

The Tactics of Surgical Treatment in Patients with Cerebellar Infarction

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The aim of the study is to specify tactics of surgical treatment of cerebellar infarction (CI).

MATERIAL AND METHODS The results of treatment of 80 patients with CI were studied. According to the clinical course of stroke, the patients were divided into 2 groups. The group of malicious cerebellar infarction included 55 patients (69%) (Group I), the group cerebellar infarction with benign course included 25 patients (31%) (Group II). Patients of Group I were divided into subgroups, in one of them surgical treatment was performed (surgical subgroup), and in the another one, only conservative (conservative subgroup) treatment was performed. In the surgical subgroup, 16 patients underwent isolated ventriculostomy, 5 — posterior fossa decompression (PFD), 18 — combination of ventriculostomy and PFD. The criteria of efficacy of surgery were recovery of consciousness and/or IV ventricle and the quadrigeminal cistern configurations. Results of treatment were assessed according to the Glasgow Outcome Scale.

RESULTS Malicious cerebellar infarctions occurred more frequently in patients with volume of ischemia exceeding 20 cm³ in the first day of the disease. The threshold value of mass effect, which may cause further a malignant cerebellar infarction, in the first day of the disease was score 3 according to the M. Jauss scale. In group of patients with malignant cerebellar infarction, surgical treatment reduced the mortality rate of occlusion and dislocation syndrome by 35.8%. The most effective type of intervention was a combination of decompressive trepanation of the posterior cranial fossa and external ventricular drainage. Combined ventriculostomy and PFD were 34 % more effective than just ventriculostomy, and 38 % more effective than just PFD.

CONCLUSION Patients with cerebellar infarction of more than 20 cm³ and signs of a mass effect in the posterior cranial fossa score 3 or higher according to M. Jauss scale, are prone to developing a malicious course of the disease. After the development of clinical manifestations of occlusive and dislocation syndrome, they need surgical treatment.

In the surgical treatment of malignant cerebellar infarction, ventriculostomy with PFD are advisable, as each operation separately does not always provide a necessary effect in decompensation of dislocation syndrome.

Keywords: cerebellar infarction, occlusive hydrocephalus, brain stem compression, surgical treatment, posterior fossa decompression, ventriculostomy

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AOH — acute occlusive hydrocephalus
 CT — computed tomography
 CI — cerebellar infarction
 CSF — cerebrospinal fluid
 EVD — external ventricular drainage
 GOS — Glasgow Outcome Scale
 ICI — isolated cerebellar infarction
 IS — ischemic stroke
 ODS — occlusive dislocation syndrome
 PCF — posterior cranial fossa
 PFD — posterior fossa decompression
 VCC — ventriculo-cranial coefficients

BACKGROUND

Isolated cerebellar infarction (ICI) accounts for 10% of ischemic strokes (IS) in the vertebrobasilar system, and 2–3% of all IS of the brain [1-3]. With the development of significant edema around the ischemic area, leading to compression of the brain stem and the development of acute occlusive hydrocephalus (AOH), we speak of a malignant cerebellar infarction (MCI) [4, 5]. Malignant cerebellar infarction is detected in 5–15% of all patients with cerebellar stroke. Against the background of conservative treatment, the mortality rate in malignant cerebellar infarction reaches 85% [6, 7].

The main causes of cerebellar infarction are hemodynamically significant stenoses of extra- and intracranial arteries, thrombosis or embolism, usually in the system of the superior cerebellar arteries [2, 3].

The main factor contributing to the development of malignant cerebellar infarction is a lesion of the cerebellum in a volume of at least 1/3 of its hemisphere [8, 9]. A significant amount of damage is associated with a lack of collateral blood supply in the cerebellar artery systems [3].

Life-threatening complications of CI (occlusive hydrocephalus, direct compression of the brain stem) are manifested by progressive depression of consciousness, hemi- and/or tetraparesis, bulbar syndrome and oculomotor disorders [10-15].

With the development of life-threatening conditions, the decision on the need for surgical treatment should be made without delay. The tactics of managing patients before the development of the occlusive dislocation syndrome (ODS) and the optimal amount of surgical intervention for developing malignant cerebellar infarction is still the subject of discussion, which is explained by the rarity of pathology, the lack of randomized studies and small numbers of published observations [16-18].

In this paper, we present our own experience in the surgical treatment of patients with malignant myocardial infarction, as well as comparing the effectiveness of various intervention methods and postoperative outcomes with outcomes with conservative treatment.

The aim of study is to clarify the tactics of surgical treatment of CI.

MATERIAL AND RESEARCH METHODS

The work is based on the analysis of the results of the examination and treatment of 80 patients with cerebellar infarction who were treated at the N.V. Sklifosovsky Institute for Emergency Medicine (n=17), V.M. Buyanov City Clinical Hospital (n=29), S.S. Yudin City Clinical Hospital (n=6), V.V. Veresyaev City Clinical Hospital (n=5), City Clinical Hospital №31 (n=5), City Clinical Hospital № 13 (n=18) Moscow. There were 59 men and 21 women. The age of patients varied from 26 to 87 years. Most patients (n=43, 54%) were of working age (from 26 to 60 years). In all patients, cerebellar infarction occurred for the first time.

Sixty-six patients were hospitalized (82%) within a day after the onset of the disease, 11 patients (14%) were admitted on the 2nd day and 3 patients (4%) were admitted on the 3rd day.

The clinical study on organs and systems and neurological examination were performed in all patients upon admission and in the dynamics. All patients had cerebral symptoms. Focal symptoms were also detected in all patients and were represented by cerebellar disorders, paresis of varying severity and prevalence, speech disorders, bulbar and oculomotor disorders.

