The Egyptian Journal of Hospital Medicine (April 2022) Vol. 87, Page 1835-1842

Posterior Inferior Cerebellar Artery Infarction, Surgical Intervention and Outcome

Abdin K. Kasim*[1], Bahaa Ghareeb Hassanin[1], Mohamed Abdala Abbas[2], Karam Kenawy[1]
[1]Department of Neurosurgery, Faculty of Medicine, Sohag University, Egypt
[2]Department of Neurology, Faculty of Medicine, Luxor University, Egypt
***Corresponding author:** Abdin K. Kasim, **Mobile:** (+20)102019 6831, **E-Mail:** abdin_mail@yahoo.com

ABSTRACT

Background: Posterior inferior cerebellar artery (PICA) infarcts are the most common of the cerebellar strokes and generally produce the largest infarcts with major complications including obstructive hydrocephalus and downward herniation of the cerebellar tonsils. Surgical intervention is widely accepted treatment choice but little data is available about the procedure and outcome.

Objective: The aim of the current work was to evaluate the role of suboccipital decompression craniectomy, atlas laminectomy, cerebellar tonsillectomy and duraplasty, in the management of isolated PICA territory infarction.

Patients and Methods: 12 patients, 7 males and 5 females, with PICA infarcts, were subjected to surgical intervention with pre-operative and 1-month post-operative clinical and radiological assessment using the Glasgow Coma Scale (GCS), the National Institutes of Health Stroke Scale (NIHSS), Barthel ADL index and Rankin scale in conjunction with CT and / or MRI brain.

Results: There are significant improvements in post-operative as compared to pre-operative assessment in different scales used in the study including the GCS, NIHSS, Barthel ADL index and Rankin Scale. Radiological evaluation showed obvious decompression of the brainstem and fourth ventricle.

Conclusions: It could be concluded that suboccipital craniectomy, atlas laminectomy and duraplasty are very effective treatment for patients with large PICA infarctions and can reverse effects of brainstem compression and hydrocephalus in most cases.

Keywords: PICA infarction, Suboccipital craniectomy, Tonsillar herniation

INTRODUCTION

Posterior inferior cerebellar artery (PICA) territory infarcts are the most common of the cerebellar strokes. The etiology of PICA infarcts includes intracranial vertebral atherosclerosis and embolism from a cardiac or proximal arterial source. Clinical features commonly include sudden vertigo, gait ataxia, and suboccipital headache. Nausea and vomiting are common. In large PICA territory infarcts, brainstem and fourth ventricle compression causing obstructive hydrocephalus and tonsillar herniation may threaten life. These patients may have decreased level of consciousness, reduced corneal reflex, ipsilateral facial palsy, and Babinski sign, denoting progression of mass effect. If conservative measures fail to control these manifestations, urgent surgery can be life-saving [(1)].

The general treatment of ischemic strokes is similar in cerebral and cerebellar infarctions. Treatment of associated edema includes anti-edema medications, control of intracranial pressure, external ventricular drains, and decompressive surgery, including resection of dead tissue in some cases. Patients developing hydrocephalus or brainstem compression are candidates for more extensive surgical treatment, if the conditions are considered reversible [(2)].

The incidence of cases of cerebellar infarction complicated with massive edema range from 20% to 50% [(3-10)]. Large infarctions of the cerebellum developing space-occupying edema are often missed until complications of mass effect cause clinical deterioration. The most dangerous complications are compression of brainstem and secondary hydrocephalus. As conservative treatment is not effective with extensive mass effect, and often fails; surgery is usually an accepted choice. Meanwhile, the best time and the factors indicating surgery remain unclear. Furthermore, it has not been sufficiently proved in prospective, multicenter, randomized controlled trials, which of the surgical procedures are more effective [(2)].

Conservative treatment includes airway protection, adequate ventilation, monitoring of circulation and prevention of vascular thrombosis and embolism. The conscious level must be closely monitored. Immediate brain imaging should be done if clinical deterioration or brainstem signs occurred [(11-13)].

Surgery includes ventricular drainage and decompressive suboccipital craniectomy. In contrast to the debate regarding surgical treatment of massive cerebral infarction, surgical intervention is a globally accepted choice in cerebellar infarcts causing mass effect and is advised strongly in current stroke guidelines [(12, 13)]. Ventricular Drainage is most commonly done through an extraventricular drain (EVD) in one lateral ventricle [(14, 15)]. Suboccipital Decompressive Craniectomy (SDC) is done to widen the posterior fossa to accommodate the swollen tissue and avoid fatal brainstem and fourth ventricle compression. Various surgical strategies include unilateral or bilateral craniectomy, opening of the foramen magnum, resection of the atlas lamina, and duraplasty. Removal of necrotic cerebellum may be used to create more space in the posterior fossa and lessen the developing cytotoxic edema [(16-18)].

Received: 10/11/2021
Accepted: 08/01/2022

The aim of the current work was to evaluate the role of suboccipital decompression craniectomy, atlas laminectomy, cerebellar tonsillectomy and duraplasty, in the management of isolated PICA territory infarction.

PATIENTS AND METHODS

From January 2017 to June 2021, at the Neurosurgical Department, Sohag University Hospitals, the data of 12 patients with isolated PICA territory infarctions subjected to surgical intervention were retrospectively reviewed and analyzed. Patients' data were protected for ethical consideration.

