ISSN 1220-8841 (Print)
ISSN 2344-4959 (Online)

ROMANIAN
NEUROSURGERY

Vol. XXXIX | No. 3 September 2025

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DOI: 10.33962/roneuro-2025-041

Romanian Neurosurgery (2025) XXXIX (3): pp. 223-227
DOI: 10.33962/roneuro-2025-041
www.journals.lapub.co.uk/index.php/roneurosurgery

Malignant cerebellar infarction with neurological and systemic complications

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ABSTRACT

We presented the successful management of a dramatic case of one patient with malignant cerebellar infarction with superior (culmen) vermian herniation and inferior (tonsillar) herniation, ventriculitis, cerebral venous thrombosis, decubitus acute pneumopathy, septic pulmonary venous thrombosis, ethanolic cytolysis of the liver, diabetus mellitus.

SUMMARY

64-year-old patient admitted to our department to my assistance 5 days after the onset of the condition, at the request of his family due to clinical and neurological deterioration being treated by another neurosurgeon by conservative treatment. Condition on admission significantly worsened compared to the moment of onset of the condition: at the onset of the condition: drowsiness (GCS 13), dysarthric speech, repeated vomiting, inability to make fine movements of the right hand, vertigo. On admission to my assistance: stupor (GCS 9), scor Rankin modified 3, Index Barthel 0, walking impossible, bilateral Babinsky sign, mainly on the right, horizontal nystagmus to the right, mucopurulent expectoration, SPO2 82% and basal gasping and wheezing rales. Cranial CT scan revealed malignant cerebellar infarction with acute internal hidrocephallus, ventriculitis, superior (culmen) vermian herniation and inferior (tonsillar) herniation. The patient was emergently operated (EVD, SDC), treatment of ventriculitis, decompression of superior and inferior cerebellar herniations, coexistent treatment of decubitus acute pneumopathy, treatment of septic pulmonnary venous thrombosis, and cerebral venous thrombosis, treatment of ethanolic cytolysis of the liver and diabetus mellitus. Evolution was very good with progressive disappearance of intracranial hypertension syndrome, neocerebellar syndrome and diminishing of vermian syndrome. Follow up 12 months.

Keywords
bilateral PICA infarction, acute internal ventriculitis, cerebral thrombosis, ischemic stroke, cerebellar infarction decompressive surgery

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ISSN online 2344-4959
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First published
September 2025 by
London Academic Publishing
www.lapub.co.uk

Jauss and colleagues[10] published a CT radiological scale of severity in posterior fossa ischemia from 0-9 points based on: 1) compression of the IV ventricle, 2) compression of the quadrigeminal cisterns, 3) dilatation of the inferior portion of the lateral ventricle.

1. Compression of the 4th ventricle (0 = no compression, 1 = unilateral compression, 2 = shifted midline, 3 = not visible)
2. Compression of the quadrigeminal cistern (0 = no compression,1 = mild with asymmetric compression ipsilateral to the infarc-tion, 2 = moderate with evidence of bilateral compression, 3 =severe bilateral compression with obscured quadrigeminal cistern)
3. Dilatation of the inferior horn of the lateral ventricle (0 = no dilatation, 1 = mild, 2 = moderate, 3 = severe)

After all CT scores, ischemia of posterior fossa was appreciated as follows[10]:
– 0–3: no or slight mass effect
– 4–6: moderate mass effect
– 7–9: severe mass effect

After Jauss radiological scale[10] the patient presented has: 1—3, 2—2, 3—2
Total of the radiologic scor of the patient is 7 points—severe mass efect!