The level of consciousness was assessed according to the Glasgow Outcome Scale (GOS). The consciousness upon admission to the hospital was not disturbed in 66 patients (82.5%), 9 patients (11.25%) had moderate and deep obtundation, 3 (3.75%) patients were in sopor, and 2 (2.5%) patients were in coma.

The computed tomography (CT) of the brain was performed upon admission to the hospital and in the dynamics in all patients. In the analysis of tomograms, the volume of the formed focus of ischemia, ventriculo-cranial coefficients (VCC) were determined to assess hydrocephalus. Quantitative assessment of the mass effect in the posterior cranial fossa (PCF) in patients with malignant CI was performed on a scale suggested in 2001 by *M. Jauss et al.* (Table).

Table

The scale of mass effect in patients with malignant cerebellar infarction, developed by M. Jauss et al. (2001)

| Mass-effect, score | Indicators | | |
|--------------------|-----------------------------|--------------------------------------|---|
| | Compression of IV ventricle | Compression of quadrigeminal cistern | The expansion of the lower horn of the lateral ventricle. |
| 0 | No | No | No |
| 1 | Minimal | Minimal unilateral | Minimal |
| 2 | More than 50% | Moderate bilateral | Moderate |
| 3 | Obliteration | Obliteration | Significant |

According to this scale, the mass effect is calculated by the sum of points. The absence or minimum mass effect corresponds to the sum of points from 0 to 3, moderate — 4–6 points, significant — 7 or more (Fig. 1) [19].

The malignant course of cerebellar infarction meant the development of a mass effect in PCF, accompanied by depression of consciousness due to compression of the brain stem and/or the development of occlusive hydrocephalus (according to CT scan data). The benign CI meant the course of the disease not disturbing the consciousness.

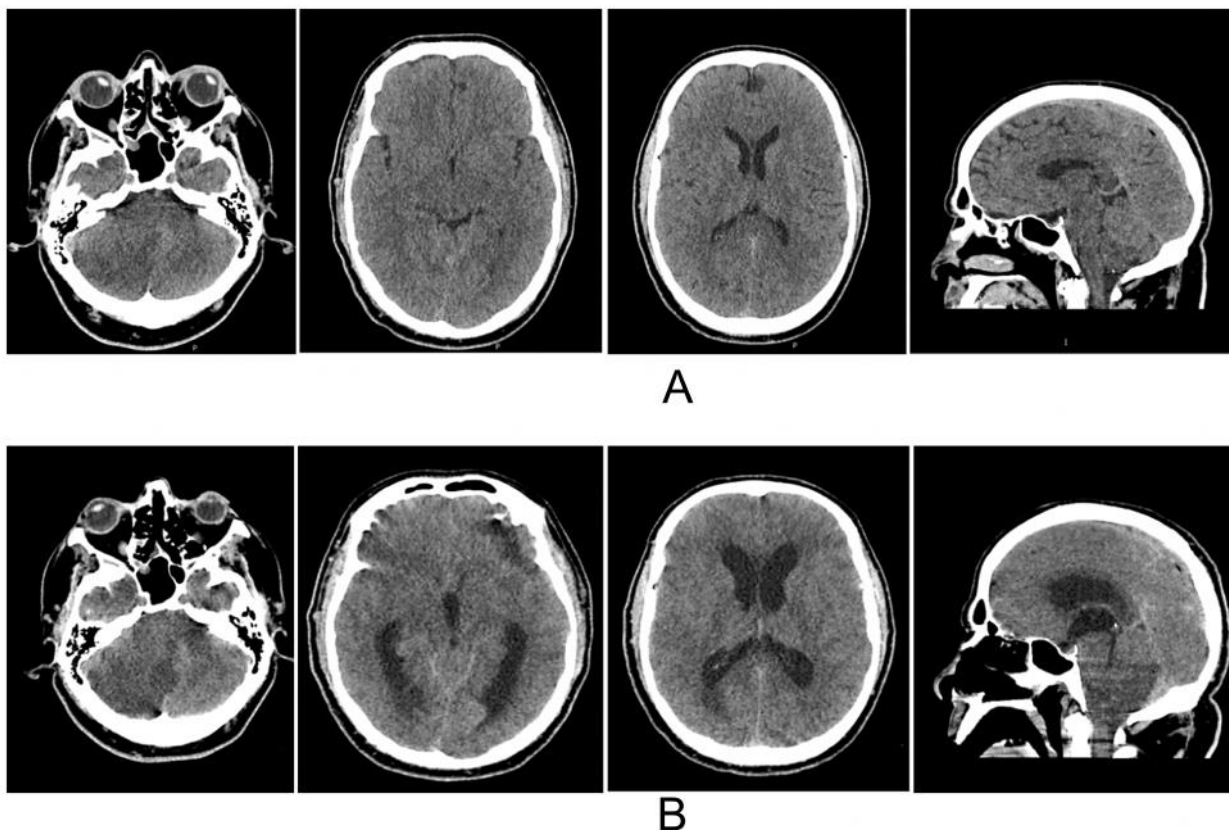


Fig. 1. Computed tomography of the brain of a 46-year-old male patient P. with cerebellar malicioust myocardial infarction. A — 12 hours from the onset of stroke, GOS score 15, ischemia focus 52 cm³, moderate bilateral compression of the quadrigeminous cistern, obliteration of the IV ventricle, VBR2 — 13%, mass effect score 5 according to M. Jauss scale; B — 28 hours from the onset of the disease, GOS score 8, volume of the ischemia focus 98 cm³, obliteration of the quadrigeminous cistern and IV ventricle, significant expansion of the lower horns of the lateral ventricles, VBR2 — 23%, mass effect score 9 according to M. Jauss: significant direct compression of the brain stem on sagittal scans

Patients were divided into two groups according to the clinical course of CI. The group of malignant cerebellar infarction included 55 patients (69%) (Group I), the group of malignant cerebellar infarction included 25 patients (31%) (Group II). Patients of both groups were compared by age, gender, volume of the ischemic focus, degree of mass effect in the PCF according to CT data, course and outcome of the disease. There were no differences in age and gender between the group ($p>0.05$). The groups differed by the level of consciousness upon admission, the volume of ischemia, the intensity of the mass-effect in the PCF according to the *M. Jauss* scale, and outcomes according to GOS ($p<0.05$).

