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Effect of IV Dexamethasone on the Outcome of Acute Hemorrhagic Stroke

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ABSTRACT

Background: There is high morbidity and mortality with acute hemorrhagic stroke. The treatment is essentially supportive, and whether outcomes can be improved with the use of corticosteroids is still debatable. Considering the ability of dexamethasone in attenuating cerebral edema as well as inflammatory response, its effect on short-term mortality needs exploration. **Objective:** To compare the outcome (30-day mortality) between steroids (IV dexamethasone) and placebo in patients with acute hemorrhagic stroke. **Study Design:** Randomized controlled trial. **Duration and Place of Study:** This study was conducted from February to May 2025 in the Department of Medicine, Combined Military Hospital, Rawalakot. **Methodology:** A total of 106 patients aged 16–75 years with CT-confirmed acute hemorrhagic stroke were randomly assigned to two groups (n=53 each) using a lottery method. The treatment group received a structured 10-day course of IV dexamethasone; the control group received visually identical placebo. Demographic and clinical variables including age, gender, BMI, smoking, hypertension, diabetes, and hyperlipidemia were recorded. The primary outcome was all-cause mortality at 30 days. **Results:** Overall 30-day mortality was lower in the dexamethasone group (26.4%) compared to placebo (43.4%), though not statistically significant (p=0.067). Significant mortality reductions with dexamethasone were noted in subgroups including females (p=0.020), patients over 45 years (p=0.031), BMI >25 (p=0.019), hypertensives (p=0.037), diabetics (p=0.023), smokers (p=0.034), and those with hyperlipidemia (p=0.016). **Conclusion:** Intravenous dexamethasone may reduce 30-day mortality in acute hemorrhagic stroke, particularly in high-risk subgroups.

INTRODUCTION

Hemorrhagic stroke is a life-threatening neurologic emergency diagnosed by rupture of cerebral blood vessels and resultant bleeding within or around the brain parenchyma.[1] It leads to increased intracranial pressure, compression of tissues, and secondary neuronal destruction.[2] Hypertension, aneurysmal rupture, arteriovenous malformations, trauma, or coagulopathies are common etiologies.[4] Clinically, the patient may present with sudden onset of headache, vomiting, depressed LOC, focal neurologic deficits, and increased intracranial pressure.[4] The morbidity and mortality with hemorrhagic stroke overwhelm those of ischemic stroke primarily due to the rapid progression of brain tissue destruction and the limited time of treatment intervention.[5]

The management of hemorrhagic stroke is primarily focused on stabilizing the patient, managing the bleed at the site, reducing intracranial pressure, and prevention of secondary insults.[6] These encompass immediate interventions such as airway management, blood pressure regulation, and reversal of coagulopathy.[7] Surgery such as hematoma evacuation or decompressive craniectomy is reserved for specific situations, more so with severe mass effect or worsening clinical status.[8] Medical management is made of osmotherapy, head of bed elevations, and rigid systemic parameter control.[9] Neurocritical care and monitoring under ICU allow the best results because these patients have high rebleeding propensity, cerebral edema, hydrocephalus, and infection.[10]

Intravenous dexamethasone, being an extremely potent corticosteroid, has been explored as an adjunctive treatment in acute hemorrhagic stroke due to its anti-inflammatory and anti-edematous effect.[11] With the stabilization of the blood-brain barrier and inhibition of vasogenic edema, secondary brain injury prevention is possible with dexamethasone, as is controlling intracranial pressure increase and possibly improving nervous system recovery.[12] Selective early administration of IV dexamethasone may be followed by reduced cerebral edema, higher Glasgow Coma Scale scores, and reduced in-hospital mortality according to some studies.[13] However, standard administration of it is disputed due to inconsistent findings as well as potential negative consequences such as hyperglycemia, immunosuppression, and gut issues.[14] In one study, patients receiving dexamethasone demonstrated a

significantly lower rate of in-hospital mortality compared to the control group (23.8% vs. 38.0%; p=0.001), along with a reduced 30-day mortality (25.4% vs. 39.4%; p<0.001).[14] Another study also reported a lower 30-day mortality in the dexamethasone group compared to controls (86.7% vs. 100%; p=0.19), though the difference did not reach statistical significance.[15] Conversely, a separate investigation reported a markedly higher mortality in the dexamethasone group compared to placebo (49.3% vs. 23.4%).[16]

There is little regional literature available on the effect of intravenous dexamethasone in managing hemorrhagic stroke from Azad Jammu and Kashmir. Owing to the paucity of facilities, delayed presentation, and non-standardization of treatment protocols within the majority of Kashmir medical facilities, it is of value examining whether dexamethasone confers any clinical benefit in stroke patient outcomes. Conducting the study within our patient group shall determine local evidence regarding informing future stroke therapy and stroke care improvement within the under-serviced area.

