

ORIGINAL PAPER/ARTYKUŁ ORYGINALNY

# Decompressive hemicraniectomy in ischaemic stroke

## *Hemikraniektomia odbarczająca w udarze niedokrwiennym mózgu*

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### Abstract

**Background and purpose:** Hemispheric ischaemic stroke complicated by oedema is associated with high mortality. The results of randomized studies showed that decompressive hemicraniectomy performed in this group of patients could be beneficial. First experiences with implementation of hemicraniectomy in patients with brain infarct in our stroke centre are presented.

**Material and methods:** Between August 2007 and July 2008, four patients with hemispheric brain infarcts complicated by malignant oedema underwent decompressive hemicraniectomy within 72 hours from symptoms onset. Stroke severity was assessed with the National Institutes of Health Stroke Scale (NIHSS). Clinical outcome was assessed 3, 6 and 12 months after the event using the modified Rankin scale (mRS).

**Results:** In the first patient, the neurosurgical procedure included only decompressive hemicraniectomy, whereas in the other three duraplasty was performed additionally. The first patient died 23 days after the stroke onset due to acute respiratory failure. Another died at four months after the event, due to infectious complications. The remaining two patients presented severe functional disability 12 months after the procedure (mRS score 4).

**Conclusions:** Decompressive surgery with duraplasty can be a life-saving procedure for patients with brain oedema. To our knowledge, the presented cases are among the first reported cases of hemispheric ischaemic stroke treated with decompressive hemicraniectomy in Poland. Extended follow-

### Streszczenie

**Wstęp i cel pracy:** Półkulowy udar niedokrwienny mózgu powikłany obrzękiem mózgu obarczony jest dużą śmiertelnością. Wyniki badań z randomizacją wskazują na skuteczność kraniektomii odbarczającej w tej grupie chorych. W pracy przedstawiono pierwsze doświadczenia z zastosowania kraniektomii odbarczającej u chorych z udarem niedokrwiennym mózgu leczonych w Klinice Neurologii Wieku Podeszłego ŚUM w Katowicach.

**Materiał i metody:** W okresie od sierpnia 2007 r. do lipca 2008 r. u 4 chorych z półkulowym udarem niedokrwiennym mózgu powikłanym obrzękiem mózgu przeprowadzono zabieg kraniektomii odbarczającej maksymalnie do 72 godz. od wystąpienia objawów udaru. Stan neurologiczny chorych oceniono, stosując skalę *National Institutes of Health Stroke Scale* (NIHSS). Oceny stanu funkcjonalnego dokonano przy użyciu zmodyfikowanej skali Rankina w 3., 6. i 12. miesiącu po udarze.

**Wyniki:** U pierwszego chorego wykonano jedynie kraniektomię odbarczającą, a u kolejnych 3 dodatkowo plastykę opony twardej. Pierwszy z pacjentów zmarł w 23. dobie od wystąpienia udaru w wyniku niewydolności oddechowej, kolejny w 4. miesiącu z powodu powikłań infekcyjnych. U pozostałych dwóch pacjentów w 12. miesiącu od udaru utrzymywał się duży stopień niesprawności (4 stopień w skali Rankina).

**Wnioski:** Kraniektomia odbarczająca może być zabiegiem ratującym życie chorych z obrzękiem mózgu. Przedstawione przypadki są jednymi z pierwszych w Polsce opisanymi przy-

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up with a larger group of patients is necessary to assess long-term outcome.

**Key words:** decompressive craniectomy, stroke, stroke management, brain oedema.

## Introduction

Hemispheric brain oedema occurs in 10-15% of cases of supratentorial ischaemic stroke due to occlusion of the middle cerebral artery (MCA) [1-3]. Clinical signs of brain oedema usually appear between the second and fifth day after stroke onset, although they may occur as early as within 24 hours in 30% of cases. Risk factors for the occurrence of brain oedema in ischaemic stroke within the MCA area include: history of hypertension and heart failure, leukocytosis, visible hypodense lesion affecting > 50% of the region supplied by the MCA as seen in computed tomography (CT), and co-occurrence of stroke within the area supplied by another artery [4].