Patients of the Group I were further divided into subgroups in which surgical (surgical subgroup) and only conservative (conservative subgroup) treatment were performed. The surgical subgroup included 37 patients, the conservative group included 18 patients. Depending on the method of surgical intervention, the subgroup of surgical treatment was also divided into three subgroups: IA — patients who underwent only ventriculostomy ($n=16$), IB — patients who underwent only posterior fossa decompression (PFD) ($n=5$), IC — combination of ventriculostomy and PFD ($n=18$). In 2 of 5 patients of the IB subgroup in the early postoperative period, additional external ventricular drainage (EVD) was required, as a result of which IB subgroup extended to 18 patients. Patients with the depression of consciousness from obtundation to deep coma on the background of ICI were operated on the 1st day after the development of ODS.

The criteria for the effectiveness of surgical treatment were recovery of consciousness to a clear and/or recovery of the configuration of the fourth ventricle and the quadrigeminal cistern. Treatment outcomes were assessed according to GOS.

The group of conservative treatment included patients who also had indications for surgery, but at the time of the examination by a neurosurgeon they were either already in decompensated state due to the progression of the ODS, or had severe concomitant pathology in the stage of decompensation, which was a contraindication to surgical treatment.

In a comparative analysis of indicators of gender, the level of consciousness upon admission to the hospital, the volume of the infarction zone before the development of ODS, there was no difference between the surgical and

conservative subgroups ($p>0.05$).

Statistical analysis of the obtained data was performed using the STATISTICA (version 6.0) StatSoft @ Inc., USA. To determine the statistical significance of the difference in signs between patient groups, nonparametric statistics were used (the Mann–Whitney U -test, χ^2 criterion, Wilcoxon T -test, Spearman's rank correlation). The difference was considered significant at the level of significance criterion $p<0.05$.

RESEARCH RESULTS

Patients of groups did not differ in age and male/female ratio ($p>0.05$). The average age of patients in the Group I was 58.72 ± 13.84 , and 61.52 ± 11.69 in the Group II. In the Group I, there were 41 men (75%), 14 women (25%), in the Group II there were 18 men (72%), and 7 women (28%).

Malignant CI developed in 30 (77%) out of 39 patients under 60, and in 25 (61%) out of 41 patients over 60, but there is no reliable dependence of the malignant course of cerebellar infarction on the age of patients ($p>0.05$).

Patients with CI admitted to the hospital on the 1st day of the onset of the disease with severe consciousness disorders and focal symptoms, in most cases had a malignant type of the disease. Of all those admitted on the first day of the onset of the disease, 54 patients (83%) had no disturbances of consciousness. In 35 of them (64%), the consciousness depressed averagely on the 2nd day from the onset of the disease on the background of ODS. In order to study the dynamics of the disease, only 40 patients data were taken into account, who were admitted to hospital on the first day of the onset of cerebellar infarction, with a level of consciousness 15 points, with confirmed ischemia (according to CT). The malignant course of the disease subsequently developed in 21 of them (52.5%).

The median volume of ischemic focus when admitted to hospital in the group of patients with malignant cerebellar infarction was higher than in patients with a benign course of cerebellar infarction – 29 cm^3 and 12 cm^3 , respectively ($p<0.05$) (Fig. 2).

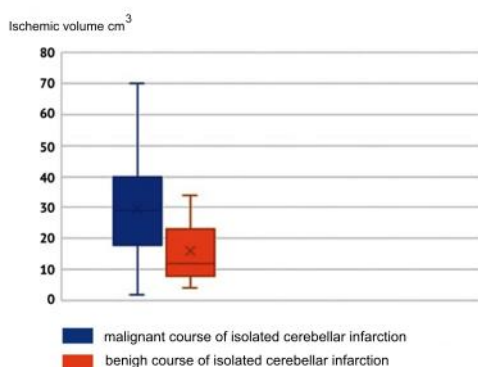


Fig. 2. The dependence of the risk of malicious cerebellar infarction on the volume of ischemia ($n=40$, $p<0.05$)

All patients admitted on the 1st day of the disease with CT confirmed cerebellar ischemia were divided according to the volume of the ischemia focus less than 20 cm^3 and more than 20 cm^3 . In 5 patients (31%) with cerebellar ischemia less than 20 cm^3 out of 16, the development of malignant cerebellar infarction was observed. Of 24 patients with ischemia more than 20 cm^3 , the malignant course was observed in 16 patients (67%) ($p<0.05$).

The dependence of the incidence of malignant cerebellar infarction on the degree of mass effect on admission (Fig. 3) was revealed. On the first day of the disease, the mass effect ranged from 1 to 6 points according to the *M. Jauss* scale. The average magnitude of the mass effect was 3.37 ± 1.92 points in the group of malignant cerebellar infarction and 1.72 ± 0.64 points in the group of benign course.