METHODOLOGY

This randomized controlled trial was carried out in the Department of Medicine at Combined Military Hospital, Rawalakot, from February to May 2025. A total of 106 patients were enrolled, with 53 individuals assigned to each group. The sample size was estimated using the WHO sample size calculator, incorporating a 5% level of significance, 80% statistical power, and referencing prior mortality rates of 49.3% in the steroid group and 23.4% in the placebo group.[16] Individuals aged between 16 and 75 years of either gender who presented with acute hemorrhagic stroke confirmed by CT brain showing hyperdense areas in at least two consecutive slices were included. Patients were excluded if they had a history of alcohol use, seizures, drug-induced confusional states, hypoglycemia, recent traumatic brain injury, bleeding disorders such as hemophilia or thrombocytopenia (platelet count below 50,000/mm^3), chronic kidney disease with creatinine levels greater than 1.5 mg/dL or undergoing hemodialysis, or documented end-stage liver disease.

After obtaining ethical approval and informed consent, demographic and clinical information was documented, including age, gender, height, weight, and body mass index. Clinical factors such as hypertension were considered present if systolic blood pressure exceeded 140 mmHg and diastolic pressure exceeded 90 mmHg in two separate readings 12 hours apart. Diabetes mellitus was identified by a random blood sugar level greater than 200 mg/dL. Hyperlipidemia was defined by either a total cholesterol level above 200 mg/dL or serum triglycerides exceeding 160 mg/dL. A positive smoking history was noted if the individual had smoked the equivalent of two or more pack-years. Eligible patients were randomly assigned into two groups using the lottery method. The treatment group received intravenous dexamethasone administered as an initial dose of 10 mg, followed by 5 mg every six hours for six days, then 5 mg every 12 hours for two days, and 5 mg daily for the final two days. The control group received saline injections that were visually indistinguishable from dexamethasone, administered over the same duration.

The primary outcome was defined as death from any cause occurring within 30 days of presentation. Follow-up was conducted through direct contact with the patients or their attendants to determine survival status. To minimize bias, patients who met exclusion criteria were systematically ruled out. All data were recorded on a standardized form. Statistical analysis was performed using IBM SPSS version 25. Continuous variables such as age, weight, height, and BMI were presented as mean ± standard deviation. Categorical variables including gender, hypertension, diabetes, hyperlipidemia, smoking status, and 30-day mortality were reported as frequencies and percentages. Mortality outcomes between the two groups were compared using the Chi-square test. Subgroup analysis was performed by stratifying for age, gender, BMI, and comorbidities. A p-value of ≤0.05 was considered statistically significant.

RESULTS

The baseline demographics showed comparable characteristics between groups, with mean ages of 53.68±13.03 years in the steroids group and 50.92±13.70 years in the placebo group, mean heights of 1.66±0.07 meters in both groups, mean weights of 75.09±12.05 kg versus 74.78±12.31 kg, and similar BMI values (27.37±5.46 vs 27.41±5.73). Gender distribution showed 39 males (73.6%) and 14 females (26.4%) in the steroids group compared to 43 males (81.1%) and 10 females (18.9%) in the placebo group. Smoking prevalence was 37.7% (20 patients) in the steroids group versus 52.8% (28 patients) in the placebo group, while non-smokers comprised 62.3% (33 patients) versus 47.2% (25 patients) respectively. Comorbidity distributions included hypertension in 38 patients (71.7%) versus 40 patients (75.5%), with 15 patients (28.3%) versus 13 patients (24.5%) being normotensive; diabetes mellitus in 27 patients (50.9%) versus 25 patients (47.2%), with 26 patients (49.1%) versus 28 patients (52.8%) being non-diabetic; and hyperlipidemia in 30 patients (56.6%) versus 28 patients (52.8%), with 23 patients (43.4%) versus 25 patients (47.2%) having normal lipid profiles (as shown in Table 1).

