CLINICAL CASE REPORTS

Secondary thrombosis of the left internal carotid artery caused by a motor vehicle accident: a radiological case

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Abstract. *A rare case of occlusion of the internal carotid artery following a motor vehicle accident in a 34-year-old female victim who initially presented with clear consciousness and had normal computed tomogram of the brain is reported. Seven hours after the accident, the patient was unexpectedly diagnosed with the left hemisphere infarction, and two days later, she suffered from right hemiplegia and coma. The follow-up brain computed tomography scan showed an acute infarction of the left hemisphere of the cerebrum and severe cerebral edema. Anticoagulation therapy was administered, and emergency craniotomy for brain decompression was carried out. After 3.5 months, she was discharged and underwent regular follow-up in the outpatient department. Four years after the motor vehicle accident, the patient had intact awareness, was functionally independent, but remained with motor aphasia, right hand paralysis, and right leg paresis.*

Introduction

The incidence of cerebral infarction associated with head trauma has been reported to be in 1.9–2.9% of patients with the head and neck injury (1).

Occlusion of the internal carotid artery (ICA) caused by a motor vehicle accident in the patient with no neck vessel dissection, no intracranial hemorrhage, and no basal skull fracture, although uncommon, is associated with substantial morbidity and mortality and remains poorly understood (2). Initial recognition by clinicians is often difficult because of the diverse clinical manifestations and the delay in presentation of symptoms (3). The extent and timing of posttraumatic cerebral hemodynamic disturbances have significant implications for the monitoring and treatment of patients with head injury (4). Early diagnosis and successful management require a high index of clinical suspicion.

Secondary thrombosis may contribute to cerebral ischemia caused by traumatic brain injury (TBI), but the pathologic mechanisms of the ischemia within the injured brain are poorly understood. Posttraumatic intravascular coagulation that causes thrombosis has been suggested as an important factor for secondary ischemia after TBI. Intravascular thrombosis may arise from diffuse intravascular coagulation (DIC); however, delayed ischemia and thrombosis have also been found in head trauma patients without DIC (5).

Several other factors may result in posttraumatic cerebral ischemia: increased intracranial pressure, systemic arterial hypotension, cerebral edema, focal tissue compression from hematomas, microvascular pathology. Another potential cause of cerebral ischemia is the posttraumatic spasm of the large cerebral arteries (vasospasm), which has been documented by angiographic and transcranial Doppler studies. After head trauma, caused by a motor vehicle accident, even without evidence of hemorrhage such, as subarachnoid hemorrhage etc., vasospasm may occur, and the resulting cerebral ischemia is considered as the mechanism of cerebral infarction after accidents (6). Posttraumatic vasospasm has been associated with the finding of cerebral infarction on late computed tomography (CT) scans and with poor outcome in some head-injured patients (7).

Ischemic brain damage has been identified histologically in approximately 90% of patients who died following closed head injury. Ischemia has been called the single most important secondary insult, and several clinical head trauma studies have linked low cerebral blood flow (CBF) to poor outcome (4).

Here we report a case of the left ICA occlusion, unexpectedly diagnosed seven hours after the motor vehicle accident, with a short review of the literature.

Case report

On May 12, 2004, a 34-year-old woman was transported to the emergency room (ER) of the Hospital of Kaunas University of Medicine (KMUH) by

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an ambulance. She complained of headache, nausea, and vomiting that she began to feel five hours after experiencing head trauma in a motor vehicle accident. It was unclear if the patient had been unconscious for some time – most likely she had had an episode of amnesia after the accident.

After a prompt neurologist's consultation in the ER, a CT scan of the head was performed (Fig. 1), but no pathological changes were observed.

The woman was discharged for an outpatient treatment and follow-up, but in two hours, she came back to the emergency room for severely disordered speech and developing paresis of the right arm and leg.

Urgently, the second head CT scan was performed (Fig. 2).

The scan showed the signs of ischemia in the territory of the left ICA. The patient was hospitalized in the ward of head trauma of the Clinic of Neurosurgery of KMUH.