Increased intracranial pressure leads to disturbances of the cerebral microcirculation, and finally to brain death [5]. Despite intensive antioedema treatment using osmotherapy, barbiturate coma, hyperventilation or hypothermia, the case fatality is as high as 80% [6]. Decompressive treatment provides the opportunity to limit the consequences of increasing brain oedema. The surgical procedure includes excision of the major part of the cranium (fragments of the frontal, temporal, and parietal bones) followed by opening of the dura [7,8]. This enables the outward shift of the oedematous tissue and decreases the intracranial pressure by up to 70% [9].

The first analysis of the efficacy of hemicraniectomy in patients with hemispheric ischaemic stroke was published in the 1970s [10]. It was not until 2004, however, that a meta-analysis of a case series including 138 patients showed that 58% of those patients died or were severely handicapped 4 months after their stroke; this was considered as a significant therapeutic benefit in view of the rather poor outcome of conservative treatment, especially in younger patients [11]. A combined analysis of three European randomized controlled trials, published in 2007, showed that hemicraniectomy in patients with hemispheric stroke due to occlusion of the MCA performed within the first 48 hours after stroke onset reduces mortality and improves functional status assessed one year after stroke [12]. Major con-

traversies are related to the functional status of patients with stroke of the dominant hemisphere associated with severe speech disturbances, although the comparison of outcome between patients with stroke within the dominant and non-dominant hemisphere did not reveal any difference in functional status [11]. This may result from the fact that scales used to assess functional status focus more on motor dysfunction [11].

**Słowa kluczowe:** kraniektomia odbarczająca, udar mózgu, leczenie udaru mózgu, obrzęk mózgu.

Current guidelines of the American Stroke Association consider hemicraniectomy as a potentially life-saving procedure (class II, level B) [13]. European guidelines published in 2008 by the European Stroke Association recommend surgical decompression in patients aged < 60 who suffer from hemispheric ischaemic stroke due to occlusion of the MCA; the procedure should be performed within 48 hours after stroke onset (class I, level A) [14].

According to our knowledge, the cases of decompressive treatment of hemispheric ischaemic stroke reported in this paper are among the first cases described in Poland. We report our experience with the use of decompressive hemicraniectomy in patients with brain oedema due to hemispheric ischaemic stroke.

## Material and methods

Since July 2007, decompressive hemicraniectomy has been performed in four patients (including three men) hospitalized in the stroke unit of Department of Neurology, Ageing, Degenerative and Cerebrovascular Diseases, Silesian Medical University of Katowice. All patients described here were diagnosed with ischaemic stroke complicated by hemispheric brain oedema. Qualification of patients for the surgical treatment was based on gradual worsening of the neurological status with incipient consciousness disturbances (Table 1). Neurological deficit of patients was assessed with the National Institutes of Health Stroke Scale (NIHSS), and their functional status was assessed with modified Rankin scale (mRS). The clinical diagnosis of hemispheric oedema was confirmed with control CT of the head. Doppler ultrasound of carotid and

vertebral arteries was performed within 48 hours after admission. The final decision on neurosurgical treatment was made after consent was obtained from the patient's next of kin. The first patient had hemicraniectomy performed only, and the other three patients had duraplasty performed as well. The detailed surgical technique of the procedure is described in available literature [7,8]. Characteristics of patients who qualified for the surgery are provided in Table 2. Table 3 presents the assessment of neurological and functional status on discharge, at day 30 after stroke, as well as 3, 6 and 12 months after treatment.

## Case reports

### Patient No. 1

A 59-year-old man was admitted because of weakness of left limbs that occurred suddenly about one hour

**Table 1.** Inclusion criteria for decompressive hemicraniectomy based on HAMLET, DESTINY and DECIMAL trials (ESO guidelines, 2008) [14]

NIHSS score > 15
Decrease in level of consciousness ( $\geq 1$ in item 1a of the NIHSS)
Infarct signs on CT of 50% or more of the MCA territory
Age 18-60 years
Surgery < 48 hours after onset

NIHSS – National Institutes of Health Stroke Scale, MCA – middle cerebral artery, CT – computed tomography

before the presentation. On admission, the patient was conscious but in limited verbal contact and with left-sided hemiplegia (NIHSS score, 17 pts) (Table 2). Head CT performed on admission revealed early signs of ischaemic stroke. The patient did not receive thrombolytic treatment because of highly elevated blood pres-