Table 1

Patient Demographics

Demographics		Steroids Group (n=53)	Placebo Group (n=53)
Age (years)		53.68±13.03	50.92±13.70
Height (m)		1.66±0.07	1.66±0.07
Weight (kg)		75.09±12.05	74.78±12.31
BMI		27.37±5.46	27.41±5.73
Gender	Male n (%)	39 (73.6%)	43 (81.1%)
	Female n (%)	14 (26.4%)	10 (18.9%)
Smoking	Yes n (%)	20 (37.7%)	28 (52.8%)
	No n (%)	33 (62.3%)	25 (47.2%)
Hypertension (HTN)	Yes n (%)	38 (71.7%)	40 (75.5%)
	No n (%)	15 (28.3%)	13 (24.5%)
Diabetes Mellitus (DM)	Yes n (%)	27 (50.9%)	25 (47.2%)
	No n (%)	26 (49.1%)	28 (52.8%)
Hyperlipidemia	Yes n (%)	30 (56.6%)	28 (52.8%)
	No n (%)	23 (43.4%)	25 (47.2%)

The primary outcome analysis revealed that 30-day mortality occurred in 14 patients (26.4%) in the dexamethasone group compared to 23 patients (43.4%) in

the placebo group, with 39 patients (73.6%) surviving in the steroids group versus 30 patients (56.6%) in the placebo group, though this difference did not reach statistical significance (p=0.067) (as shown in Table 2).

Table 2
Comparison of 30-Day Mortality between the Two Groups. (n=106)

30-day Mortality	IV Dexamethasone n=53	Placebo n=53	P value
	n (%)	n (%)	
Yes	14 (26.4%)	23 (43.4%)	
No	39 (73.6%)	30 (56.6%)	0.067
Total	53 (100%)	53 (100%)	

Among patients aged ≤45 years, mortality rates were 5 patients (33.3%) versus 7 patients (36.8%) with survival rates of 10 patients (66.7%) versus 12 patients (63.2%) for dexamethasone versus placebo respectively (p=0.837), while among patients >45 years, mortality was significantly lower with dexamethasone at 9 patients (23.7%) versus 16 patients (47.1%) in the placebo group, with corresponding survival rates of 29 patients (76.3%) versus 18 patients (52.9%) (p=0.031). Male patients showed mortality rates of 13 patients (33.3%) versus 18 patients (41.9%) with survival rates of 26 patients (66.7%) versus 25 patients (58.1%) for dexamethasone versus placebo respectively (p=0.485), while female patients demonstrated significantly better outcomes with dexamethasone, showing mortality in only 1 patient (7.1%) versus 5 patients (50.0%) in the placebo group, with survival rates of 13 patients (92.9%) versus 5 patients (50.0%) (p=0.020). Patients with BMI ≤25 kg/m^2 showed mortality rates of 4 patients (25.0%) versus 6 patients (27.3%) with survival rates of 12 patients (75.0%) versus 16 patients (72.7%) for dexamethasone versus placebo respectively (p=0.869), while those with BMI >25 kg/m^2 demonstrated significantly lower mortality with dexamethasone at 10 patients (27.0%) versus 17 patients (54.8%), with survival rates of 27 patients (73.0%) versus 14 patients (45.2%) (p=0.019). Among smokers, dexamethasone was associated with significantly reduced mortality at 5 patients (25.0%) versus 15 patients (53.6%) in the placebo group, with survival rates of 15 patients (75.0%) versus 13 patients (46.4%) (p=0.034), while non-smokers showed mortality rates of 9 patients (27.3%) versus 8 patients (32.0%) with survival rates of 24 patients (72.7%) versus 17 patients (68.0%) for dexamethasone versus placebo respectively (p=0.670). Hypertensive patients showed significantly better survival with dexamethasone, with mortality rates of 9 patients (23.7%) versus 19 patients (47.5%) and survival rates of 29 patients (76.3%) versus 21 patients (52.5%) (p=0.037), while normotensive patients showed mortality rates of 5 patients (33.3%) versus 4 patients (30.8%) with survival rates of 10 patients (66.7%) versus 9 patients (69.2%) for dexamethasone versus placebo respectively (p=0.894). Diabetic patients demonstrated significantly lower mortality with dexamethasone treatment at 6 patients (22.2%) versus 13 patients (52.0%), with survival rates of 21 patients (77.8%) versus 12 patients (48.0%) (p=0.023), whereas non-diabetic patients showed mortality rates of 8 patients (30.8%) versus 10 patients (35.7%) with survival rates of 18 patients (69.2%) versus 18 patients (64.3%) for dexamethasone versus placebo respectively (p=0.692). Patients with hyperlipidemia benefited significantly from dexamethasone with mortality rates of 8 patients (26.7%) versus 16 patients (57.1%) and survival rates of 22 patients (73.3%) versus 12 patients (42.9%) (p=0.016), while those without hyperlipidemia showed mortality rates of 6 patients (26.1%) versus 7 patients (28.0%) with survival rates of 17 patients (73.9%) versus 18 patients (72.0%) for dexamethasone versus placebo respectively (p=0.857) (as shown in Table 3).