On examination, she had a temperature of 36.6°C, heart rate of 66 beats/min, and BP of 140/80 mm Hg. Respiratory and abdominal examinations were unremarkable.

Though the patient was conscious, she showed signs of sensomotor aphasia and could not precisely perform the tasks she was asked to do. She was not able to move her right hand, and the right leg (in a swathe) had accelerated muscle tension and increased reflexes; deviation of movements of her eyeballs to the left was observed. The pupils were equal, reacting to the light. No meningeal signs were observed.

Conclusions drawn during otorhinolaryngologic consultation: no verbal contact achieved, the symptoms of eye divergence and movement toward the side of the stimulated ear were observed.

Conclusions drawn during the ophthalmologic consultation: the pupils equal, reacting to the light; pink, flat, confined optic discs; wide, full-blooded veins. No pathological changes were observed in the retina bilaterally.

The results of transcranial dopplerography (TCDG) showed no ultrasonographic signals from the left ICA. The signals from the left posterior cerebral artery (PCA) were reactively increased. The signals from the right major head vessels were unremarkable. No signs of intracranial hypertension were observed. TCDG revealed a complete occlusion of the left ICA.

Neurosurgeon's conclusions after the cerebral angiography procedure were as follows: thrombosis of the left ICA at the level of the bifurcation; growing thrombus in the left ICA; extensive ischemic stroke of the left cerebral hemisphere.

In CT scans, performed on the 31st hour after the accident, a wide zone of ischemia in the left middle artery (MCA) and anterior cerebral artery
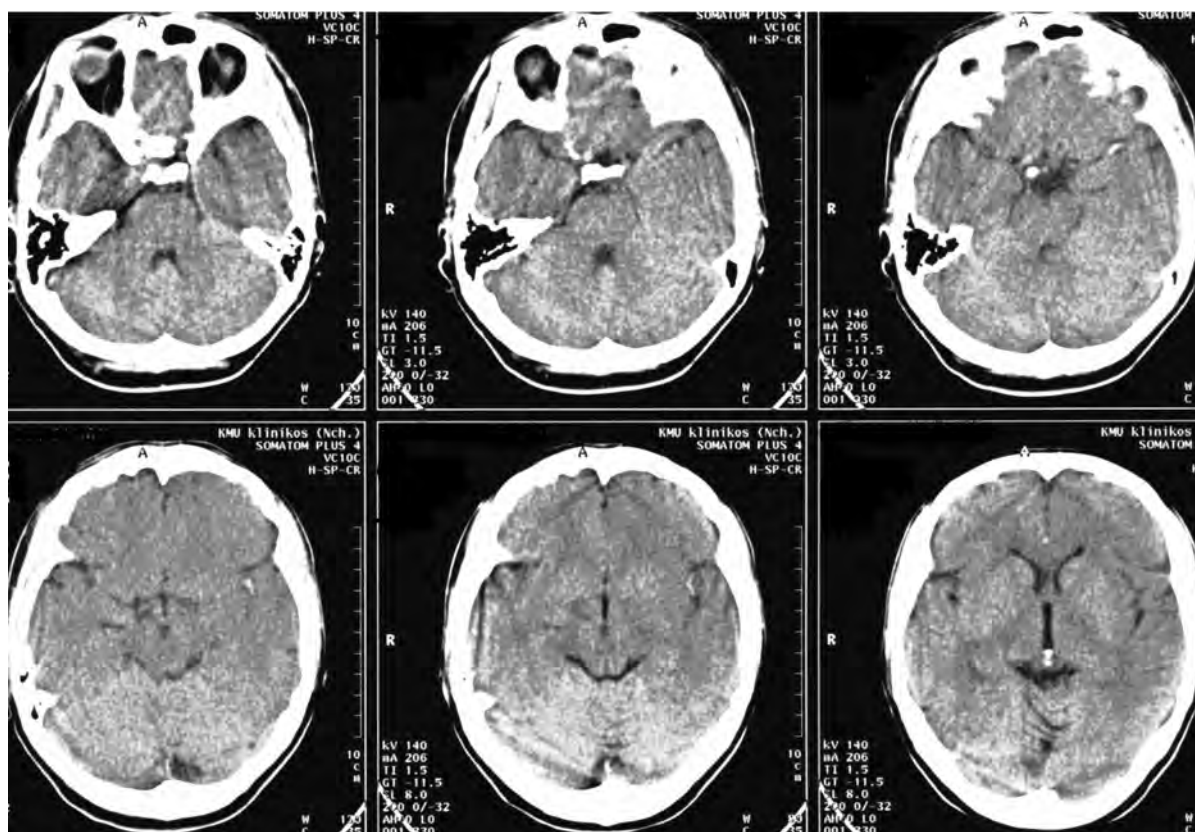