**Table 2.** Characteristics of patients treated with decompressive hemicraniectomy

Patient No.	Age/Sex	Occluded artery	NIHSS on admission	Treatment with rt-PA	Interval between onset of stroke and surgery	NIHSS before the surgery	Other methods of treatment
1	60/M	right MCA	17	no	43	18	thiopental ( <i>i.v.</i> pump) mannitol <i>i.v.</i> 75 g/day
2	60/M	right ICA	8	unknown*	69	22	thiopental ( <i>i.v.</i> pump) mannitol <i>i.v.</i> 150 g/day
3	58/F	right ICA	13	yes (< 3 h)	44	20	thiopental ( <i>i.v.</i> pump) mannitol <i>i.v.</i> 200 g/day
4	66/F	left ICA	13	no	57	25	thiopental ( <i>i.v.</i> pump) mannitol <i>i.v.</i> 80 g/day dexamethasone <i>i.v.</i> 12 mg/day

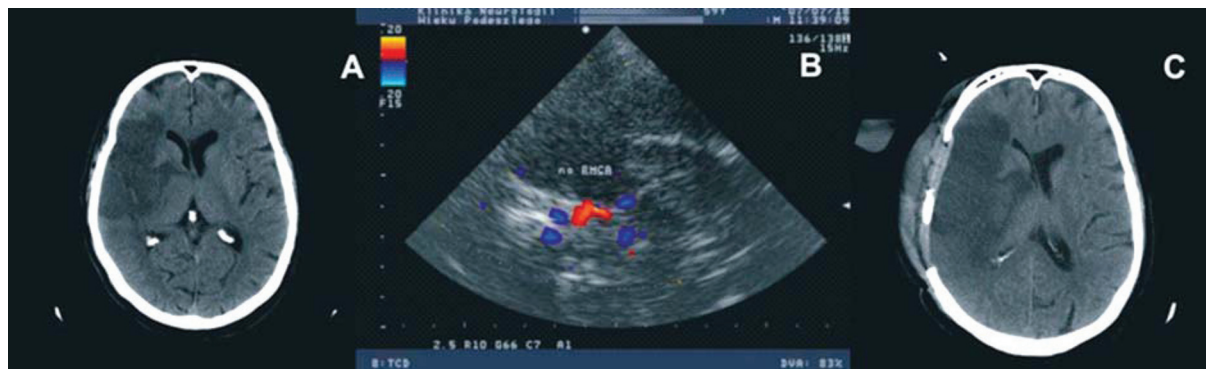
M – male, K – female, NIHSS – National Institutes of Health Stroke Scale, MCA – middle cerebral artery, ICA – right internal carotid artery

\*Patient enrolled in the clinical trial with masked allocation to the treatment either with rt-PA or with placebo

**Table 3.** Outcome after decompressive surgery

Patient No.	Age/Sex	Modified Rankin Scale				Discharge		
		day 30	3 months	6 months	12 months	Time	NIHSS score	Referral
1	60/M	6	6	6	6	day 23	–	none (death)
2	60/M	5	5	5	4	day 80	17	rehabilitation unit
3	58/M	5	4	4	4	day 73	12	rehabilitation unit
4	66/F	5	5	6	6	day 67	18	rehabilitation unit

M – male, K – female, NIHSS – National Institutes of Health Stroke Scale



**Fig. 1.** A 59-year-old patient with middle cerebral artery territory ischaemic stroke (A), with occlusion of the middle cerebral artery diagnosed with transcranial colour-coded sonography (B), in whom hemicraniectomy without duraplasty was performed (C)



**Fig. 2.** A 60-year-old patient with a right hemisphere ischaemic stroke (A), with occlusion of the right internal carotid artery diagnosed with colour-coded sonography (B), in whom hemicraniectomy with duraplasty was performed (C)

sure that persisted despite the blood pressure lowering treatment. Transcranial colour-coded Doppler showed occlusion of the right MCA (Fig. 1).

On the second day, the gradual decrease of consciousness was observed. Control head CT performed 36 hours after the onset of symptoms revealed massive oedema of the right cerebral hemisphere, with compression and shift of the ventricular system to the left.