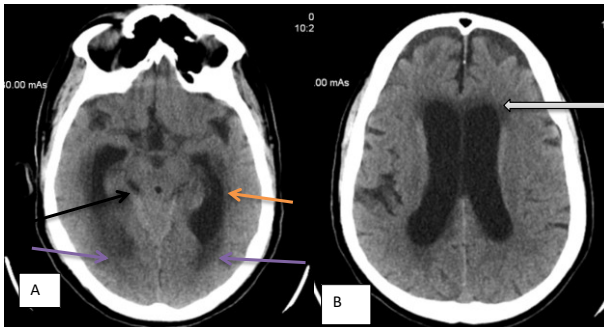

Figure 1. CT Scan cranian contrast: A. Superior vermian (Culmen) herniation, predominantly on the right (Black arrow-reduction of the quadrigeminal cistern). Acute internal hydrocephalus. (Orange arrow-increase in size of the ventricular temporal horns). Ventriculitis (mauve arrow accumulation of purulent secretions in the distal ventricular portion) B. Acute internal hydrocephalus Periventricular hypodensity (white arrow)

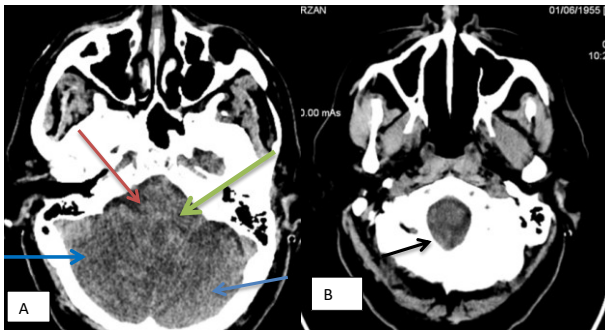

Figure 2. Cranial CT scan A. Hypodensity in the PICA territory of the right cerebellar hemisphere and left cerebellar hemisphere, left vertebral artery ischemia-blue arrow. Hypodensity in the vermian territory-green arrow. Completely compressed fourth ventricle (red arrow) B. Cerebellar tonsillar hernia, predominantly right-black arrow

Case report

A 64-year-old patient admitted to our department to my assistance 5 days after the onset of the condition, at the insistence of his family due to clinical and neurological deterioration being treated by another neurosurgeon by conservative treatment. Condition on admission significantly worsened compared to the moment of onset of the condition: at the onset of the condition: drowsiness (GCS 13), dysarthric speech, repeated vomiting, inability to make fine movements of the right hand, vertigo. On admission to my assistance: stupor (GCS 9), scor Rankin modified 3, Index Barthel 0, walking impossible, bilateral Babinsky sign, predominantly on the right, nystagmus on the right, mucopurulent expectoration, SPO2 82% and basal gasping and wheezing rales. Cranial CT scan revealed malignant cerebellar infarction with acute internal hydrocephalus, ventriculitis, superior (culmen) vermian herniation and inferior (tonsillar) herniation.

NB Due to the very serious clinical and neurological condition, I did not have the time necessary to obtain a preoperative brain MRI.

Preoperatively, we urgently treated acute pneumonia (antibiotics, mucolytics, intravenous and aerosol bronchodilators, oxygen on a mask, aspiration of purulent secretions for 10 hours), treated preoperative intracranial hypertension (Mannitol 1 g/kgb per day) and administered platelet mass (1 unit per 10 kg body weight) (the patient was treated with 2 platelet antiagregants - Aspenter and Clopidogrel). We then performed neurosurgical intervention - EVD, SDC, predominantly on the right,

aspiration of ischemic tissue (necrosectomy), resection of posterior arch of C1.

SDC is a safe procedure that is indicated if the initial medical treatment has not been favorable or the patient's condition deteriorates rapidly. The mortality of SDC is between 29.5% and has an 83% chance of not causing mild disability The purpose of this is to provide space for the edematous cerebellum, relieving compression of the fourth ventricle and the brainstem, surgical evacuation of infarcted tissue (necrosectomy).

Functional outcomes were assessed using the Glasgow outcome scale, modified Rankin scale, and Barthel index at discharge and 2 months postoperatively.