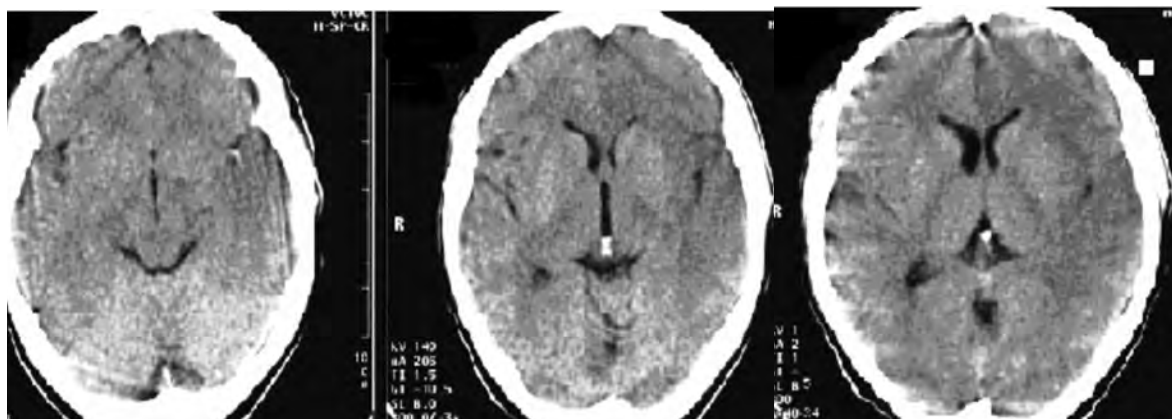*Fig. 1.* Initial computed tomography scan of the head – no pathological changes are observed

Fig. 2. Attenuation of the left lentiform nucleus, loss of the left insular ribbon and left hemispheric sulcus effacement – early signs of ischemia of the left internal carotid artery – are seen in these scans
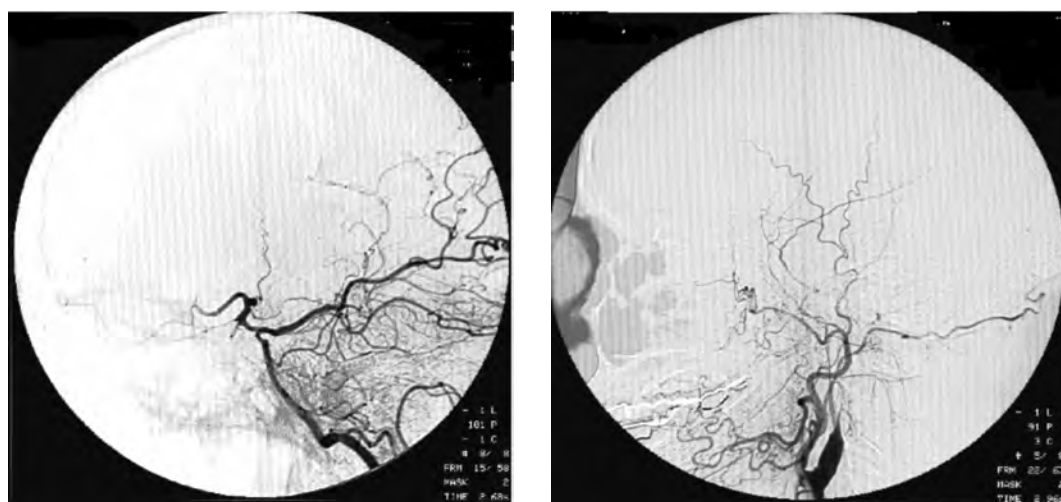

Fig. 3. Angiograms of the left internal carotid artery: occlusion of the left internal carotid artery at the level of the bifurcation

(ACA) regions, accompanied by the shift of midline structures about 1 cm to the right and terminal narrowing of basal cisterns, was observed (Fig. 4).

