The aim of this paper was to show the cognitive and emotional deficits noted in a patient following a stroke within the rehabilitation process. A 61-year-old female patient after an ischemic stroke with left-sided paresis and neuropsychological disorders. Rehabilitation was carried out in accordance with the ICD-9 procedures over 3 weeks with additional sessions with a neuropsychologist, occupational therapist and speech therapist. The subjective and objective condition of the patient partially improved and the program was recommended to be continued at home. Stroke causes cognitive disorders, including those affecting higher cortical functions. Their etiopathogenesis and the resulting problems hindering the rehabilitation program are herein presented.

Cognitive and emotional disorders restricting patient functioning and the rehabilitation processes themselves were observed. Neuropsychological therapy was an essential part of the treatment, which was helpful in the improvement of the patient’s quality of life.

Key words: neuropsychological disorders, right side paresis, dysphagia, dysarthria
INTRODUCTION

Strokes are one of the most common forms of damage to the central nervous system. Each year, approx. 60,000 people suffer a stroke in Poland. A stroke constitutes a clinical syndrome characterised by the sudden occurrence of a focal neurological deficit caused by impaired cerebral circulation (Danielsson et al., 2015; Hagino et al., 2015; Katz-Leurer Hagino et al., 2015; Chun Hagino et al., 2015; Cheng B et al., 2015; Huan et al., 2015). The patient may or may not lose consciousness. Strokes may result from a cerebral haemorrhage or a thrombus or embolism in the cerebral arteries. Strokes are divided into the haemorrhagic and the ischaemic, which consist in cerebral infarction (softening). Ischaemic strokes are divided into thrombotic and embolic strokes. An estimated 85% of strokes are ischaemic and approx. 15% are haemorrhagic (Dean & Kautz Huan 2015; Kang & Lim 2015; Biedal et al., 2013). Depending on which blood vessel is blocked, the brain area supplied by this artery becomes inactive, resulting in characteristic neurological symptoms associated with this area (Katz-Leurer et al., 2015; Chun et al., 2015; Cheng et al., 2015; Huan et al., 2015). Usually, a stroke affects the internal carotid artery, whose occlusion (stenosis) results in hemiparesis, hemisensory impairment and hemianopsia; in more severe cases, the patient may suffer from consciousness disorders. The occlusion (stenosis) of the anterior cerebral artery causes paresis of the lower limb contralateral to the lesion. The occlusion (stenosis) of the posterior inferior cerebellar artery (lateral medullary syndrome) results in acute symptoms such as dizziness, nausea, vomiting and dysphagia. Circulatory impairment in the area supplied by the basilar artery causes oculomotor deficits, difficulty in swallowing, dysarthria and facial paresis. Hemiparesis, sensory disturbances and cranial nerve paresis also occur. Patients with circulatory impairment in the area supplied by the basilar artery may display cerebellar symptoms (so-called cerebellar syndrome).

Strokes, both ischaemic and haemorrhagic, are multifactorial in character. The main risk factors for a stroke are hypertension, heart diseases, diabetes, dyslipidaemias, increased levels of fibrinogen and haematocrit, hyperthyroidism, long-term nicotine dependence, age, vascular dementia, long-term alcoholism and genetic factors.

The aim of this paper was to show the cognitive and emotional deficits in a patient after a stroke within the rehabilitation process.

CASE STUDY

A 61-year-old female patient was admitted to the Department of Rehabilitation to undergo rehabilitation following an ischemic stroke that had resulted in the occlusion (stenosis) of the posterior inferior cerebellar artery and circulatory impairment in the area supplied by the basilar artery. The patient experienced left-sided hemiparesis, dysphagia and dysarthria. Her medical history showed vascular dementia had been present for several years, nicotine dependence, hy-
pertension (which had not been regularly treated), diabetes mellitus type 2 and subclinical hyperthyroidism. Her respiratory and circulatory performance was normal. A neurological examination showed the patient to be verbally responsive; orientation in space and time was disturbed, her mood was decreased, there was psychomotor impairment but with no meningeal symptoms. The patient had central left-sided facial paresis, left-sided pyramidal syndrome, paresis of the left limbs (left upper limb MRC 3/5, left lower limb MRC 3+/5), Babinski sign on the left and superficial sensibility without dysmetria. On admission, the patient scored 9/20 on the functional Barthel Activities of Daily Living (ADL) Index. During the hospital stay, the patient underwent rehabilitation according to the ICD-9 procedures and sessions with a psychologist, occupational therapist and speech therapist; sessions adjusted to the patient’s abilities (Kuśnicki 2011; Blaszkowska et al., 2013). The main neuropsychological disturbances observed at the Department of Rehabilitation included considerable difficulty in sustaining attention when performing tasks. The patient was also easily distracted (loss of concentration). Moreover, the patient had attention deficits in the form of a slower processing of the information received by the system, that is a set of functions which cooperate with each other and other brain functions. It should be noted that the brain mechanism of attention and concentration involves such structures as the brainstem, thalamus, prefrontal cortex and the parietal association cortex, connected functionally by the activating part of the reticular formation. The patient also showed disturbed verbal fluency, that is disturbances in the fluency and speed of recalling and producing (in speech or writing) words from a given category (semantic fluency) or beginning with a given letter or sound (phonemic fluency). Moreover, the patient had mood swings. The patient tended to manipulate objects within her reach and use them to provoke aggressive behaviour. The patient showed a lack of motivation to undergo therapy. This factor could have largely depended on the pharmacological treatment. The patient received anti-anxiety drugs, hypnotics and sedatives, which resulted in psychomotor impairment. Moreover, the patient showed disturbances regarding the assessment of her own abilities and the executive function impairment associated with it. The patient had considerable difficulties with respect to planning and controlling her own actions and did not undertake any spontaneous activity. After the therapeutic sessions, she spent her free time doing nothing. She did not initiate conversations and haltingly responded to questions. The patient attempted to initiate verbal constant several times. During an attempt at a conversation, she was able to express her opinion. If the subject of the conversation was associated with issues of an emotional value to the patient, she commented on the parts of the conversation she found interesting. Observations of the patient showed that she gradually regained her motivation to act and the so-called “will to live.” The patient found conversations difficult due to the limited mobility of the tongue and lips resulting from the facial paresis caused by the stroke. The patient’s speech was quiet and monotone. Importantly, despite the difficulties associated with attempts at articulating words and forming sentences, the patient said the following:
"I like talking to you, I feel needed and seen by other people." We believe that the patient ceased to function according to accepted social requirements as a result of the cognitive and emotional deficits resulting from post-stroke complications and comorbidities and not because of motor dysfunctions. In such a patient a considerable improvement in cognitive, behavioural and social functions may be achieved through the systematic use of neuropsychological therapy. It should focus on eliminating and reducing the unwanted behaviour and reinforcing (or, in severe cases, re-educating) the frequency of the desired purposive behaviour by using positive reinforcement.