Clinical Evaluation:

Preoperative patients' clinical history and neurological examination were reviewed with special attention to data related to cerebellar signs, cranial nerve affection and intracranial hypertension. Demographic data were recorded, and all the patients were subjected to pre-operative assessment using GCS, NIHSS, Barthel ADL index and Modified Rankin Scale (MRS).

Radiological Evaluation:

All patients underwent CT scan of the brain and magnetic resonance imaging (MRI) brain. Radiological evaluation was aimed at delineating the cause, extent and effect of infarction to help surgical planning.

Surgical procedure:

Patients were operated in a prone position under general anesthesia. A vertical midline skin incision was made from the external occipital protuberance to the middle of cervical spine. Skin and subcutaneous tissues were incised and the underlying fascia and muscles were cut in the midline by the diathermy and separated subperiosteally from the suboccipital skull and the atlas lamina. Bilateral craniectomy was done inferior and medial to the transverse sinuses and enlarged to sufficiently open the foramen magnum with resection of the atlas lamina to access the cerebellar tonsils and provide sufficient surgical field. Under the surgical microscope, the dura was opened in a Y-shaped fashion to enhance the decompression. If brainstem and 4th ventricle decompression was considered insufficient, removal of infarcted tonsil was done. Finally, a dural graft was harvested from the nuchal fascia for duroplasty to give lax water tight dural closure with a suction drain left for about 48 hours.

Ventricular drains were used only when clinical picture of intracranial hypertension, including disturbance of conscious level, predominated and ventricular dilatation was evident in radiology. Ventricular drains were left for 2 to 3 days after removal of suction drains.

Data were evaluated for all patients 1-month post-operatively using GCS, NIHSS, Barthel ADL index and Rankin Scale. In addition, CT brain was done within the first 48 post-operative hours to roll out complications and after 1 month for follow up.

Ethical Consideration:

An approval of the study was obtained from Sohag University Academic and Ethical Committee. Every patient signed an informed written consent for acceptance of participation in the study. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Statistical analysis

The collected data were coded, processed and analyzed using the SPSS (Statistical Package for Social Sciences) version 22 for Windows® (IBM SPSS Inc, Chicago, IL, USA). Data were tested for normal distribution using the Shapiro Walk test. Qualitative data were represented as frequencies and relative percentages. Chi square test ($\chi 2$) to calculate difference between two or more groups of qualitative variables. Quantitative data were expressed as mean ± SD (Standard deviation). Independent samples t-test was used to compare between two independent groups of normally distributed variables (parametric data). P value < 0.05 was considered significant.

RESULTS

Demographic and clinical data:

Demographic and clinical data of patients are shown in table 1 and figure 1. They include 7 males and 5 females, with mean age of 55.58±6.88 (mean±SD) years. Hypertension was present in 58.3 % of cases.

Table (1): Demographic and clinical data of the study cases:

Item		Value
Age (years)	Mean±SD	55.58±6.88
Sex	Male	7(58.3%)
	Female	5(41.7%)
Chronic illnesses	DM	2(16.7%)
	Hypertension	7(58.3%)

Bulbar affection and unsteadiness were the most common clinical manifestations (figure 1).

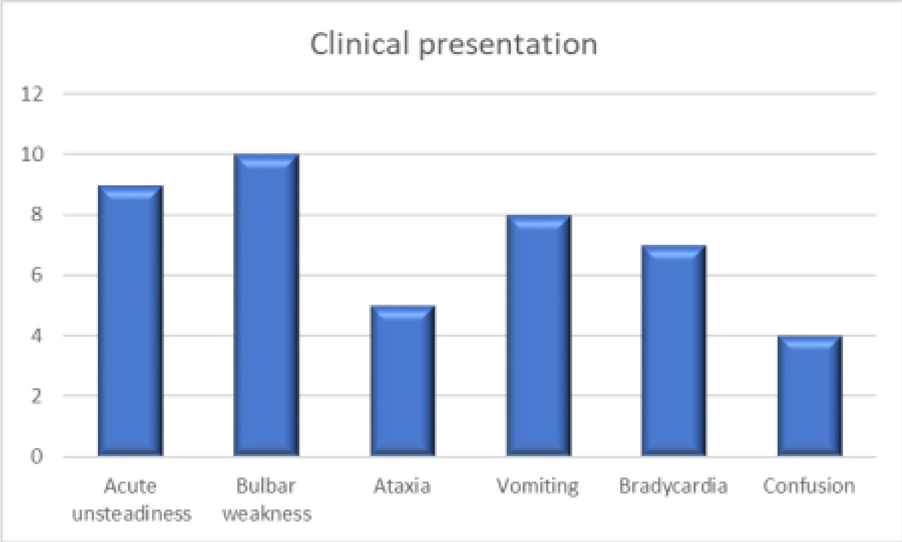

Figure (1): Clinical presentation of the study group

Radiological data:

Left side PICA territory infarction was present in 75 % of cases and hydrocephalus was radiologically evident in 50 % of cases (figure 2).