Thirty-two hours after the head injury, decompressive left temporoparietal craniectomy was performed due to increasing brain edema.

Next day after the operation, the patient was in the neurosurgical intensive care unit (ICU). She was unconscious, mechanically ventilated and sedated, so it was impossible to evaluate neurological status. Six days after the decompression, a nasogastric tube for enteral feeding was inserted. The patient's conscious state was 4 points on the Glasgow coma scale (GCS): motor, 2; verbal, 1; and eyes, 1.

The patient's condition gradually improved, and two weeks later, her conscious state was 10 points on the GCS.

On CT scan of the head, performed 19 days after the decompression (May 31, 2004), the hypodense zone of a smaller extent in the left temporoparietal region was observed, but gross mechanical shift of the brain and herniation across the falx and tentorium remained. Lesser degree of brain edema

and better differentiation of basal cisterns were observed. The patient was transferred from the ICU to the ward of head trauma of KMUH.

From June 2 to July 5, 2004, the patient showed an improvement of the conscious state, communication, complex movement, and eating. On July 5, 2004, she was transferred to the Clinic of Rehabilitation of KMUH.

The patient's neurological status in the Clinic of Rehabilitation was described as follows: conscious, unable to communicate verbally due to motor aphasia, able to perform basic tasks. Together with right hemiplegia, a partial central injury of right *n. facialis* was diagnosed.

Her Barthel index score was 10 points and functional independence measure (FIM), 26 points.

In the Clinic of Rehabilitation, an individual rehabilitation program composed of kinesitherapy, ergotherapy, massage, functional electrical stimulation (FES), thermo procedures, speech and language therapy (SLT), psychologist's and social worker's consultations was constituted and performed for the patient. During the rehabilitation course, self-sup-
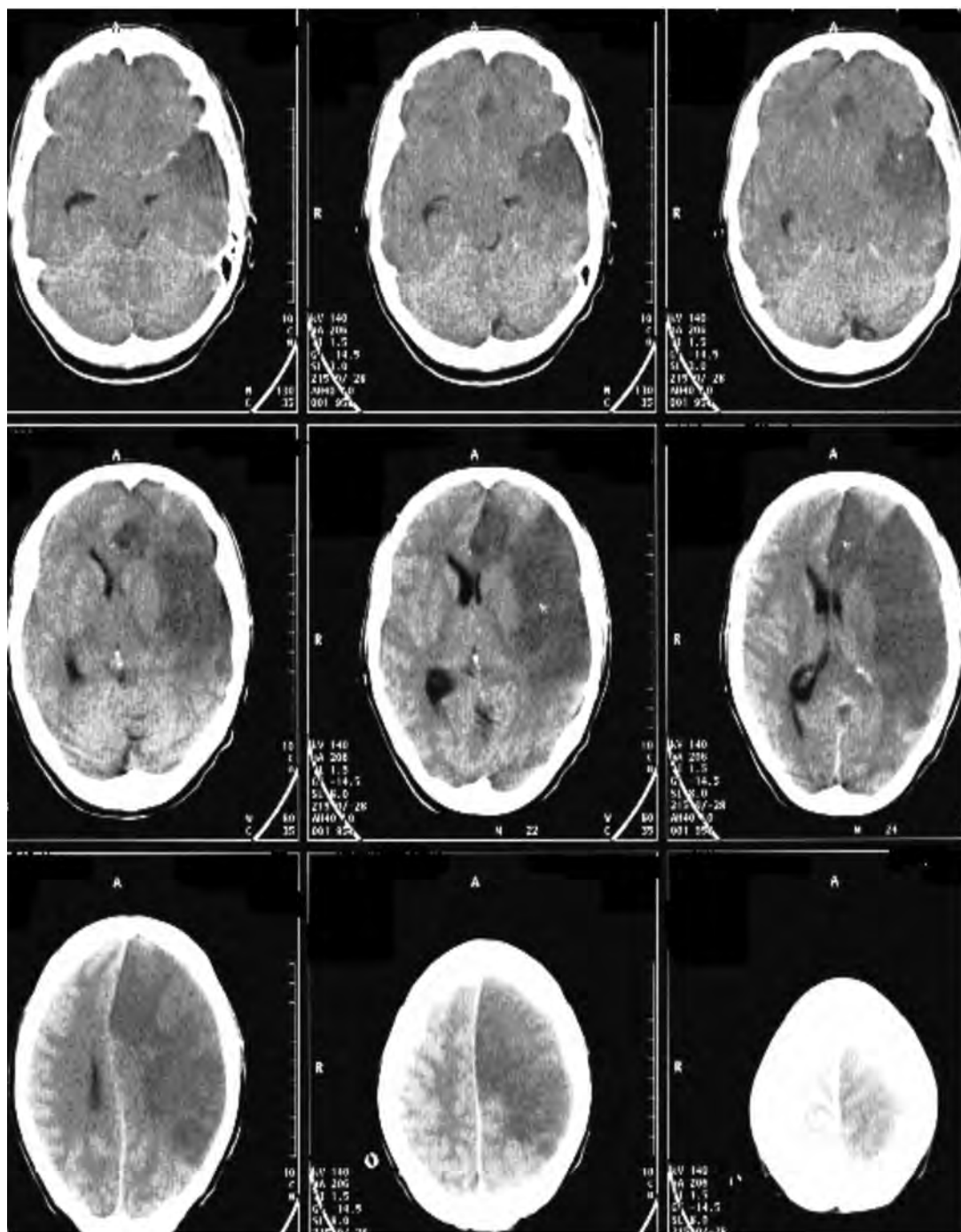
Fig. 4. Computed tomography scans performed on the 31st hour after the accident