In the evolution, the neurological condition improved significantly, the patient became conscious postoperatively, slightly drowsy GCS 14. During hospitalization, the treatment of meningitis with Klebsiella that occurred in the context of immunosuppression (lymphopenia, type 2 diabetes mellitus, chronic alcoholism with hepatic cytolysis) was performed with antibiotics (Meropenem 8 g /day) administered intravenous and intraventricular External ventricular drainage was performed, subsequently lumbar subdural drainage - CSF collection, antibiotic treatment, Diabetes mellitus and ethanolic hepatic cytolysis were also treated with vitamins B1, B6, Essentiale 600, 3 Cp/day, Silymarin I cp of 1000 mg/day. Cerebrolysin 30 mg/day iv was administered.

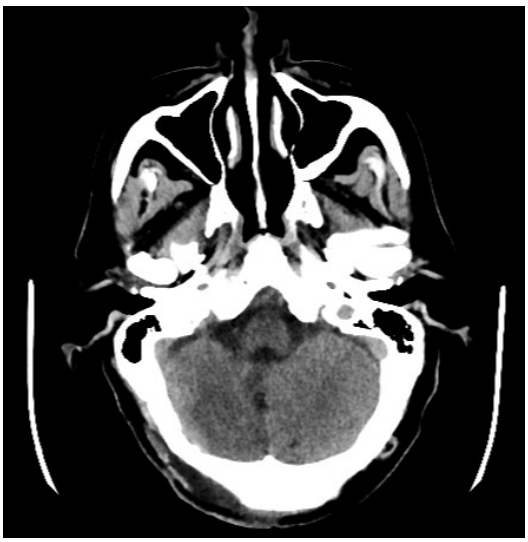

Figure 3. Native axial cranial CT scan. Postoperative condition. Decompressed IV ventricle (red arrow)

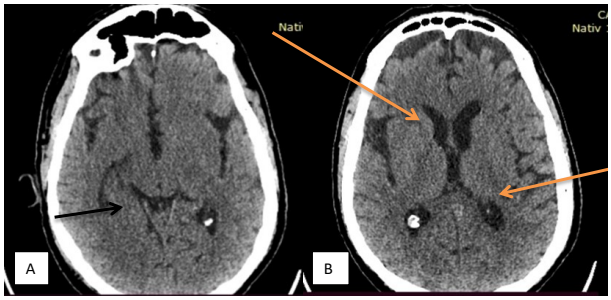

Figure 4. A. Postoperative axial CT scan. Normalization of the quadrigeminal cistern (disappearance of the culmen hernia) - black arrow. B. Normalization in ventricular dimensions- Orange arrow.

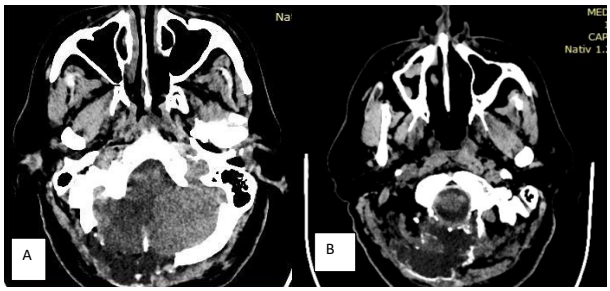

Figure 5. A.Postoperative CT Scan. Infratentorial Occipital craniectomy. Persistence of right cerrebellar hemisphere and vermian hypodensity. . B. Disappearance of tonsillar herniation.

Due to decreased immunity (lymphopenia, chronic alcoholism, diabetes mellitus, ventriculitis), skin necrosis occurred, which required wound re-suturing. Wound healing was delayed.

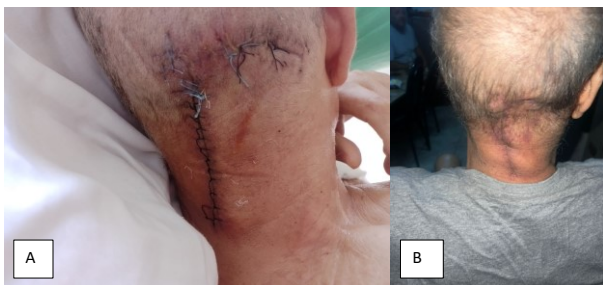

Figure 6. A. Intrahospital aspect of the wound, surgical, in the process of healing. B 12 months postoperatively.