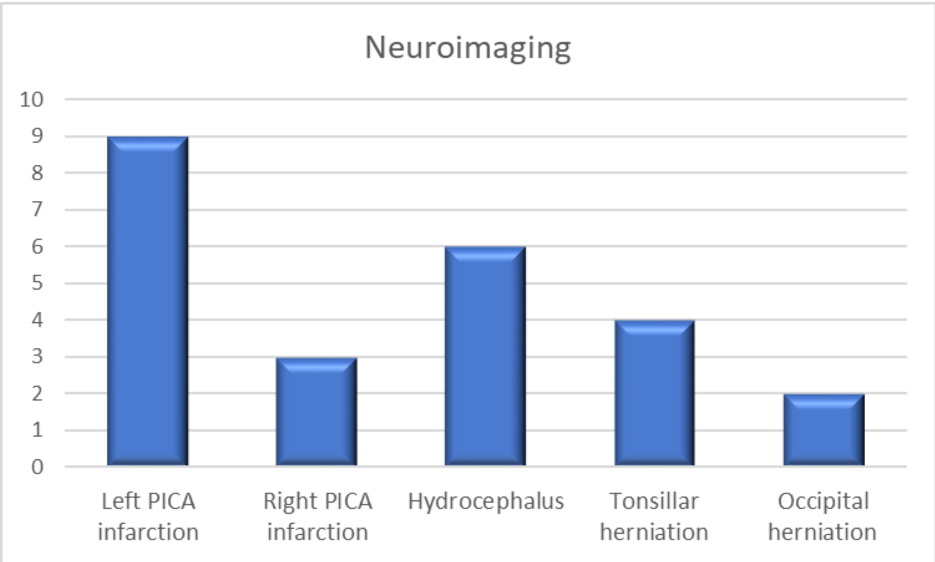

Figure (2): Preoperative neuroimaging examination

Surgical Procedures:

While decompressive craniectomy and C1 laminectomy were done in all patients, cerebellar tonsillectomy and duroplasty were adopted in most cases and EVD was needed only in 25 % of cases (table 2).

Table (2): Surgical procedures used in the study cases:

Management	No of Cases	%
External Ventricular Drain	3	25
Decompressive Craniectomy	12	100
Cerebellar Tonsillectomy	10	83.3
Atlas Laminectomy	12	100
Duroplasty	9	75%

Outcome scales evaluation:

There were significant improvements in post-operative as compared to pre-operative assessment in different scales used in the study (table 3) including the GCS, NIHSS, Barthel ADL index and MRS.

Table (3): Outcome of the procedure compared to the preoperative data:

Item		Preoperative	Postoperative	Paired t test	P value
GCS	Eye opening	2.67±0.49	3.83±0.39	7.000	**<0.001**
	Verbal	3.67±0.99	4.75±0.45	3.767	**0.003**
	Obeys orders	4.83±0.84	5.92±0.29	4.733	**0.001**
	Total GCS	11.17±1.95	14.50±1.00	6.504	**<0.001**
NIHSS	1a	1.50±0.52	0.33±0.49	2.889*	**0.004**
	1b	1.25±0.75	0.17±0.39	2.919*	**0.004**
	1c	1.17±0.84	0.08±0.29	2.739*	**0.006**
	2	0.58±0.52	0.00±0.00	2.646*	**0.008**
	3	0.17±0.39	0.00±0.00	1.414*	0.157
	4	0.00±0.00	0.00±0.00	-	-
	5	0.92±0.67	0.17±0.39	2.714*	**0.007**
	6	0.92±0.67	0.25±0.45	2.309*	**0.021**
	7	1.83±0.39	1.17±0.94	2.602	**0.025**
	8	0.00±0.00	0.00±0.00	-	-
	9	0.00±0.00	0.00±0.00	-	-
	10	1.75±0.45	0.83±0.39	6.167	**<0.001**
	11	0.00±0.00	0.00±0.00	-	-
	Total	10.08±2.39	3.00±2.05	14.636	**<0.001**
Barthel ADL index	Bowel	1.42±0.90	1.75±0.45	1.190*	0.234
	Bladder	1.25±0.87	1.83±0.39	2.070*	**0.038**
	Grooming	0.00±0.00	1.00±0.85	2.585*	**0.010**
	Toilet	0.25±0.45	1.25±0.45	2.972*	**0.003**
	Feeding	0.17±0.39	1.25±0.45	3.357*	**0.001**
	Transfer	0.17±0.39	2.17±0.58	3.274*	**0.001**
	Mobility	0.67±0.89	2.33±0.49	3.025*	**0.002**
	Dressing	0.08±0.29	1.42±0.67	3.017*	**0.003**
	Stairs	0.17±0.39	1.33±0.49	3.071*	**0.002**
	Bathing	0.00±0.00	0.25±0.45	1.732*	0.083
	Total score	4.17±3.27	14.58±2.94	19.184	**<0.001**
MRS		4.25±0.62	1.83±1.19	9.298	**<0.001**

* Wilcoxon test was used instead of paired t test due to non-parametric data

CASE PRESENTATIONS

Case 1: A 63-years old man, having DM and HTN presented with vomiting, RT hemi-ataxia and bradycardia. Pre and post operative CT and MRI brain (Fig 3 A and B) showed isolated Rt PICA infarction. SDC, C1 laminectomy, RT subpial tonsillectomy and duraplasty were done with marked postoperative improvement.