Hemicraniectomy was performed 43 hours after the onset of stroke. During the surgery, the surgeon decided not to perform duraplasty (Fig. 1). After the surgery, thiopental-induced coma was initiated and maintained for another 4 days. Head CT performed at day 7 showed decreased oedema of the right cerebral hemisphere without shift of the ventricular system. Mechanical ventilation was withdrawn at day 11, and intravenous osmotherapy was stopped at day 18. The hospital stay was complicated by urinary tract infection and respiratory tract infection. During the following days, a gradual improvement of neurological status was observed,

including a normal level of consciousness. The patient started to stand up at the beginning of the fourth week of the hospital stay. At day 23, sudden cardiac arrhythmia occurred with cardiopulmonary failure and consequent death. The autopsy did not reveal any extracerebral cause of death.

### Patient No. 2

A 60-year-old man was admitted at the third hour after the onset of left-sided hemiparesis. Neurological examination on admission revealed left central facial palsy, left-sided hemiparesis, left-sided sensory loss and hemineglect (NIHSS, 8 pts.). Head CT excluded intracerebral haemorrhage and did not reveal any signs of acute ischaemic stroke. The patient entered the clinical trial that assessed the efficacy of treatment with recombinant tissue plasminogen activator given between 3 and 4.5 hours after the onset of stroke. Duplex Doppler performed at day 2 showed occlusion of the right internal

carotid artery (Fig. 2). At the border of day 2 and day 3, gradual worsening of neurological deficit was observed, including decrease of consciousness. CT scan revealed oedema of the right cerebral hemisphere with midline shift to the left. Antioedema treatment was initiated; thiopental-induced coma and mechanical ventilation were started. After consultation with the monitoring centre of the trial, the method of treatment used in the trial remained masked, because it had no impact on the potential surgical treatment performed at least 24 hours after the potential use of a thrombolytic drug.

Decompressive hemicraniectomy with duraplasty was performed 69 hours after the onset of stroke (Fig. 2). Head CT at day 1 after the surgery showed partial haemorrhagic transformation of the ischaemic lesion and continuing mass effect with compression and shift of the ventricular system, as well as with bulging of the brain structures through the craniectomy window. Barbiturate coma was maintained for the next 5 days after the surgery, mechanical ventilation was continued for the next 14 days after the surgery, and osmotic drugs were used up to day 26 after the surgery (Table 2). Magnetic resonance imaging performed at day 24 after the craniectomy showed decreased features of increased intracranial pressure without midline shift. The hospital stay was complicated by infections of the urinary and respiratory tracts and by pulmonary embolism. Neurological status of the patient gradually improved during the following days; intensive physiotherapy and speech therapy were continued. The patient was transferred to the rehabilitation centre at day 80 of hospital stay. Functional status of the patient was graded on mRS as 5 at 6 months after stroke onset, and as 4 at 12 months after stroke onset.

### Patient No. 3

A 58-year-old man was admitted within the first hour after the onset of left-sided hemiparesis. Neurological examination on admission revealed gaze deviation to the right, left-sided hemianopia, left central facial palsy, dysarthria and left-sided hemiparesis (NIHSS, 13 pts.). Head CT on admission showed hyperdense right MCA and subtle effacement of the sulci within the right cerebral hemisphere. The patient was qualified for thrombolytic treatment according to the POLKARD criteria. At day 2, the patient deteriorated clinically – disorders of consciousness and unstable blood pressure occurred. Head CT showed oedema of the right cerebral hemisphere and barbiturate coma was initiated.

Decompressive hemicraniectomy with duraplasty was performed 43 hours after the onset of stroke. Thiopental coma was prolonged to day 7 after the surgery because of the presence of oedema of the right cerebral hemisphere with compression of the ventricular system and its subtle shift to the left, as seen in repeated head CT studies (day 1 and day 6 after the surgery). Mechanical ventilation was withdrawn at day 8 after the surgery, and the osmotic drugs were stopped at day 29 after the surgery (Table 2). Duplex Doppler revealed occlusion of the right internal carotid artery with the collateral circulation within the circle of Willis. During the following days, the blood pressure remained unstable and required numerous modifications of treatment. The hospital stay was complicated by urinary tract infection and respiratory tract infection. The patient received intensive physiotherapy and speech therapy; his neurological status gradually improved (mRS = 4) and he was transferred to the rehabilitation centre at day 73. One year after stroke onset, the patient's functional status had not changed substantially (mRS = 4).