**DISCUSSION**

The case described shows that the patient had numerous concomitant diseases which might have contributed to the occurrence of a stroke. She suffered from vascular dementia, which very often results in a series of strokes and has a considerable influence on the impairment of cognitive functions (Maruszewski 1993; Ciarkowska 1992). Higher cortical functions, such as memory, orientation, comprehension, calculation, learning capacity, language and judgement, are impaired. Cognitive function impairment is accompanied by decreased control over emotional and social responses as well as over behaviour and motivation (Herzyk & Kądzielawa 1998). Dementia causes a significant decrease in intellectual functions and usually influences the basic everyday activities: washing, getting dressed, eating and personal hygiene. Dementia is associated with memory and thinking impairment. Memory function impairment is reflected by disturbed encoding, storing and recalling of recently received information; the patient displayed impairment with respect to short-time memory and working memory (memory storing data during its interpretation). The patient has diabetes mellitus type 2, which has a negative influence in the case of cerebrovascular diseases, often leading to an ischaemic stroke, and results in cognitive deficits. This is a metabolic disease having multifactor determinants and is characterised by hyperglycaemia resulting from increased hepatic glucose production, progressive impairment of insulin secretion in the pancreas and decreasing glucose degradation in the muscles associated with the development of insulin resistance. The resistance of the adipose tissue, muscles and liver to insulin constitutes the main pathophysiological mechanism in the development of diabetes. Diabetes negatively influences the structure and functions of the CNS (mainly the brain), causing both short- and long-term disturbances in the energy balance of neurons due to hyperglycaemia, which may result in secondary damage to neurons and cerebral blood vessels. The risk of impairment or structural damage to the brain due to metabolic imbalance may lead to changes in the form of an increased risk of ischaemic stroke and chronic sequelae on the structural, neurophysiological and neurobehavioral levels. Increased blood glucose levels cause chronic complications, circulatory diseases and doubly increase the risk of vascular dementia (Pąchalska 2008). They also cause cognitive and emotional...
impairment, e.g. verbal and spatial memory impairment, psychomotor disturbances and decreased short-time memory and working memory, which stores low amounts of data over short periods of time. Mood swings develop. The patient has subclinical hyperthyroidism, whose symptoms are very similar to those of affective and bipolar disorders. The symptoms accompanying this disease include frequent fractures, reduced affect, psychomotor agitation and tachycardia. The history-taking revealed also long-term nicotine dependence. Nicotine affects the peripheral and central nervous system in various ways. It may have both a stimulating and inhibitory effect. The basic types of the effect of nicotine on the neurotransmitter systems concern cholinergic and aminergic synapses. Nicotine also causes the release of catecholamines, which results in cardiovascular stimulation (increased heart rate, higher blood pressure and cardiac output as well as narrowed blood vessels). In the central nervous system, the presence of nicotine activates several neural pathways, leading to the release of acetylcholine, noradrenaline, serotonin, dopamine, and has an effect on the endocrine system. The release of acetylcholine is largely responsible for the increased vigilance and agitation caused by nicotine (Pąchalska 2011). Nicotine generally stimulates the entire brain. All these factors have a negative influence both on the condition of the blood vessels, something that particularly important in the case of a stroke, and on decreasing cognitive and emotional activity. The patient actively participated in neuropsychological therapy during her 3-week stay at the Department. The treatment program included exercises directed toward orientation in space, time and person as well as attention and memory (Pąchalska 2009; 2011). The orientation was partly improved, but remained unstable; there was psychomotor impairment, the patient remained passive, had attention deficits, difficulties sustaining attention on the task, a decreased rate of information processing and deficits in short-term and working memory. The patient requires further neuropsychological therapy conducted at home.

CONCLUSIONS

Stroke patients suffer from cognitive and emotional disorders. The main goal of physiotherapy and rehabilitation is to restore a patient’s independence in everyday life and improve their quality of life. A clinical psychologist plays a significant role in such treatment.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

REFERENCES


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