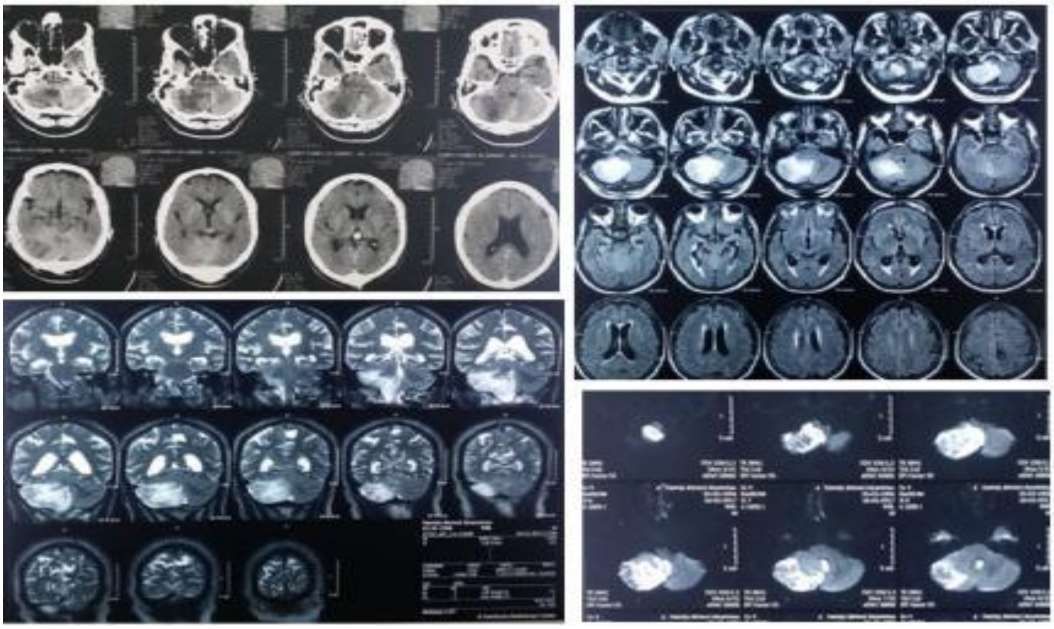

Figure (3): A: Preoperative CT and MRI scans of Case 1.

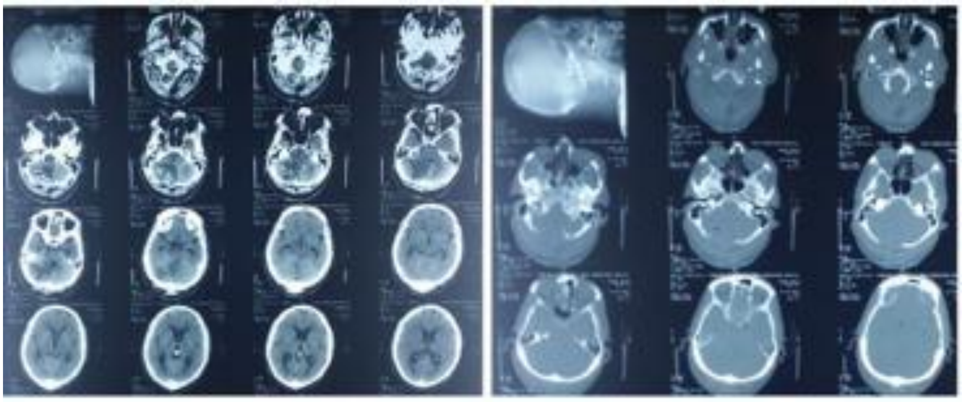

Figure (3) B: Postoperative CT scan of Case 1.

Case 2: A 46-years old man having HTN presented with headache, vomiting and LT hemiattaxia. CT and MRI brain (Fig 4 A) revealed isolated Lt PICA infarction and hydrocephalus. SDC, C1 laminectomy, Lt subpial tonsillectomy and duraplasty were done without a ventricular drain. Post operatively showed marked clinical and radiological (Fig 4 B) improvement.