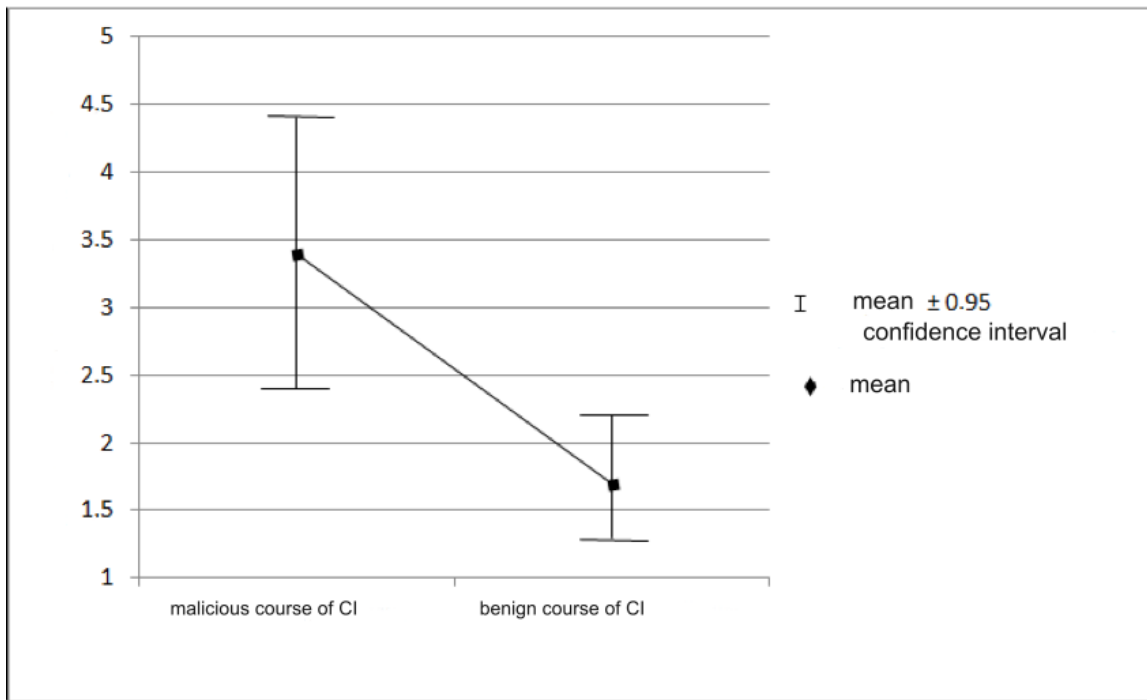


Fig. 3. The risk of malicious cerebellar infarction depending on the degree of mass effect in the PCF on the first day of the disease ($p < 0.05$)

A comparative analysis of indicators of gender, level of consciousness upon admission to hospital, the volume of infarction area and VCC2 did not reveal significant differences between the surgical and conservative treatment groups for malignant cerebellar infarction ($p > 0.05$).

The level of consciousness before the operation in all subgroups of the operated patients averaged 9.9 ± 2.8 points according to GOS. The average volume of ischemia prior to surgery was $63.2 \pm 19 \text{ cm}^3$, the mass effect on the *M. Jauss* scale was 7.3 ± 1.2 points.

When comparing the outcomes of surgical and conservative treatment, it was found that there were 29.5% more good outcomes and cases of moderate disability in patients of the surgical group than in the group of conservative treatment. The mortality rate in the operated patients was a quarter lower and amounted to 48.6% versus 77.8% in patients of the conservative group ($p < 0.05$) (Fig. 4).

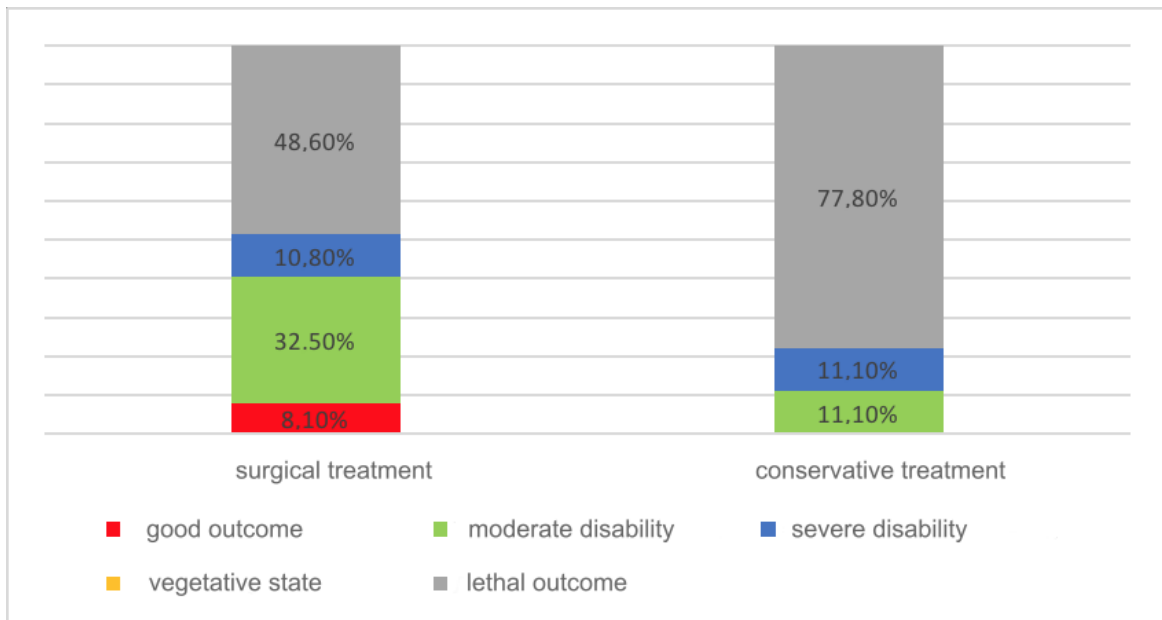


Fig. 4. Results of surgical and conservative treatment in patients with malicious CI ($n=55$, $p < 0.05$)

A positive trend after surgery among patients who underwent only ventriculostomy was observed in 7 patients out of 16, and the complete recovery of consciousness occurred averagely on the 12th day. The recovery of the configuration of basal cisterns and fourth ventricle was noted averagely on the 11th day after the operation. The postoperative mortality in patients who underwent ventriculostomy was 56% (9 patients). Four patients had mild disability upon discharge, and 3 patients had severe disability (Fig. 5).