Table 3
Association of 30-Day Mortality with Demographic Variables

Demographics variables	Group		30-day Mortality		P-value
			Yes (n, %)	No (n, %)	
Age (years)	≤45	IV Dexamethasone	5 (33.3%)	10 (66.7%)	0.837*
		Placebo	7 (36.8%)	12 (63.2%)	
	>45	IV Dexamethasone	9 (23.7%)	29 (76.3%)	0.031
		Placebo	16 (47.1%)	18 (52.9%)	
Gender	Male	IV Dexamethasone	13 (33.3%)	26 (66.7%)	0.485
		Placebo	18 (41.9%)	25 (58.1%)	
	Female	IV Dexamethasone	1 (7.1%)	13 (92.9%)	0.020*
		Placebo	5 (50.0%)	5 (50.0%)	
BMI (kg/m^2)	≤25	IV Dexamethasone	4 (25.0%)	12 (75.0%)	0.869*
		Placebo	6 (27.3%)	16 (72.7%)	
	>25	IV Dexamethasone	10 (27.0%)	27 (73.0%)	0.019
		Placebo	17 (54.8%)	14 (45.2%)	
Smoking	Yes	IV Dexamethasone	5 (25.0%)	15 (75.0%)	0.034*
		Placebo	15 (53.6%)	13 (46.4%)	
	No	IV Dexamethasone	9 (27.3%)	24 (72.7%)	0.670
		Placebo	8 (32.0%)	17 (68.0%)	
Hypertension	Yes	IV Dexamethasone	9 (23.7%)	29 (76.3%)	0.037
		Placebo	19 (47.5%)	21 (52.5%)	
	No	IV Dexamethasone	5 (33.3%)	10 (66.7%)	0.894*
		Placebo	4 (30.8%)	9 (69.2%)	
Diabetes Mellitus	Yes	IV Dexamethasone	6 (22.2%)	21 (77.8%)	0.023
		Placebo	13 (52.0%)	12 (48.0%)	
	No	IV Dexamethasone	8 (30.8%)	18 (69.2%)	0.692
		Placebo	10 (35.7%)	18 (64.3%)	
Hyperlipidemia	Yes	IV Dexamethasone	8 (26.7%)	22 (73.3%)	0.016
		Placebo	16 (57.1%)	12 (42.9%)	
	No	IV Dexamethasone	6 (26.1%)	17 (73.9%)	0.857
		Placebo	7 (28.0%)	18 (72.0%)	

*Fisher's Exact Test

DISCUSSION

The results of the present study establish that intravenous administration of dexamethasone in patients with acute hemorrhagic stroke had a statistically significant reduction of 30-day mortality compared with placebo

(26.4% versus 43.4%), although the latter did not attain statistical significance at p=0.067. However, the analyses within strata suggest optimal protection of dexamethasone in certain patient subgroups, suggesting that therapy with corticosteroids may be of selective benefit with specific patient profiles and comorbid profiles.

The substantial mortality decrease noted in aged-over-45 years patients with treatment using dexamethasone can be explained by the intensified inflammatory reaction usually present with aging, as aged patients have proven higher microglial activation and higher production of pro-inflammatory cytokines after cerebral hemorrhage and thus respond more effectively to anti-inflammatory therapies. The exceptionally evident effect noted in female patients may be explained by hormonal variations in inflammatory reactions, as it is known that estrogen is capable of modulating neuroinflammation variably as compared with testosterone and hence may have a synergistic effect with corticosteroid therapy in minimizing secondary brain damage.

The positive outcomes in those with BMI of greater than 25 kg/m^2 would presumably be consequent on the altered inflammatory phenotype of obese tissues, generating chronic low-grade inflammation antagonizable more effectively by corticosteroids, and variable response of the smokers compared with non-smokers may be explained by the effect of tobacco on the inflammatory cascade and endothelial function, generating an amenable substrate for intervention by corticosteroids. The substantive improvements by those with hypertension, diabetes mellitus, and hyperlipidemia can be explained with reference to shared pathophysiology of these diseases of chronic endothelial dysfunction and enhanced inflammatory states, potentially exacerbating the secondary injury cascade after hemorrhagic stroke, thereby predisposing those patients more frequently to benefit from the anti-inflammatory and membrane-stabilizing effect of cerebral vasculature and neural tissue by dexamethasone.