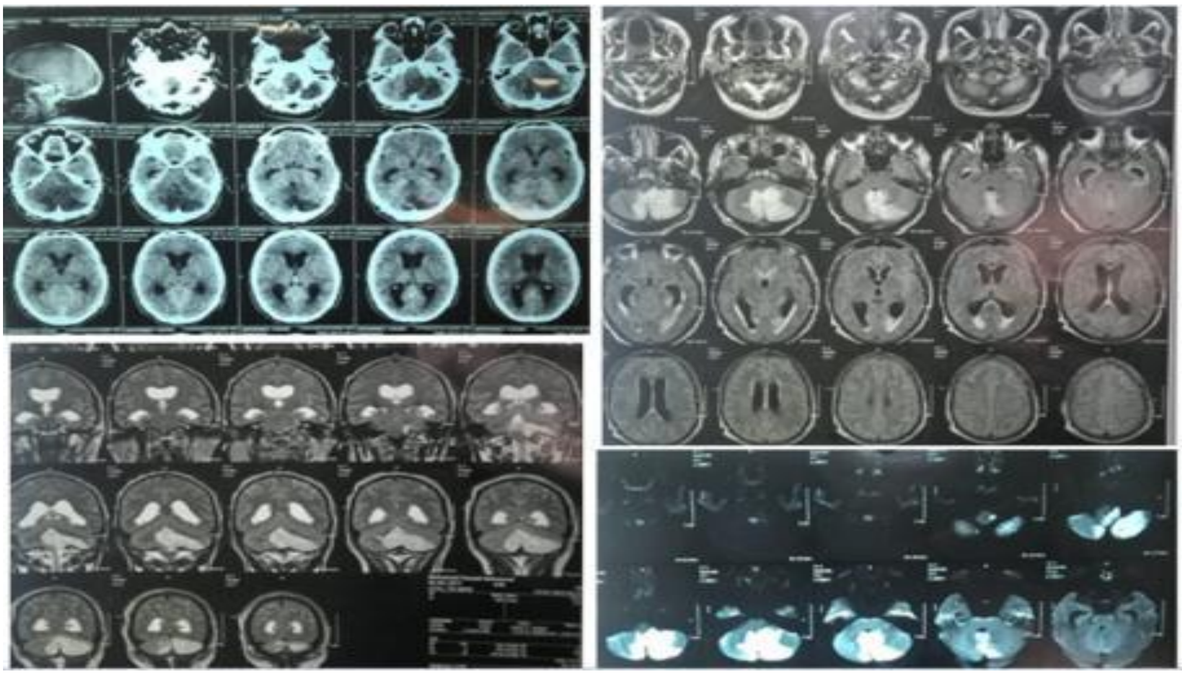

Figure (4): A: Preoperative CT and MRI scans of Case 2.

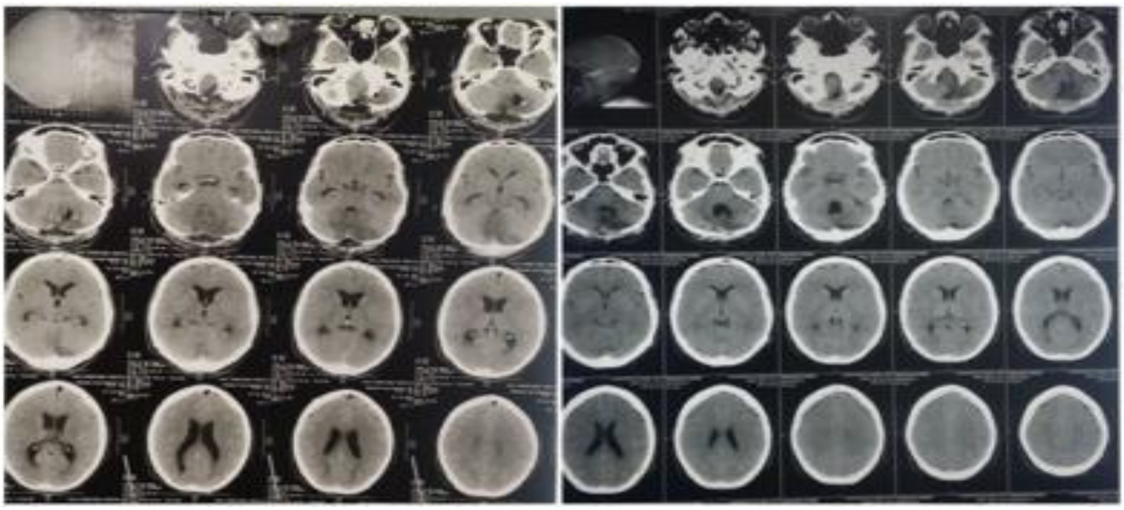

Figure (4): B: Postoperative and 1 month postoperative CT scans of Case 2.

Case (3): A 65-years old woman having HTN presented with headache, vomiting and LT hemiattaxia and bradycardia. CT and MRI brain (Fig 5 A) revealed isolated Lt PICA infarction and early hydrocephalus. SDC, C1 laminectomy, Lt subpial tonsillectomy and duraplasty were done without a ventricular drain. Post operatively showed both clinical and radiological (Fig 5 B) improvement.