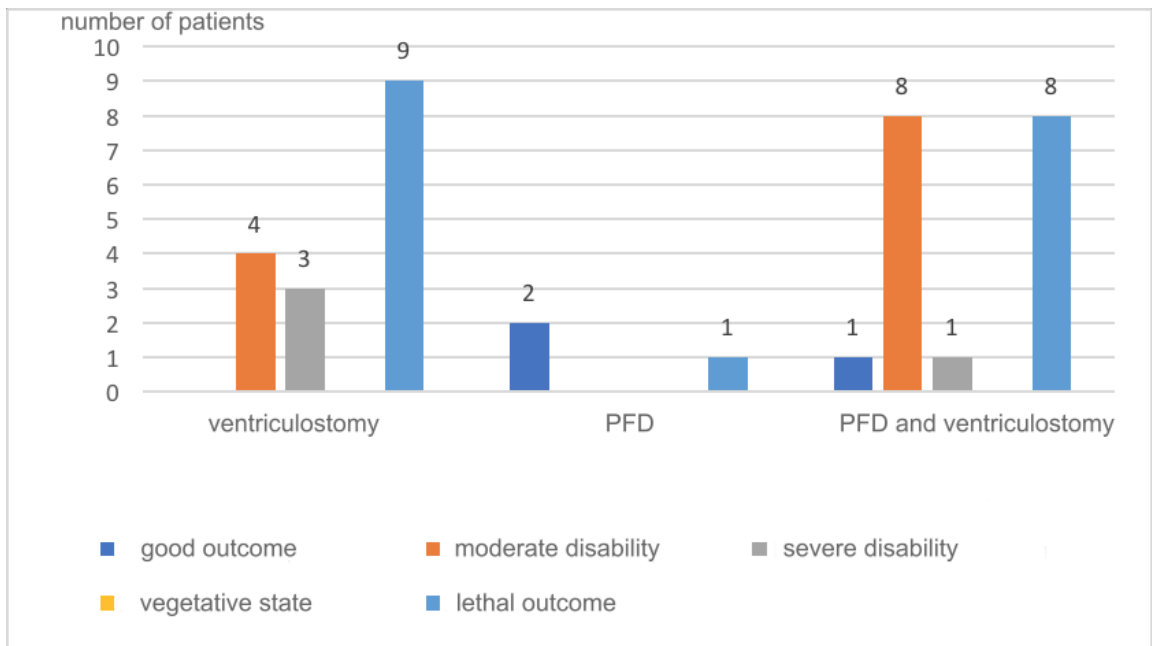


Fig. 5. Results of surgical treatment of patients depending on the method of intervention (n=37)
 Note: PFD — posterior fossa decompression

Of 5 patients who underwent PFD only one patient died due to the progression of the ODS, two patients were discharged with good recovery (Fig. 5). In 2 patients in the postoperative period, AOH and coma developed, so EVD followed PFD.

In 16 patients, ventriculostomy and PFD were performed simultaneously. The positive dynamics was observed in 14 patients (including those 2 patients who had an additional ventriculostomy due to the failure of PFD), the complete recovery of consciousness after the surgery was noted averagely on day 4, of which 6 patients already had the recovery of the quadrigeminal cistern configuration on the 1st day after surgery. The postoperative mortality among 18 patients who underwent PFD in combination with ventriculostomy was 50%. Upon the discharge, one patient had a good outcome, 8 (50%) patients had moderate disability, and one patient had a severe disability (Fig. 5).

All patients who died were in the group of malignant cerebellar infarction (40%; n=32). The absence of regression of ODS in the surgical group was the cause of death in 50% of patients (n=9). The causes of death in 5 patients (27.8%) were purulent-septic complications, myocardial infarction in 3 patients (16.6%), and gastrointestinal bleeding in one patient (5.6%). In the group of conservative treatment, the cause of death was ODS in 12 patients (85.8%), and purulent-septic complications in 2 (14.2%). In patients with malignant CI surgical treatment reduced the mortality rate of ODS by 35.8% ($p < 0.05$) (Fig. 6).