The hospitalization was significantly prolonged due to the therapeutic effort and nursing necessary to cure the ventriculitis, cerebral venous sinus thrombosys and pulmonary venous thrombosys, acute pneumopathy, ethanolic hepatic cytolysis and

the treatment of diabetes mellitus, finally reaching 2 months.

State upon discharge: moderate vermian syndrome, with difficulty walking and a wide base of support, but improved compared to the preoperative status. Rankin modified index 3, Barthel index 50/100, GCS 13

Postoperative followup: 12 months: Patient walk without a frame, with slight intermittent imbalance. Dysarthria disappeared, GCS 15, GAS 5, Indice Barthel 100/100, Scar Rankin Modified 1

Sensitive disorders persist such as intermittent right brachial and left crural hypoesthesia, significantly improved in intensity compared to the preoperative moment, GCS 15, GAS 5, Indice Barthel 100/100, Scar Rankin Modified 1. Sensitive disorders remained, such as intermittent right brachial and left crural hypoesthesia, significantly improved in intensity compared to the preoperative moment.

Discussion

The most common etiology of CI is atheroscleroyis of the great arteries, followed by cardio embolism, whereas injury to the vertebral arteries, such as dissections, is the most common traumatic cause of cerebellar infarction[1,2].

Cerebellar infarction is an infrequent pathology that represents 3% of ischemic strokes with associated high morbidity and mortality. This pathology is mainly caused by the occlusion of the vascular flow to 3 main arteries of the vertebrobasilar system: the posterior inferior cerebellar artery (PICA-which is usually more frequently associated with this pathology), the anterior inferior cerebellar artery (AICA) and superior cerebellar artery (SCA)[4,5,6]

Large infarcts of the cerebellar hemispheres, particularly those affecting the posterior inferior cerebellar artery and infarcts affecting multiple arterial territories may be accompanied by the development of brain swelling due to cytotoxic and vasogenic edema [10]

Timely escalation of treatment is crucial and should be guided by clinical and neuroradiological rationales. Patients in a coma after hydrocephalus and/or local brainstem compression may also benefit from more aggressive surgical treatment[10]. In case of extensive mass effect, conservative treatment strategies are not well validated and are often unsuccessful inclinical practice[10]

Surgical treatment is associated with much higher survival rates: 81.6% for patients treated with EVD alone,76.8% for those treated with SDC alone, and 77.5% forthose treated with both EVD and SDC[10]. Decompressive surgery is a potentially life-saving treatment in patients with malignant space-occupying cerebellar infarction[3]. The 90-day mortality rate was 27.6%[3]. A good functional outcome 90 days after surgery was achieved in 41.4% .

It has been estimated that without surgery, about 80% of patients who have developed signs of brain stem compression will die, usually within hours to a few days standard[7]

Despite advances in medical therapy for stroke, it has been estimated that 20% of patients with massive cerebellar infarction[11-14]deteriorate clinically as a result of mass effect of the infarct volume. This process may result in morbidity and mortality as a result of either brainstem compression or obstruction of the fourth ventricle, causing acute hydrocephalus and tonsillar herniation.

In the operated patient, we had to aggressively treat acute internal hydrocephalus and ventriculitis by external ventricular drainage. This was also useful for intraventricular administration of antibiotics. Also, the direct approach to massive cerebellar ischemia (of the ipsilateral cerebellar hemisphere, predominantly on the right and vermian (infratentorial occipital craniectomy, cerebellar parenchymal aspiration, posterior C1 laminectomy (for bulbar decompression caused by cerebellar tonsillar hernia)) was indispensable.