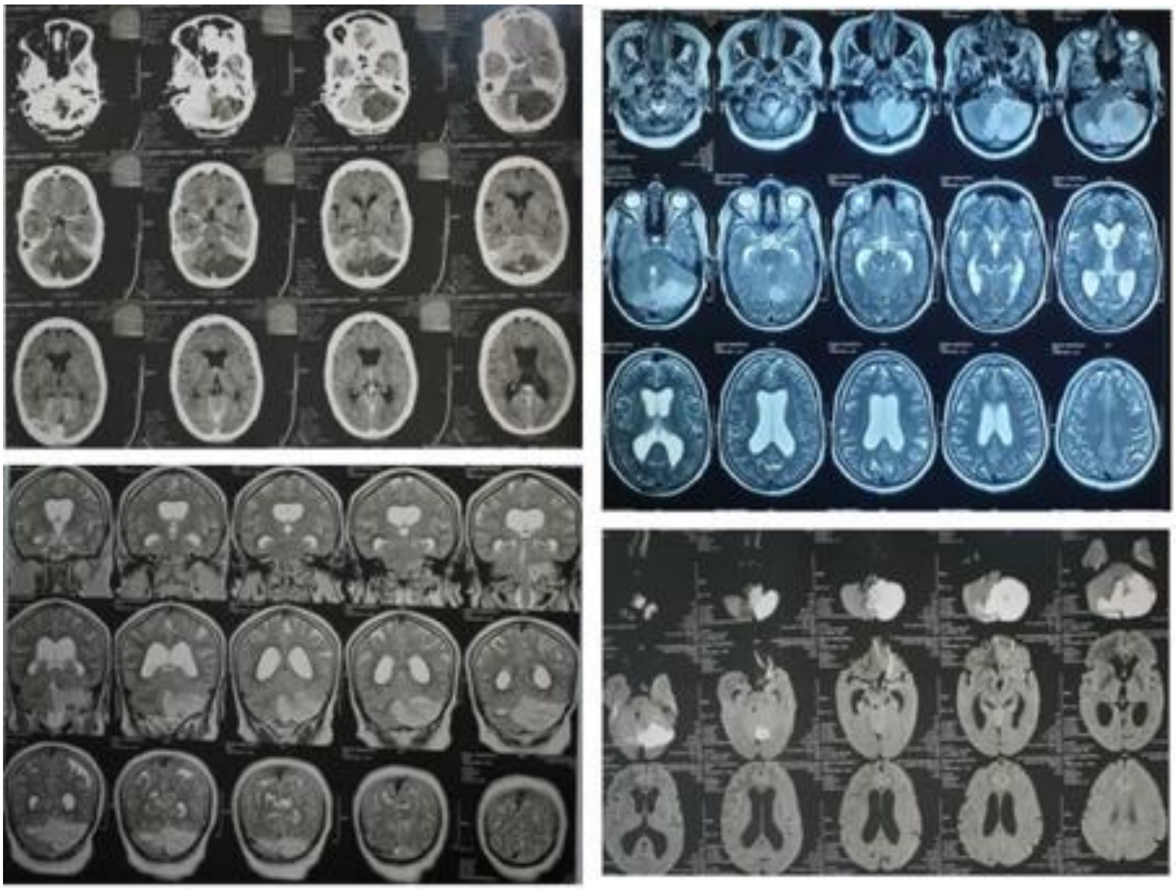

Figure (5): A: Preoperative CT and MRI scans of Case 3.

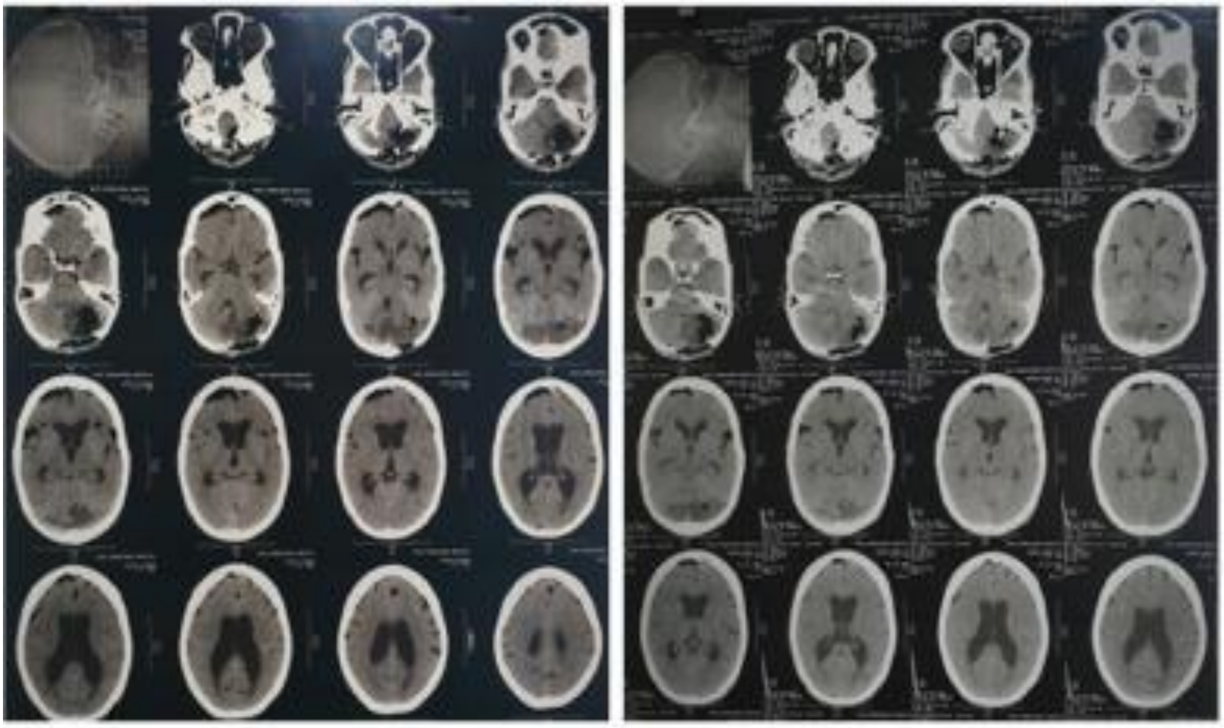

Figure (5): B: Postoperative and 1 month postoperative CT scans of Case 3.

DISCUSSION

Posterior inferior cerebellar artery (PICA) infarcts are the commonest strokes of the cerebellum and produce the biggest infarcts with serious complications including herniation of the cerebellar tonsils causing brainstem compression and obstructive hydrocephalus.

