behavioral output without perseveration [5]. Similar behavioral changes including disorders of executive function, personality changes, and mood disturbances are found in patients with lesions in other brain regions, most notably the basal ganglia [5]. The most commonly reported changes associated with basal ganglia lesions include loss of motivation and initiative, loss of cognitive flexibility, reduction in both mental and behavioral activities, blunting of affect, and reduced or altered verbal output [5]. This phenomenon has been explained on the basis of the connectivity between separate regions of the basal ganglia, each of which projects to a different part of the frontal lobes [5,14].

Visuospatial or, less frequently, visual recognition problems were noted in 31% individual cases reviewed [5]. Complex visuospatial deficits are usually associated with lesions of the lateral occipitoparietal cortex bilaterally, an area that is thought to be particularly vulnerable in anoxic encephalopathy [5]. In most reported cases this has resulted in less profound perceptual disturbances than either a visual object agnosia or a Balint's syndrome [5].

Language has generally been considered to be preserved in anoxia [5,40]. Although language has not been examined extensively, in a number of studies it has been reported to be normal. For instance, Volpe et al. [5] reported normal performance on the controlled word association test, preserved capacity to paraphrase proverbs and perfect performance on the Token Test in their participants. Zola-Morgan et al. [40] tested case R.B. with Boston Naming and a screening aphasia battery and found no abnormalities other than an amnesic syndrome.

Case presentation

13. 06. 2016r. A 63-year-old female (retired engineer with a university degree) fell in front of the house after leaving the car. She suffered from hypertension, hypercholesterolemia and panhisterectomy in April 2016. Passer-by (former international mission soldier) stated lack of pulse and started effective cardiopulmonary resuscitation. Emergency ambulance was called. Resuscitation lasted 10 min. Resuscitation was continued in the emergency department after sudden cardiac arrest in ventricular fibrillation. The intubated patient with assisted respiration was trans-

ferred to a cardiologist. She underwent coronary angiography and aspiration thrombectomy. The head neurotransmission (CT) test revealed discrete subclavian ischemic lesions in both hemispheres of the brain. On the third day, the patient was extubated. In the following days, a gradual improvement of general condition was observed. Five days after cardiac arrest, the neuropsychological deficits and lack of motor deficits were identified in the neurological consultation. An amnestic syndrome was diagnosed in the psychiatric consultation. Three weeks after OHCA, the patient was discharged home. A month later, she was admitted to a neurological rehabilitation unit. On reception in preserved verbal-logical contact. Properly oriented in time and place. During the interview, she complained about retrograde amnesia (3 months before OHCA) and anterograde amnesia (1.5 months after OHCA). Pharmacotherapy has been coordinated by a cardiologist. She was included in neurorehabilitation in this particular neuropsychology training and occupational therapy.

Our interest in the presented case is due to the dynamics of the observed neurobehavioral changes due to unfavorable initial prognosis after OHCA and improved outcomes. The cardiological and neurological condition was initially severe. The patient was effectively diluted three days after OHCA. Previous attempts failed. Cardiological treatment was prolonged and stabilized after 3 weeks and ended in therapeutic success.

Methods

Neuropsychological assessment was based on cognitive screening in three measurements [35]. Screening was performed using Addenbrookes Cognitive Examination-Revised (ACE-III), which covers orientation, attention, memory, verbal fluency, language and visuospatial ability [17,32]. The first measurement was done one month after the event, the second one was extended with other neuropsychological tests after three months, and the third screening after nine months after the OHCA. The detection and quantification of acquired neuropsychological deficits rests upon the comparison of an individual's current cognitive functioning with an estimate of their premorbid ability [19,33,37]. We use Wechsler Adult Intelligence Scale IQ (WAIS-R PL). More comprehensive cognitive examinations included [32]:

- attention: Color Trails Test (CTT),
- memory and learning: California Verbal Learning Test (CVLT), Reys

Complex Figure Test (ROCF);
- executive functions: Tower of London(TOL), Verbal Fluency,
- language: spontaneous speech and Token Test (TT),
- visuospatial skills and motor performance: Clock Drawing Test (CDT), Performance IQ (WAIS – R PL),
- personality: observation, behavior screen ECAS, Background Questionnaire – Adult [32].