In the left, a wide zone of ischemia in the regions of the middle cerebral artery and the anterior cerebral artery, accompanied by the shift of midline structures about 1 cm to the right and terminal narrowing of basal cisterns.

port of the patient slightly improved: she was able to eat by herself, partially control her excretion functions, but the right plegia and full motor aphasia remained, causing immobility and need for constant assistance and care.

On August 30, 2004, the patient was discharged home; her Barthel index score was 15 points.

On September 15, 2004, she came back to the Department of Neurosurgery and underwent cranioplas-tic surgery to reconstruct the skull defect. The operation and postoperative period went through without complications, and she was discharged home.

On April 25, 2005 she presented to the Department of Neurology complaining of severe neuralgic headache, and oral analgesics were prescribed for a symptomatic treatment. She had intact awareness, but transcortical motor aphasia, paresis and flexion contracture of the right hand remained. She lived
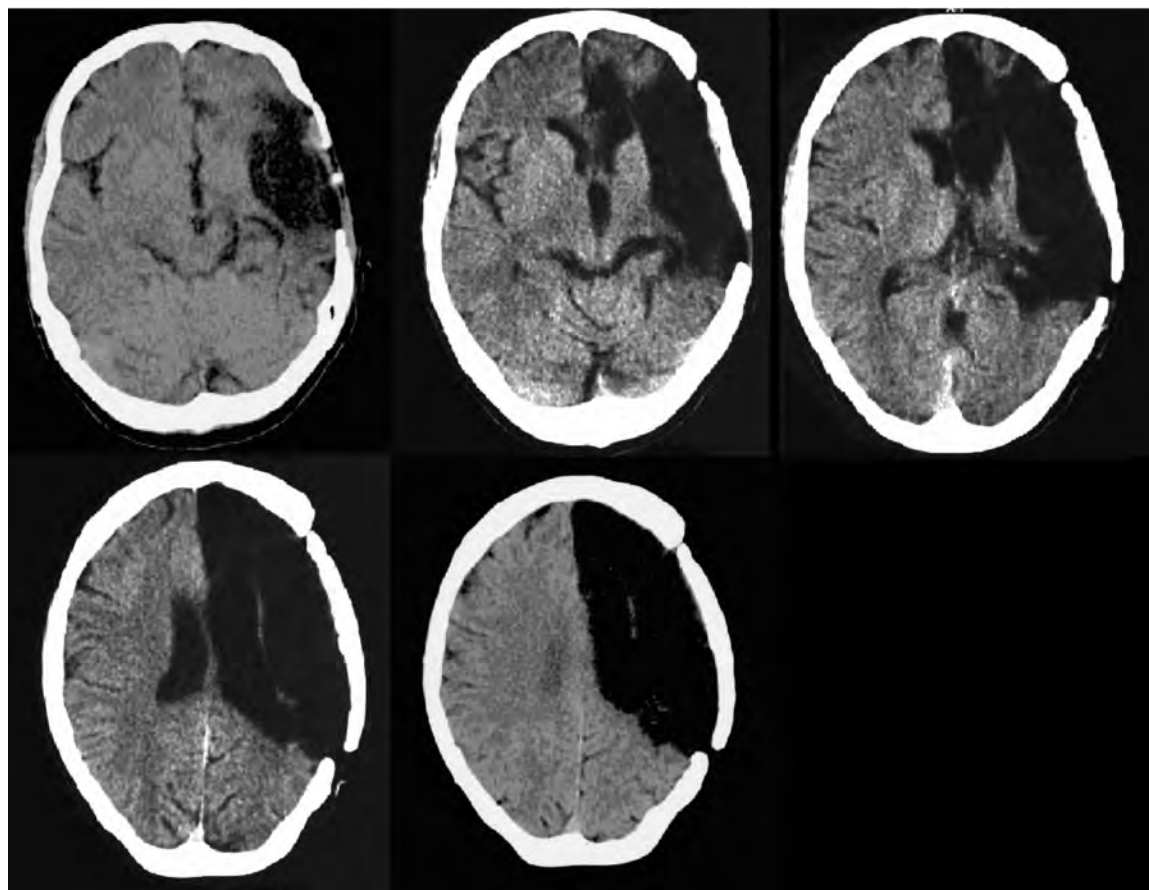
Fig. 5. Follow-up computed tomography scans of the head 2.5 years after the accident
Postcraniotomy and a wide poststroke brain lesion zone of liquor density in the left frontotemporoparietal region.

on her own, was functionally independent, able to walk with a stick. A control CT scan of the head was performed – residual postischemic alterations (wide cortical and subcortical poststroke lesions in the left MCA and ACA regions of the brain) were observed.

The same changes of the brain were seen on the follow-up CT scan, performed 2.5 years after the accident (Fig. 5).

After 4.5 years following the accident, the patient was functionally independent, walking with a cane, and needed no special attendance or help in the daily living.

Discussion

Even though the precise mechanism of cerebral infarction in this patient who had no neck injury or no basal skull fracture could not be evaluated, it is necessary to pay attention to the possibility of an unexpected cerebral infarction occurring after head injury.

Acute ischemia in the complete territory of the carotid artery may lead to massive cerebral edema with raised intracranial pressure and progression to coma and death due to uncal, cingulate, or tonsillar herniation (8).

Decompressive craniectomy, particularly when performed early after vessel occlusion, has been shown to reduce the volume of the infarction (8–10), has improved cerebral perfusion, survival, and neurological outcome (8, 11). These beneficial effects are likely to be linked to increases in collateral circulation (12) and reductions in tissue edema (13) together with improvements in oxygenation and energy metabolism in ischemic penumbra. Brain tissue monitoring in patients undergoing craniectomy for ischemic stroke has demonstrated that decompression leads to a reduction in intracranial pressure and an increase in brain tissue oxygen levels (14, 15).