### Patient No. 4

A 66-year-old woman was admitted because of consciousness disturbances and weakness of the right limbs that appeared suddenly about an hour before admission. Neurological examination on admission revealed sensory-motor aphasia, right central facial palsy and right-sided hemiparesis (NIHSS, 13 pts.). Head CT excluded intracerebral haemorrhage and showed early signs of ischaemia within the area supplied by the left MCA. The patient did not receive thrombolytic treatment because of thrombocytopenia. At day 2, the patient's general and neurological status deteriorated. Control head CT showed oedema of the left cerebral hemisphere with compression and shift of the ventricular system to the right.

Decompressive hemicraniectomy was performed 57 hours after the onset of stroke. Thiopental-induced coma was maintained for the following three days, and osmotic drugs were withdrawn after 10 days (Table 2). On the second day of her hospital stay, emergent therapy with corticosteroid (intravenous dexamethasone, 12 mg daily) was used because of rapidly increasing signs of increased intracranial pressure; it was withdrawn gradually afterwards. Control head CT performed at day 10 after the surgery showed continued oedema of the left hemisphere, decreased compression of the left lateral ventricle without midline shift, and bulging of

the brain structures into the craniotomy window. After the withdrawal of barbiturate sedation, respiratory failure requiring mechanical ventilation continued for two weeks after surgery. Treatment was complicated by infections of the respiratory and urinary tracts. Despite speech therapy, global aphasia persisted. At day 67, the patient was transferred to the rehabilitation centre; after 10 days an episode of aspiration occurred and she developed subsequent Mendelson syndrome and septic complications. The patient was then transferred to the intensive care unit, where she died at day 108 after the onset of stroke.

## Discussion

Introduction of decompressive hemicraniectomy to the treatment of patients with hemispheric ischaemic stroke complicated by brain oedema requires good co-operation between the staff of the stroke unit and the neurosurgical team. The essence of this co-operation is proper understanding of the purpose of the procedure. Although surgical decompression in patients with hemispheric oedema due to ischaemic stroke has been performed for more than 50 years [11], it was not until 2004 when a meta-analysis of a case series was published and a meta-analysis of three randomized trials published in 2007 started a widespread discussion on this topic [11,12].

Surgical treatment in cases where neuroimaging shows no pathological lesions, or only an ischaemic area is visible, might be questionable. It should be kept in mind, however, that decompressive hemicraniectomy is a preventive measure aimed at avoiding the consequences of a massive brain oedema. It is too late to perform that procedure if there are clear signs of oedema with brain herniation. It is important to note that compression of the nervous tissue due to the oedema is not the major threat, as shown by *in vitro* studies [15]. Much more important are disturbances of the cerebral circulation, caused by increased intracranial pressure, including disturbances of the microcirculation within the compressed tissue [5]. The real challenge in qualification of patients for that procedure is related to the accurate assessment of the risk of brain oedema in the first few days after stroke onset.

The criterion of 48 hours, used in randomized studies, was chosen arbitrarily. Benefit from this type of treatment has been described also in cases treated beyond the 48-hour window [11,16,17]. Our decision to

perform hemicraniectomy in the second patient described here was based on those observations. American Stroke Association guidelines published in 2007 do not provide a recommended time-window for the procedure [13]. Only the recommendations of ESO from May 2008 clearly suggest a 48-hour window and the age of potential candidates (below 60) [14]. The results of the HAMLET study published in 2009 also confirm lack of efficacy if the procedure is performed after 48 hours [18]. The optimal timing of the procedure is still an open question. Some data suggest that the procedure should be performed as quickly as possible, even before 24 hours after the onset of symptoms [19]. It might be doubtful, however, whether it is possible to assess the risk of brain oedema accurately within 24 hours after the onset of symptoms; consequently the possibility exists that some patients might be treated unnecessarily.