Conclusion

- Decompressive surgery is a safe, life-saving treatment for malignant space-occupying cerebellar infarction.
- The best results are when neurosurgical interventions are performed with great emergency to reduce or avoid the risks and vital complications of malignant cerebellar ischemia.
- The concomitant emergent treatment of all the patient's pathologic conditions and complications (Ventriculitis, meningitis, acute internal hydrocephalus, culmen and tonsillar herniations, acute decubitus pneumonia, ethanolic hepatic cytolysis, type 2 diabetes mellitus, cerebral and pulmonary venous thromboses) was mandatory

for saving the patient's life and recovering back to an acceptable quality of life.

- The residual sequela (vermian syndrome) is under treatment, with progressive improvement.

Credit authorship contribution statement

Balasa Daniel: Main Senior Neurosurgeon, Investigation, Visualization, Surgical treatment, Neuroreabiltation strategy expert, Software, Supervision, Validation, Writing – original draft , Writing – review & editing,. Voicu Ionel: Second Senior Neurosurgeon. Surgical treatment. Scupra Tase Costel: Resident Neurosurgeon: Validation, Writing – review & editing, Tunas Alexandru : Senior Neurosurgeon, Expert in Neuroreabilitation therapeutic strategies, Methodology , Writing – review & editing Supervision, Candea Iulia –Senior anestesiologist. Chief of anestesiology Team, Barbulescu Constantin: Senior anestesiologist , ICU expert.

Acknowledgements

The authors declare no conflict of interest. We thank all medical staff involved in the treatment of stroke patients at departments of Neurosurgery and ICU of our hospital

References

1. J.T. Kim, M.S. Park, K.H. Choi, Kim N.K.Han, T.H. Park. Clinical outcomes of posterior versus anterior circulation infarctus with low National Institutes of Health Stroke scale scores, Stroke 48 (1) (2017) 55-62
2. D.E. Searliz, L. Pazdera, E Korbel, Caplan L.R., Vysata. Symptoms and signs of posterior circulation ischemia in new England Medical Center posterior circulation registry, Arch Neurol. 69 (2012) 346-351
3. Baki E., Baumgart L. , Kehl V. ,Hess F. Functional Outcomes After Decompressive Surgery in Patients with Malignant Space-Occupying Cerebellar Infarction
4. Edlow JA, Newman-Toker DE, Savitz SI. Diagnosis and initial management of cerebellar infarction. Lancet Neurol 2008;7:951–964
5. Nayar VV, Day AL. Surgical management of cerebellar stroke—hemorrhage and infarction. Schmidek and Sweet Operative Neurosurgical Techniques. Elsevier; 2012. p. 837–844.
6. Heiferman DM, Loftus CM. Management of cerebellar hematomas and infarcts.
7. Primer on Cerebrovascular Diseases. Elsevier; 2017. p. 799–804.
8. Heros RC: Cerebellar hemorrhage and infarction. Stroke 1982;13: 106-109
9. Kim T.W J Korean Soc Geriatr Neurosurg 2021;17(2):31-37
10. Jauss M, Krieger D, Hornig C, Schramm J, Busse O. Surgical and medical management of patients with massive cerebellar infarc- tions: results of the German-Austrian Cerebellar Infarction Study. J Neurol 1999;246:257–64
11. Wijdicks EFM, Sheth KN, Carter BS, Greer DM, Kasner SE, Kimberly WT, et al. Recommendations for the Management of Cerebral and Cerebellar Infarction With Swelling: A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke. 2014;45:1222-1238.

12 .Kase CS, Norrving B, Levine SR, Babikian VL, Chodosh EH, Wolf PA, et al. Cerebellar infarction. Clinical and anatomic observations in 66 cases. Stroke. 1993;24:76-83.

13 Koh MG, Phan TG, Atkinson JLD, Wijdicks EFM. Neuroimaging in deteriorating patients with cerebellar infarcts and mass effect. Stroke. 2000;31: 2062-2067.

14 Macdonell RALM, Kalnins RMM, Donnan GAM. Cerebellar infarction: natural history, prognosis, and pathology. Stroke. 1987;18:849-855.