In one of the first observational studies (16), authors from Charlottesville compared the results in patients undergoing decompressive craniectomy with historical control data from the National Traumatic Coma Data bank. They reported good outcomes in 37% of the patients as opposed to 16% of controls, with better results if decompression was performed within 48 hours of injury. Similar results have been reported from Griefswald by Guerra et al.: a favorable outcome rate of 58% and mortality rate of 19% (17). In 2000, De Luca et al. (18) reported

a 41% favorable outcome rate and an 18% mortality rate in a series of 22 patients.

The results of a few controlled studies suggest that an early start of intensive treatment is an important aspect of expert care (19–21). Functional recovery after focal brain lesions is dependent on the adaptive plasticity of the cerebral cortex and of the unaffected elements of the functional network (22). For the motor system, it has been convincingly demonstrated that after cortical injury, the adjacent spared cortical tissue as well as more remote cortical areas are altered resulting in a functionally modified network (23). Consequently, newly learned movements after focal cortical injury are represented over larger cortical territories (24) – an effect which is dependent on the intensity of rehabilitative training (25). Along with these changes in excitatory and inhibitory neurotransmitter systems, widespread structural changes with dendritic sprouting and synapse formation take place in spared regions of the damaged hemisphere, but also in the sensorimotor cortex of the hemisphere contra – lateral to the injury (26, 27). One concept, which is becoming increasingly recognized, is that not only the brain is capable of reorganization after a stroke as we have stated earlier but that the brain is primed to recover early after a stroke and that delays in rehabilitation will reduce the opportunity for maximal neurological recovery (28). Hence, the statement "time is brain recovery" appears to hold true for rehabilitation (29).

Evidence shows that we struggle to provide sufficiently intensive treatment and a sufficient number of therapies required improving outcomes. Bernhardt et al. (30) conducted an observational behavioral mapping study on 64 stroke patients in 5 acute stroke units. The authors reported that patients engaged in minimal therapeutic activity or moderate therapeutic activities for only 12.8% of the therapeutic day (8 AM to 5 PM). Patients were resting in bed 53% of the time and were alone for 60.4% of the time. Therapist contact constituted only 5.2% of the day. Previous studies have shown similar limited therapist–patient contact time in stroke units. Similarly, Lenze et al. (31) have reported that poor participation in therapy was common during inpatient rehabilitation and resulted in lower improvements in FIM scores and longer lengths of stay even when controlling for admission FIM scores (32).

Conclusions

It is necessary to pay attention to the possibility of an unexpected cerebral infarction occurring in a time interval after head injury, even though there are no early clinical and/or radiological manifestations.

Craniectomy performed timely is efficient for increasing brain edema caused by extensive ischemic stroke for decompression purpose and increases survival rates and chances for better neurological outcome.

Early and intensive rehabilitative training after a stroke increases the chances for maximal neurological recovery and best possible functional outcome: self-care, self-efficacy, and quality of life.

Po autoavarijos išsivysčiusi antrinė kairiosios vidinės miego arterijos trombozė (radiologinis atvejis)

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Raktažodžiai: vidinės miego arterijos okliuzija, kompiuterinė tomografija, kraniotomija, reabilitacija.

Santrauka. Straipsnyje aptariame retą atvejį, kai po autoavarijoje patirtos traumos sąmoningos 34 metų moters galvos kompiuterinėje tomogramoje patologinių pokyčių nerasta, tačiau, praėjus 7 val. po traumos, pacientei diagnozuota kairiosios vidinės miego arterijos okliuzija. Po dviejų dienų ligonei išsivystė dešiniosios pusės hemiplegija ir ištiko koma. Vėliau atliktoje kompiuterinėje tomogramoje rastas ūminis išeminis kairiojo smegenų pusrutulio infarktas ir ryški smegenų edema. Ligonė gydyta antikoaguliantais, galvos smegenų dekompresijai atlikta skubi kraniotomija. Pacientė iš ligoninės išrašyta po 3,5 mėn., skirta ambulatorinė stebėsena. Praėjus keturiems metams po autoavarijos, ligonė buvo sąmoninga, savarankiška, tačiau dėl motorinės afazijos nekalbėjo, paralyžiuota dešinė ranka, dešinės kojos parezė.

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