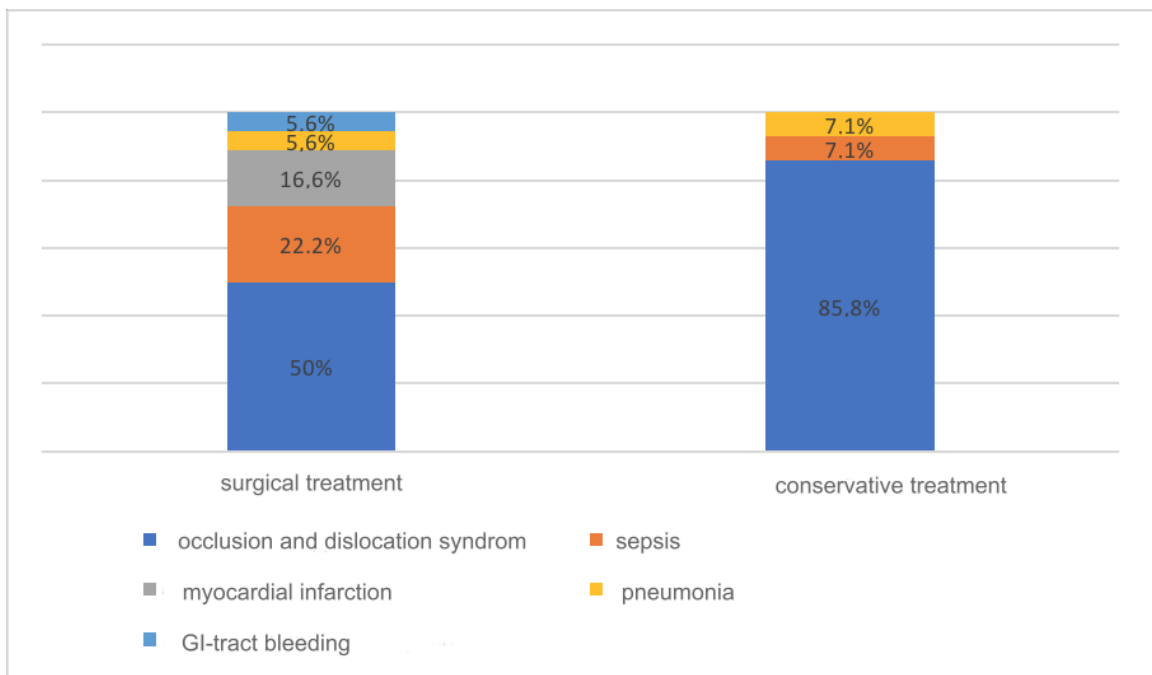


Fig. 6. Causes of death in the surgical and conservative treatment groups (n = 32, $p < 0.05$)

The efficacy of surgical treatment among patients who underwent ventriculostomy and PFD, was higher by 34% ($p < 0.05$) compared with isolated ventriculostomy and by 38% ($p > 0.05$) compared to patients with isolated PFD (Fig.

7).

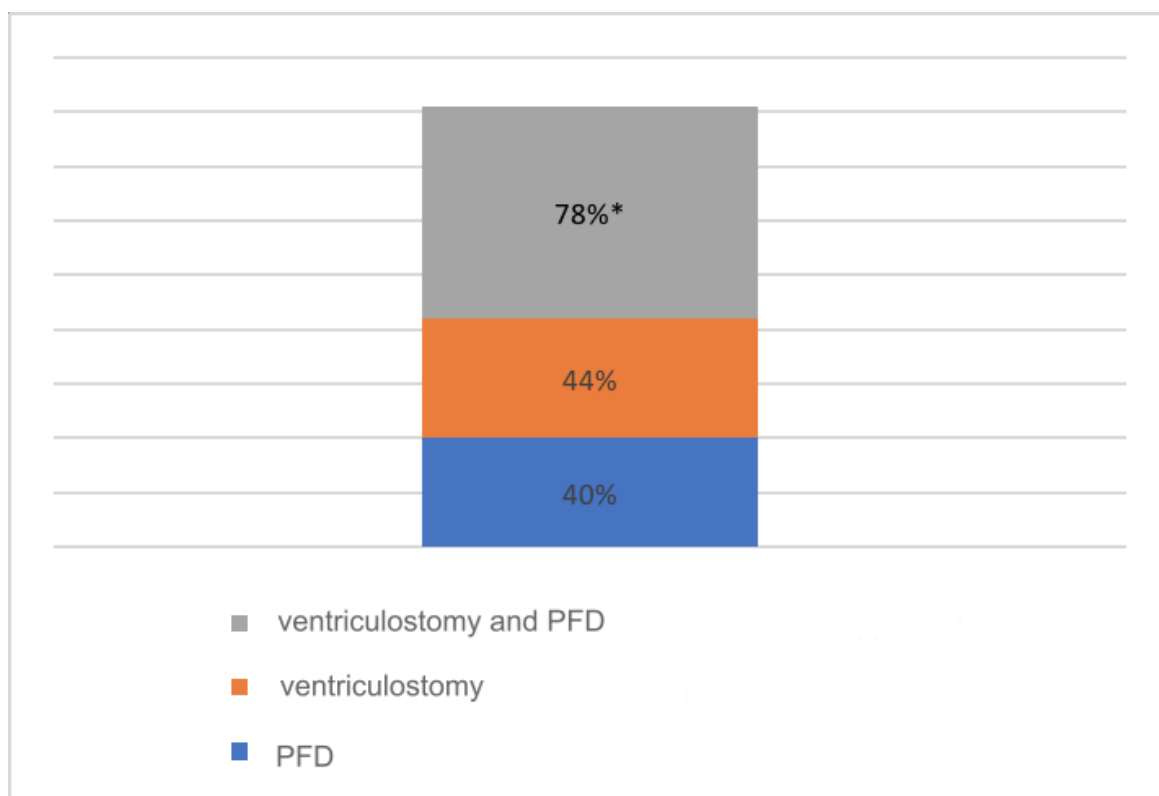


Fig. 7. The efficacy of methods for surgical treatment of CI (n=37) (* — $p < 0.05$ compared with the values of ventriculostomy operations subgroup)

DISCUSSION

Cerebellar infarctions, which have a tendency to cause mass effect, are often detected already with the development of ODS [20]. In our study, a malignant course occurred more frequently in patients with ischemia greater than 20 cm^3 ($p < 0.05$) on the 1st day of the disease. When examining 90 patients with CI, *G. Koh et al.* showed that the average volume of infarction leading to the development of AOH and brain stem compression is 24 cm^3 [21].