Stroke incidence increases parallel to age, doubling every decade after the fifth one. The mean age of ischemic stroke was 69.2 years in 2005[19]. In the current study, the mean age of the studied population is about 55.6 years, which is similar to other Egyptian studies [20]. Hypertension is found in more than half of the victims and DM in one sixth, this is similar to that reported in other Egyptian studies [21-24].

The PICA is an important blood supply of cerebellum, and its territory has the commonest cerebellar ischemic strokes, forming 40% [25]. The cerebellar and choroidal PICA branches have heavy connections with the anterior inferior cerebellar artery (AICA) and the superior cerebellar artery (SCA) while medullary branches are lacking such anastomosis. Thus, the most commonly affected region of the PICA infarct is the dorsolateral medulla. This causes what is known as the lateral medullary syndrome (LMS).

LMS, known as Wallenberg syndrome or PICA syndrome, is due to vascular damage to the lateral part of medullary oblongata. The responsible vessels are frequently the PICA or the VA [26, 27]. LMS is the typical presentation of PICA infarcts. The clinical picture of LMS varies depending on the site affected. Nausea, vomiting, vertigo, and nystagmus seem to be due to vestibular nucleus and vestibular–cerebellar pathway involvement. Dysphagia, dysarthria and dysphonia, with ipsilateral loss of gag reflex due to nucleus ambiguous defect, hiccough caused by glossopharyngeal and vagus nerve, are more significant in LMS patients related to patient care. Ataxia relates to cerebellar peduncles, spinocerebellar fibers and cerebellar hemisphere affection. Ipsilateral Horner syndrome can result from sympathetic fibers defects. Various combinations of the above symptoms can occur in LMS patients while most of them have sensory symptoms [26-29].

In the current study, clinical presentation of patients included acute unsteadiness, bulbar weakness, ataxia, vomiting, bradycardia and confusion. Bulbar affection and unsteadiness were the most common clinical manifestations.

MRI is superior in the early detection of infarcts and T2WI (T2-weighted images) are more sensitive than FLAIR (fluid-attenuated inversion recovery) in revealing posterior fossa infarcts [30, 31]. DWI (Diffusion-weighted magnetic resonance imaging) is widely preferred for early identification of infarcts. In the current study, T2WI, FLAIR and DWI were used to delineate PICA infarction. Left PICA territory infarction was present in 75% of cases and hydrocephalus was radiologically evident in 50 % of cases.

The available literature on massive cerebellar infarction supports that medical therapy alone has mortality rate of about 42.9% to 85% in the comatose patients [2]. Meanwhile, surgery is linked to a higher success reaching 76.8% for decompressive craniectomy and 77.5% if augmented by EVD. Evidence of a poorer outcome with older age is noticed by many authors [32]. In the current study, there were significant improvements in post-operative as compared to pre-operative assessment in the mean of different scales used in the study including the GCS, NIHSS, Barthel ADL index and Rankin Scale.

CONCLUSIONS

It could be concluded that although many cerebellar infarcts are benign, the early detection of those at risk for massive edema is essential. We should remember that late deterioration can occur and must not be missed. Suboccipital decompression craniotomy, atlas laminectomy in combination with duraplasty, seems to be the surgical treatment of choice. If patients present with a reduced level of consciousness secondary to brainstem or 4th ventricle compression, ipsilateral excision of the infarcted cerebellar tonsil can help to save the patient and may prevent the need for permanent CSF shunts. However, Patients with extensive brainstem damage, have a poor prognosis, and are not good candidates for surgery.

LIMITATIONS

Owing to the small number of patients, the available data do not supply sufficient evidence for the optimal surgical procedure or even accurately define timing of surgery. Furthermore, recent studies have shown that long-term outcome after space-occupying cerebellar infarction is more diverse than expected, and hence more studies are required to identify prognostic parameters. Randomized controlled trials are needed to define the exact value of SDC, tonsillectomy, EVD or a combination of either of them. Neurosurgeons and neurologists have to design researches to highlight the blind points of space-occupying cerebellar infarctions.

Financial support and sponsorship: Nil.
Conflict of interest: Nil.

REFERENCES

1. **Kuybu O, Tadi P, Dossani R (2021):** Posterior Cerebral Artery Stroke. StatPearls [Internet]. https://www.ncbi.nlm.nih.gov/books/NBK532296/
2. **Neugebauer H, Witsch J, Zweckberger K *et al.* (2013):** Space-occupying cerebellar infarction: complications, treatment, and outcome. Neurosurg Focus, 34(5): 8-12.
3. **Chen H, Lee T, Wei C (1992):** Treatment of cerebellar infarction by decompressive suboccipital craniectomy. Stroke, 23:957–961.