Unfortunately, we have no diagnostic method that might support the early evaluation of the risk of involved hemispheric oedema in case of stroke. Neurologists in treatment of stroke patients are deeply aware of that problem in relation to the thrombolytic treatment, where each minute left decreases the real opportunity to obtain a good outcome. Similarly we believe that decompressive surgery should be considered as an emergency procedure and should be performed as quickly as possible after the indications for the surgery are established. The surgical technique is also of major importance. Craniectomy alone results in a 15% decrease of intracranial pressure, while adding duraplasty provided a decrease of intracranial pressure by 70% [9]. The best decompression can be achieved, therefore, with craniectomy combined with duraplasty. Lack of duraplasty in the first described case might be a potential mistake that consequently led to the permanent lesions in the brainstem. On the other hand, quite delayed acute cardiopulmonary failure in a patient who previously improved neurologically, regained consciousness and had no cardiopulmonary disturbances, may put in doubt primary cerebral cause of death.

Recently published guidelines related to the management of acute ischaemic stroke include indications for surgical treatment; they lack, however, recommendations related to the management of patients after craniectomy. We used barbiturate sedation and osmotic treatment with mannitol; patient no. 4 received corticosteroids additionally. As data related to the efficacy of those methods are lacking [13], one may ask about the necessity of their use in the management of our patients. The rationale for that type of management comes from the practices of our centre in presence of brain oedema

in the course of ischaemic stroke and simultaneously in the face of the lack of recommendations related to post-operative management of those patients. The greatest doubts are related to the use of thiopental-induced coma. It is associated with relatively high risk of cardiovascular complications and requires a prolonged period of intubation and mechanical ventilation, which are associated with additional complications (as observed also in all our patients). It also affects the long-term outcome unfavourably [16]. The use of corticosteroids is also associated with a high risk of complications, mainly infectious ones, and therefore it is not recommended at present [13]. The decision to use corticosteroids in one of the patients described here resulted from the rapidly progressive brain oedema and the high risk of herniation.

Patients' qualification and surgical treatment in the early stage of brain oedema increase the probability of maintaining respiratory function after the procedure, which is another argument for quick surgery in those cases. Although the efficacy of mannitol in the treatment of brain oedema due to ischaemic stroke is unproven [20], both ASA and ESO permit the use of osmotic therapy [13,14].

During the subsequent stages of management, complications typical for immobilized stroke patients were observed in the reported cases, obviously influenced by the persistent severe neurological deficit. Further complications occurred in the patients during the late rehabilitation process, and they were lethal in the last case described here.

Continuation of rehabilitation presents another problem after the successful management of acute ischaemic stroke and its complications. After rehabilitation in institutions, patients received physiotherapy at home and regained modest independence at 6 months after the stroke, although they could not stand or walk, even with the assistance of other persons. Their functional status did not change also after 12 months. Therefore, we decided against another surgical procedure of reconstruction of the cranium; we assumed that the risk of head trauma in those patients was small. The patients described here stayed at home and their families were responsible for their care despite the severe functional deficit and severe dependency; it allowed them to maintain contact with the next of kin. The location of the stroke is important in this matter. In patients no. 1-3, the lesion affected the right hemisphere, and therefore the patients could communicate with significant others because of lack of speech difficulties. In the case of left hemispheric stroke, a severe deficit of speech abilities

should be expected. The communication with the family and/or caregivers before the decision related to the surgical decompression should highlight the fact that the basic benefit of surgical treatment is decreased mortality, but the risk of severe functional deficit and severe communication difficulties in case of left hemispheric stroke is high.

The prognosis related to survival and functional status after ischaemic stroke with hemispheric brain oedema was presented in the analysis of the DECIMAL, DESTINY, and HAMLET studies [12]. Unfavourable outcome defined as 5 or 6 (death) points on mRS was noted in 5% and 71% of patients, respectively, in case of conservative treatment in comparison to 4% and 22%, respectively, in case of surgical treatment ( $p < 0.001$ ). Functional status evaluated after one-year follow-up was assessed as 3 or 4 points on mRS in 60% of patients. We cannot compare our results with the above-mentioned ones due to very small absolute numbers.

According to ESO guidelines from 2008, decompressive hemicraniectomy in patients with hemispheric ischaemic stroke may be a life-saving method of treatment. According to evidence-based medicine criteria, the use of decompressive hemicraniectomy has the same strength of recommendation as the use of thrombolytic therapy in a 3-hour time window (class I, level A) [14].

## Conclusions

1. Decompressive surgery with duraplasty can be a life-saving procedure for patients with hemispheric brain oedema.
2. To our knowledge, the presented cases are among the first reported cases of hemispheric ischaemic stroke treated with decompressive hemicraniectomy in Poland.
3. Extended follow-up with a larger group of patients is necessary to assess long-term outcome.