In our study, it was noted that the presence of a mass effect in the PCF on the first day of cerebellar infarction did not always lead to a disturbance of the patient's consciousness. The threshold value of dislocation, which can later cause malignant cerebellar infarction on the first day of the disease, was 3 points on the *M. Jauss* scale.

H. Neugebauer et al. (2013) in the analysis of works on the treatment of malignant myocardial infarction showed that with conservative treatment, the overall mortality rate was 42.9%, and in patients with coma it was 85%. Survival rates in surgical treatment ranged from 76.8 to 81.6% [20].

Some authors consider following factors as indications for the surgical treatment of CI:

- deep obtundation (GOS 13) and below or the depression of consciousness by 2–3 or more points during the observation period (averagely 2–3 days);
- compression of the fourth ventricle;
- cerebellar-tentorial dislocation and/or disappearance of subarachnoid cisterns of PCF;
- the appearance (according to CT data) of a mass effect of 4–5 points or more according to the *M. Jauss* scale [12–14, 19, 22–24].

The followers of ventriculostomy consider it sufficient to perform only the EVD or endoscopic perforation of the rhomboid fossa, explaining their choice by the lesser trauma and the opportunity to achieve good results without performing PFD [25]. Opinions about the safety of drainage of cerebrospinal fluid (CSF) under the conditions of mass-effect in SCF are different. A number of authors are opposed to place EVD due to an increased risk of cerebellar-tentorial herniation [26–28].

Some authors believe that after performing ventriculostomy, the dynamic observation is necessary, and if dislocation grows, then PFD is performed [14, 15, 29]. So, *A. Raco et al.* (2003) suggest that, only EVD should be performed in AOH with a subsequent assessment of the neurological status and PFD id performed only in the absence of positive dynamics. However, in the presence of signs of compression of PCF cisterns and the brain stem, the authors immediately performed PFD. With such a differentiated approach to the treatment of 17 patients with malignant CI, they achieved good and satisfactory results in 86.4% of cases and a postoperative mortality rate of 13.6% [14].

One of the first works demonstrating the advantages of primary PFD is an article of *L.M. Auer et al.* (1986). Of the 13 patients with massive CI, the conservative treatment performed in 5 of them, and 4 patients died. One patient was treated with EVD, but the patient also died in the postoperative period. PFD was performed in 7 patients, and all patients survived. The authors concluded that patients with progressive depression of consciousness should immediately undergo surgical treatment. The purpose of surgery is to eliminate the mass effect and the compression of the brain stem by PFD, as well as the management of AOH [15].

The work of *E. Juttler et al.* may confirm the effectiveness of the combined surgical tactics (2009). EVD was performed in patients in a more compensated condition, with a level of consciousness prior to surgery from 6 to 14

points (averagely 14 points) and a significant mass effect 4–6 points according to the scale of *M. Jauss*. PFD was performed in patients with a level of consciousness 3–15 points (averagely 12 points) and a mass effect of 4–9 points according to the *M. Jauss* scale. In the 2nd group (the group of more severe patients who underwent PFD + EVD), the mortality rate was lower, the postoperative period of hospital stay was shorter and ventriculoperitoneal bypass implantation was required 2 times less [22].

In our opinion, combined interventions are most effective. In patients with a progressive course of the disease, it reduces the time until elimination of dislocation and occlusion, which is spent during step-by-step operations, and are accompanied by better postoperative results.

CONCLUSION

Patients with cerebellar infarction with a volume of more than 20 cm³, accompanied by a mass effect in the posterior fossa of 3 or more points according to the *M. Jauss* scale, are prone to the development of a malignant course of the disease. With the development of the clinical picture of the occlusive dislocation syndrome, the surgical treatment is indicated.

In the surgical treatment of malignant cerebellar infarction, it is advisable to perform decompressive trepanation of the posterior cranial fossa together with ventriculostomy, since each separate operation does not exclude further progression of the occlusive dislocation syndrome.