We collected information on demographic factors and lifestyle characteristics.

Result

The results of the neuropsychological testing are summarized in table 1, which include clinical analysis of test results, evaluation of the severity level (mild, moderate, severe, or very severe) of the cognitive disorders.

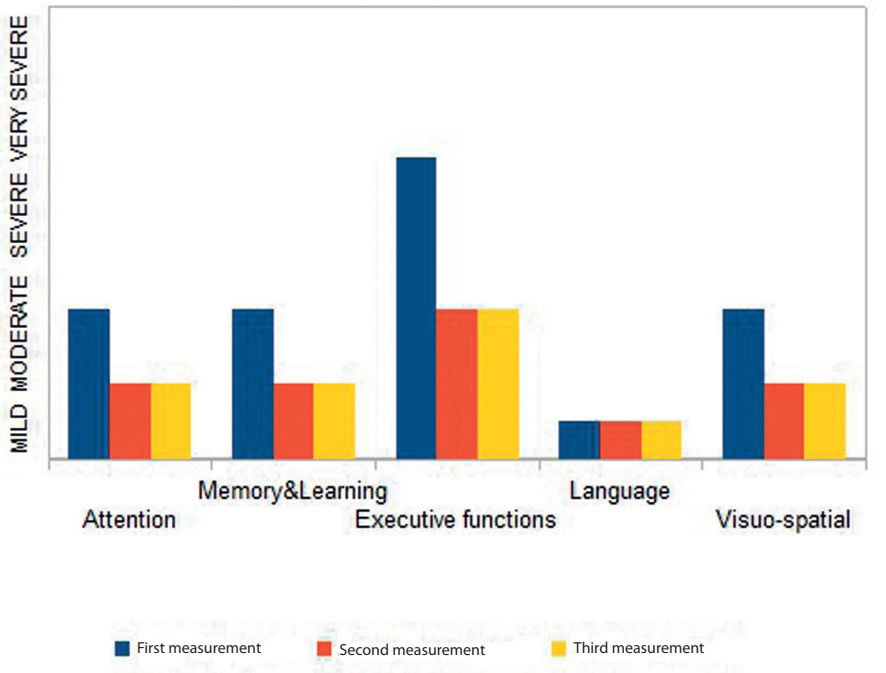

Table 1. Presentation of results

The results of the subsequent measurements revealed the weakest performance in the first study. Depth of cognitive disorders was at me-

dium and deep levels (executive functions). In the subsequent studies the results were better. There were no differences between the second and third measurements (mild cognitive impairment). Language assessment didn't show aphasia (despite mild naming difficulties) and linguistic impairments. Based on the results of the WAIS-R (PL) test, a high level of premorbid ability is estimated. Full Scale (FSIQ WAIS-R PL) is a high-intelligence intellectualized test. The VERBAL VIQ scores are above average, while Performance PIQs indicate a high level of performance. The highest test scores were obtained in the following sub-subjects: Similarities, Picture Completion, Digit Symbol. They indicate a high premorbid ability. Attention should be paid to the answer given in the Comprehension (question about the fire in the cinema). Giving responses reveals a tendency to impulsive reactions. WAIS-R PL factor analysis reveals a reduction in processing speed. It is clear from the qualitative interpretation of the task that the patient is able to solve the visuospatial tasks (most sensitive to brain damage) but due to the psychomotor slowdown most often exceeded the set time limit.

Based on clinical observation of our patient we identified some type of behavioral and/or emotional dysfunction. These are impulsiveness, self--awareness disorders and childlike behavior. We didn't find symptoms of depression and generalized anxiety disorder. Family was reporting changes in behavior and personality. We collected information in interviews with her husband and daughter. They have observed the repetitive, irresistible urge to perform an action, the experience of loss of voluntary control over this intense urge and the tendency to perform repetitive acts in a habitual or stereotyped manner, for example buying many unnecessary things. She was characterized with the tendency to act prematurely without foresight, resulting in behavioral disinhibition.