## Disclosure

Authors report no conflict of interest.

## References

1. Moulin D.E., Lo R., Chiang J., et al. Prognosis in middle cerebral artery occlusion. *Stroke* 1985; 16: 282-284.
2. Ng L.K., Nimmannitya J. Massive cerebral infarction with severe brain swelling: a clinicopathological study. *Stroke* 1970; 1: 158-163.

3. Mendel T. "Malignant" middle cerebral artery territory infarction. *Neurol Neurochir Pol* 2005; 39: 366-371.
4. Kasner S.E., Demchuk A.M., Berrouschot J., et al. Predictors of fatal brain edema in massive hemispheric ischemic stroke. *Stroke* 2001; 32: 2117-2123.
5. Rudzinski W., Swiat M., Tomaszewski M., et al. Cerebral hemodynamics and investigations of cerebral blood flow regulation. *Nucl Med Rev Cent East Eur* 2007; 10: 29-42.
6. Hacke W., Schwab S., Horn M., et al. 'Malignant' middle cerebral artery territory infarction: clinical course and prognostic signs. *Arch Neurol* 1996; 53: 309-315.
7. Coplin W.M., Cullen N.K., Policherla P.N., et al. Safety and feasibility of craniectomy with duraplasty as the initial surgical intervention for severe traumatic brain injury. *J Trauma* 2001; 50: 1050-1059.
8. Schwab S., Steiner T., Aschoff A., et al. Early hemicraniectomy in patients with complete middle cerebral artery infarction. *Stroke* 1998; 29: 1888-1893.
9. Smith E.R., Carter B.S., Ogilvy C.S. Proposed use of prophylactic decompressive craniectomy in poor-grade aneurysmal subarachnoid hemorrhage patients presenting with associated large sylvian hematomas. *Neurosurgery* 2002; 51: 117-124.
10. Ivamoto H.S., Numoto M., Donaghy R.M. Surgical decompression for cerebral and cerebellar infarcts. *Stroke* 1974; 5: 365-370.
11. Gupta R., Connolly E.S., Mayer S., et al. Hemicraniectomy for massive middle cerebral artery territory infarction: a systematic review. *Stroke* 2004; 35: 539-543.
12. Vahedi K., Hofmeijer J., Juettler E., et al. Early decompressive surgery in malignant infarction of the middle cerebral artery: a pooled analysis of three randomised controlled trials. *Lancet Neurol* 2007; 6: 215-222.
13. Adams H.P., Jr., del Zoppo G., Alberts M.J., et al. Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: the American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. *Stroke* 2007; 38: 1655-1711.
14. European Stroke Organisation (ESO) Executive Committee; ESO Writing Committee. Guidelines for management of ischemic stroke and transient ischaemic attack 2008. *Cerebrovasc Dis* 2008; 25: 457-507.
15. Sahay K.B., Mehrotra R., Sachdeva U., et al. Elastomechanical characterization of brain tissues. *J Biomech* 1992; 25: 319-326.
16. Foerch C., Lang J.M., Krause J., et al. Functional impairment, disability, and quality of life outcome after decompressive hemicraniectomy in malignant middle cerebral artery infarction. *J Neurosurg* 2004; 101: 248-254.
17. Pillai A., Menon S.K., Kumar S., et al. Decompressive hemicraniectomy in malignant middle cerebral artery infarction: an analysis of long-term outcome and factors in patient selection. *J Neurosurg* 2007; 106: 59-65.
18. Hofmeijer J., Kappelle L.J., Algra A., et al. Surgical decompression for space-occupying cerebral infarction (the Hemicraniectomy After Middle Cerebral Artery infarction with Life-threatening Edema Trial [HAMLET]): a multicentre, open, randomised trial. *Lancet Neurol* 2009; 8: 326-333.
19. Chen C.C., Cho D.Y., Tsai S.C. Outcome of and prognostic factors for decompressive hemicraniectomy in malignant middle cerebral artery infarction. *J Clin Neurosci* 2007; 14: 317-321.
20. Bardutzky J., Schwab S. Antiedema therapy in ischemic stroke. *Stroke* 2007; 38: 3084-3094.