REFERENCES

1. Levada O.A. Etiology, clinical variants, diagnosis and treatment tactic of cerebellar infarction. *Lik Sprava*. 2003; (5–6): 3–6. PMID: 14618791
2. Milandre L., Brosset C., Gouirand R., Khalil R. Pure cerebellar infarction. Thirty cases. *Presse Med*. 1992; 21(33): 1562–1565. PMID: 1470612
3. Tohgi H., Takahashi S., Chiba K., Hirata Y. Cerebellar infarction. Clinical and neuroimaging analysis in 293 patients. The Tohoku Cerebellar Infarction Study Group. *Stroke*. 1993; 24(11): 1697–1701. PMID: 8236346. DOI: 10.1161/01.str.24.11.1697.
4. Macdonell R., Kalnins R., Donnan G. Cerebellar infarction: natural history, prognosis, and pathology. *Stroke*. 1987; 18(5): 849–855. PMID: 3629642. DOI: 10.1161/01.str.18.5.849.
5. Mohr J. *Stroke: Pathophysiology, Diagnosis, and Management*. 5th ed. Philadelphia: Elsevier, 2011: 750–752.
6. Greenberg J., Skubick D., Shenkin H. Acute hydrocephalus in cerebellar infarct and hemorrhage. *Neurology*. 1979; 29(3): 409–413. PMID: 571991. DOI: 10.1212/wnl.29.3.409.
7. Klugkist H., McCarthy J. Surgical treatment of space-occupying cerebellar infarctions — 4 ½ years post-operative follow-up. *Neurosurg Rev*. 1991; 14(1): 17–22. PMID: 2030823.
8. Andoh T., Sakai N., Yamada H., et al. Cerebellar infarction: analysis of 33 cases. *No Shinkei Geka*. 1990; 18(9): 821–828. PMID: 2234303.
9. Sypert G., Alvord E. Cerebellar infarction. A clinicopathological study. *Arch Neurol*. 1975; 32(6): 357–363. PMID: 1131069. DOI: 10.1001/archneur.1975.00490480023001.
10. Hornig C., Rust D., Busse O., et al. Space-occupying cerebellar infarction. Clinical course and prognosis. *Stroke*. 1994; 25(2): 372–374. PMID: 8303748. DOI: 10.1161/01.str.25.2.372.
11. Simard J., Chen M., Tarasov K.V., et al. Newly expressed SUR1-regulated NC (Ca-ATP) channel mediates cerebral edema after ischemic stroke. *Nat Med*. 2006; 12(4): 433–440. PMID: 16550187. PMCID: PMC2740734. DOI: 10.1038/nm1390.
12. Jauss M., Krieger D., Hornig C., et al. Surgical and medical management of patients with massive cerebellar infarctions: results of the German-Austrian Cerebellar Infarction Study. *J Neurol*. 1999; 246(4): 257–64. PMID: 1036769. DOI: 10.1007/s004150050344.
13. Tsitsopoulos P.P., Tobieson L., Enblad P., Marklund N. Surgical treatment of patients with unilateral cerebellar infarcts: clinical outcome and prognostic factors. *Acta Neurochir (Wien)*. 2011; 153(10): 2075–2083. PMID: 21833781. DOI: 10.1007/s00701-011-1120-4.
14. Raco A., Caroli E., Isidori A., Salvati M. Management of acute cerebellar infarction: one institution's experience. *Neurosurgery*. 2003; 53(5): 1061–1065. PMID: 14580272. DOI: 10.1227/01.neu.0000088766.34559.3e.
15. Auer L.M., Auer T., Sayama I. Indications for surgical treatment of cerebellar hemorrhage and infarction. *Acta Neurochir (Wien)*. 1986; 79(2–4): 74–79. PMID: 3962746. DOI: 10.1007/bf01407448.
16. Krylov V.V., Nikitin A.S., Dash'yan V.G., et al. *Surgery of a massive ischemic stroke*. Moscow: GEOTAR–Media Publ., 2016. 136 p. (In Russian).
17. Godkov I.M., Luk'yanchikov V.A. The surgical treatment of patients with cerebellar ischemic stroke. *Neyrokhirurgiya – Russian Journal of Neurosurgery*. 2015; (3): 84–90. (In Russian).
18. Asratian S.A., Nikitin A.S. Dislocation syndrome induced by massive cerebellar infarction and indications for urgent operation. *Zhurnal neurologii i psikiatrii im SS Korsakova*. 2012; 112(12) Pt2: 55–60. (In Russian).
19. Jauss M., Muffelmann B., Krieger D., et al. A computed tomography score for assessment of mass-effect in space-occupying cerebellar infarction. *J Neuroimaging*. 2001; 11(3): 268–271. PMID: 11462293. DOI: 10.1111/j.1552-6569.2001.tb00045.x.
20. Neugebauer H., Witsch J., Zweckberger K., Juttler E. Space occupying cerebellar infarction: complications, treatment, and outcome. *Neurosurg Focus*. 2013; 34(5): E8. PMID: 23634927. DOI: 10.3171/2013.2.focus.12363.
21. Koh M., Phan T., Atkinson J., Wijedicks E. Neuroimaging in deteriorating patients with cerebellar infarcts and mass effect. *Stroke*. 2000; 31(9): 2062–2067. PMID: 10978030. DOI: 10.1161/01.str.31.9.2062.
22. Juttler E., Schweickert S., Ringleb P.A., et al. Long-term outcome after surgical treatment for space-occupying cerebellar infarction: experience in 56 patients. *Stroke*. 2009; 40(9): 3060–3066. PMID: 19574554. DOI: 10.1161/strokeaha.109.550913.
23. Pfefferkorn T., Epinger U., Linn J., et al. Long-term outcome after suboccipital decompressive craniectomy for malignant cerebellar infarction. *Stroke*. 2009; 40(9): 3045–3050. PMID: 19574555. DOI: 10.1161/strokeaha.109.550871.
24. Tsitsopoulos P.P., Tobieson L., Enblad P., Marklund N. Clinical outcome following surgical treatment for bilateral cerebellar infarction. *Acta Neurol Scand*. 2011; 123(5): 345–351. PMID: 20636449. DOI: 10.1111/j.1600-0404.2010.01404.x.
25. Baldauf J., Oertel J., Gaab M.R., Schroeder H.W. Endoscopic third ventriculostomy for occlusive hydrocephalus caused by cerebellar infarction. *Neurosurgery*. 2006; 59 (3): 539–544. PMID: 16955035. DOI: 10.1227/01.neu.0000228681.45125.e9.
26. Kase C., Norrving B., Levine S., et al. Cerebellar infarction. Clinical and anatomic observation in 66 cases. *Stroke*. 1993; 24(1): 76–83. PMID: 8418555. DOI: 10.1161/01.str.24.1.76.
27. Chen H.J., Lee T.C., Wei C.P. Treatment of cerebellar infarction by decompressive suboccipital craniectomy. *Stroke*. 1992; 23(7): 957–961. PMID: 1615544. DOI: 10.1161/01.str.23.7.957.
28. Heros R.C. Surgical treatment of cerebellar infarction. *Stroke*. 1993; 23(7): 937–938. PMID: 1615540. DOI: 10.1161/01.str.23.7.937.
29. Khan M., Polyzoidis K.S., Adegbite A.B., McQueen J.D. Massive cerebellar infarction: "conservative" management. *Stroke*. 1983; 14(5): 745–751. PMID: 6658959. DOI: 10.1161/01.str.14.5.745

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