4. **Cuneo R, Caronna J, Pitts L *et al.* (1979):** Upward transtentorial herniation: seven cases and a literature review. Arch Neurol., 36:618–623.
5. **Heros R (1992):** Surgical treatment of cerebellar infarction. Stroke, 23:937–938.
6. **Hornig C, Rust D, Busse O *et al.* (1996):** Space-occupying cerebellar infarction. Clinical course and prognosis. Stroke, 25:372–374.
7. **Jauss M, Krieger D, Hornig C *et al.* (1999):** Surgical and medical management of patients with massive cerebellar infarctions: results of the German-Austrian Cerebellar Infarction Study. J Neurol., 246:257–264.
8. **Shenkin H, Zavala M (1982):** Cerebellar strokes: mortality, surgical indications, and results of ventricular drainage. Lancet, 2: 429–432.
9. **Tohgi H, Takahashi S, Chiba K *et al.* (1993):** Cerebellar infarction. Clinical and neuroimaging analysis in 293 patients. Stroke, 24:1697–1701.
10. **Mohsenipour I, Gabl M, Schutzhard E *et al.* (1999):** Suboccipital decompressive surgery in cerebellar infarction. Zentralbl Neurochir., 60:68–73.
11. **Frontera J, Lewin J, Rabinstein A *et al.* (2016):** Guideline for reversal of antithrombotics in intracranial hemorrhage: a statement for healthcare professionals from the Neurocritical Care Society and Society of Critical Care Medicine. Neurocrit Care, 24:6–46.
12. **Schwamm L, Audebert H, Amarenco P *et al.* (2009):** on behalf of the American Heart Association Stroke Council; Council on Epidemiology and Prevention; Interdisciplinary Council on Peripheral Vascular Disease; Council on Cardiovascular Radiology and Intervention. Recommendations for the implementation of telemedicine within stroke systems of care: a policy statement from the American Heart Association. Stroke, 40:2635–2660.
13. **European Stroke Organisation (ESO) Executive Committee (2008):** Guidelines for management of ischaemic stroke and transient ischaemic attack 2008. Cerebrovasc Dis., 25:457–507.
14. **Baldauf J, Oertel J, Gaab M *et al.* (2006):** Endoscopic third ventriculostomy for occlusive hydrocephalus caused by cerebellar infarction. Neurosurgery, 59:539–544.
15. **Yoshimura K, Kubo S, Nagashima M *et al.* (2007):** Occlusive hydrocephalus associated with cerebellar infarction treated with endoscopic third ventriculostomy: report of 5 cases. Minim Invasive Neurosurg., 50:270–272.
16. **Jauss M, Krieger D, Hornig C *et al.* (1999):** Surgical and medical management of patients with massive cerebellar infarctions: results of the German-Austrian Cerebellar Infarction Study. J Neurol., 246:257–264.
17. **Jüttler E, Schweickert S, Ringleb P *et al.* (2009):** Long-term outcome after surgical treatment for space-occupying cerebellar infarction: experience in 56 patients. Stroke, 40:3060–3066.
18. **Pfefferkorn T, Eppinger U, Linn J *et al.* (2009):** Long-term outcome after suboccipital decompressive craniectomy for malignant cerebellar infarction. Stroke, 40:3045–3050.
19. **Boehme A, Esenwa C, Elkind M (2017):** Stroke Risk Factors, Genetics, and Prevention. Circ Res., 120:472-495.
20. **Meneci T, El-Sheshiny A, Mohamed M (2021):** Ischemic stroke registry in Egypt: Hospital based study. Al-Azhar Med J., 50(1): 35 – 46.
21. **Khedr E, El-fetoh N, Al Attar G *et al.* (2013):** Epidemiological study and risk factors of stroke in Assiut Governorate, Egypt: community-based study. Neuroepidemiology, 40: 288-294.
22. **Farghaly W, El-Tallawy H, Shehata G *et al.* (2013):** Epidemiology of nonfatal stroke and transient ischemic attack in Al-Kharga District, New Valley, Egypt. Neuropsychiatr Dis Treat., 9:1785-1790.
23. **Broessner G, Beer R, Lackner P *et al.* (2009):** Prophylactic, endovascularly based, long-term normothermia in ICU patients with severe cerebrovascular disease: bicenter prospective, randomized trial. Stroke, 40: 657-665.
24. **Khedr E, Fawi G, Abdela M *et al.* (2014):** Prevalence of Ischemic and Hemorrhagic Strokes in Qena Governorate, Egypt: Community-based Study. Journal of Stroke and Cerebrovascular Diseases, 23: 1843-1848.
25. **Venti M (2012):** Cerebellar Infarcts and Hemorrhages. Front Neurol Neurosci., 30:171-5.
26. **Saleem F, Joe M (2020):** Lateral Medullary Syndrome. https://www.ncbi.nlm.nih.gov/books/NBK551670/
27. **Lui F, Tadi P, Anilkumar A (2020):** Wallenberg Syndrome. https://www.ncbi.nlm.nih.gov/books/NBK470174/
28. **Kim J (2003):** Pure lateral medullary infarction: clinical-radiological correlation of 130 acute, consecutive patients. Brain, 126(8):1864-72.
29. **Kim H, Lee H, Park J (2018):** Clinical course and outcome in patients with severe dysphagia after lateral medullary syndrome. Ther Adv Neurol Disord., 11: 864-69.
30. **Moreau F, Patel S, Lauzon M *et al.* (2012):** Cavitation after acute symptomatic lacunar stroke depends on time, location, and MRI sequence. Stroke, 43(7):1837-42.
31. **De Cocker L, Geerlings M, Hartkamp N *et al.* (2015):** Cerebellar infarct patterns: The SMART-Medea study. Neuroimage Clin., 8: 314-21.
32. **Jüttler E, Schweickert S, Ringleb PA, Huttner HB, Köhrmann M, Aschoff A(2009):** Long-term outcome after surgical treatment for space-occupying cerebellar infarction: experience in 56 patients. Stroke, 40:3060–3066.