The neuropsychological assessment of the person with OHCA performed in the hospital showed impairment recovery in a wide spectrum of cognitive and behavioral functions other than memory. Memory disturbance is the most frequently reported neuropsychological impairment after a cardiac anoxic event [3,4,8,15,16,19]. Our measured cognitive impairments were classified as minor cognitive impairments. This result shows importance of educational level and including aspects of high premorbid ability [19]. In this case study our patient didn't show memory disturbance, however she showed executive and personality

dysfunction. Our findings confirm processing speed impairment [1,22]. Executive and personality dysfunction and frontal lobe affection are also frequently reported and our results support this finding [5, 24]. This behavior is sometimes difficult to distinguish from compulsivity due to their phenomenological similarities [24]. Frontal syndrome can often occur following infarction [6,11,18]. This syndrome is characterized by disinhibition and actions without foresight of the consequences, such as increased risk-taking behavior [24,29] and sometimes compulsions with the inability to inhibit repetitive, irresistible urges to perform action [24]. Frontal syndrome is associated with structural lesions in the frontal brain areas [24] or with connectivity between separate regions of the basal ganglia, each of which projects to a different part of the frontal lobes [5].

Discussion

The presented study provides three major points. First, fast effective cardio-pulmonary resuscitation minimizes the extent of brain damage. Secondly, the dynamic improvement of the neurological state in the first days after resuscitation is expected to return to full recovery. Thirdly, high premorbid ability resources affect the quality of life in the future [39]. Our data confirm the results of a previous study [25,34].

Mild cognitive deficits are common in OHCA survivors with a high functional premorbid status [34,36]. OHCA survivors are heterogeneous in terms of clinical status [6,26]. Earlier studies demonstrated that the functional consequences of brain injury range from complete recovery to mild cognitive deficits to severe cognitive-behavioural and motor impairments, minimally conscious state (MCS) or even permanent vegetative state (VS) [5,10,20]. Although massive and variable impairments usually occur early after OHCA [22,39], the most frequent in the post-acute phases are memory and executive abnormalities, sometimes coupled with subtle motor deficits [6,40]. In contrast, aphasia and perceptual impairments are rare [5,13,23]. The dynamics of cognitive and neurological symptoms in successive phases of post-cardiac arrest syndrome remain poorly understood [25]. Despite studies of early [18] or chronic stage impairments after OHCA [22,30], little is known about the natural course of recovery, patterns of chronic impairment, rehabilitation needs, or the effectiveness of therapies [25,26].

It has been postulated that rehabilitation should be modelled on principles established for other sudden brain injuries [21,26]. Accordingly, rehabilitation should be comprehensive, conducted in specialized units and started early post-injury when the patient is stable [1,18]. For patients with consciousness disorders, environmental management, passive physiotherapy, and sensory stimulation are provided [26]. For less severely impaired patients, progressive training addresses impaired cognitive and sensorimotor functions, and compensatory techniques, occupational therapy, environmental modifications and behavioral adaptation are recommended [1,21,26]. Pharmacotherapy may be useful, but the data is limited [25].

In our patient, behavioral observation and neuropsychological tests results suggested that post-anoxic cognitive decline caused the reduction of awareness. This fact must not be neglected in studies on emotional situation and quality of life in brain-damaged patients. Thus, with regard to the permanent cognitive limitations caused by cerebral anoxia, families and society have to accept that despite optimal acute and rehabilitative treatment the overall goal of rehabilitation cannot be complete recovery, but only optimal adaptation and compensation hopefully leading to independence in ADLs and social competence.

Our patient is in a group with mild cognitive impairment. But this condition is not satisfactory for the family, which expects further improvement and return of premorbid function. Family help was offered to improve internal communication and to better understand mutual problems.

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