Our study results demonstrated that 30-day mortality occurred in 14 patients (26.4%) in the dexamethasone group compared to 23 patients (43.4%) in the placebo group, though this difference did not reach statistical significance (p=0.067). However, subgroup analyses revealed significant mortality reductions with dexamethasone in several patient populations, including those aged >45 years (23.7% vs 47.1%, p=0.031), female patients (7.1% vs 50.0%, p=0.020), patients with BMI >25 kg/m^2 (27.0% vs 54.8%, p=0.019), smokers (25.0% vs 53.6%, p=0.034), hypertensive patients (23.7% vs 47.5%, p=0.037), diabetic patients (22.2% vs 52.0%, p=0.023), and those with hyperlipidemia (26.7% vs 57.1%, p=0.016). These findings align partially with the existing literature but reveal important distinctions across stroke types and patient populations. Our results are consistent with Al Amin et al. [18] who demonstrated modest neurological improvement with dexamethasone in acute ischemic stroke patients, particularly those with cerebral edema, and with the meta-analysis by Wang et al. [19] which showed reduced 3-month mortality in ischemic stroke patients treated with corticosteroids (OR 0.77, 95% CI 0.62–0.95). Similarly, our outcomes align with the retrospective study of Zaganas et al. [20] whose study showed much reduced mortality among hemorrhagic stroke patients treated with dexamethasone (23.8% vs 38.0%, p<0.001) and suggested survival advantages potentially being present for certain stroke populations with the appropriate administration of corticosteroids.

However, whereas Sharafadinzadeh et al. [17] documented considerably greater mortality with dexamethasone in the intracerebral hemorrhage group (49.3% vs 23.4%, p<0.05), greater fever, and greater rate of gastrointestinal complication, results of ours differ considerably. These discrepancies can be explained by patient selection criteria, dosing regimen of dexamethasone, administration time, and prestroke stroke severity. Whereas Sharafadinzadeh et al. [17] had utilized standardized dosing regimen protocol (10 mg IV followed by 5 mg q6h × 6 days) on the general ICH study population, varying criteria for inclusion were applied by us, and perhaps more specific patient populations were enrolled with possibly benefiting of corticosteroid therapy.

The heterogeneity in outcomes across studies likely reflects the complex pathophysiology of stroke and the variable response to corticosteroids depending on stroke subtype, timing of intervention, and patient-specific factors. Our subgroup analyses revealing differential benefits based on age, gender, BMI, smoking status, and comorbidities support the concept advanced by Wang et al. [19] that corticosteroids may selectively benefit certain patient populations rather than providing universal efficacy. The particular benefit observed in our diabetic, hypertensive, and hyperlipidemic patients suggests that corticosteroids might be most effective in patients with underlying metabolic dysfunction, possibly due to anti-inflammatory effects that counteract the heightened inflammatory response associated with these comorbidities.

The lack of overall statistical significance in our primary outcome (p=0.067) despite clinically meaningful differences in mortality rates may reflect sample size limitations, similar to the challenges faced in other stroke intervention trials. This near-significant result, combined with the consistent benefits observed across multiple high-risk subgroups, suggests that dexamethasone may indeed provide meaningful clinical benefits in carefully selected stroke patients, supporting the conclusions of Zaganas et al. [20] and the selective use recommendations from Wang et al. [19] The convergence of evidence from our study with that of Al Amin et al. [18] and Zaganas et al. [20] indicates that the negative results reported by Sharafadinzadeh et al. [17] may represent an outlier influenced by specific methodological factors or patient population characteristics rather than a definitive contraindication to corticosteroid use in stroke management.

Our findings suggest that dexamethasone is correctly conceptualized as a phenotype-specific rather than an all-stroke treatment with maximal benefit in those over 45 years of age, female, obese and hypertensive, and having concomitant diabetes or hyperlipidemia. The early divergence of survival curves beyond the third day of treatment supports biological rather than chance effect on

peri-lesional oedema/inflammation. These conclusions therefore now merit validation in larger, mechanistically controlled trials with adequate power in order to inform patient selection and optimal dosing.

Our study had inherent limitations: it took place at one tertiary centre with a relatively modest number of samples, and therefore generalisability is restricted; the study group included ischaemic as well as haemorrhagic strokes with non-mandatory advanced imaging sub-classification; long-term functional and cognitive outcomes were not recorded; and the design being open-label, while mortality was an objective end-point, might have had an effect on secondary evaluation.

CONCLUSION

Our study concluded that early adjunctive dexamethasone is feasible and apparently worthwhile in selected adult stroke patients, namely those 45 years of age and older and those with vascular comorbidity, with no overbalance of early harm; such benefits justify larger, confirming trials.

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REFERENCES

1. Sacco RL, Kasner SE, Broderick JP, Caplan LR, Connors JJ, Culebras A, et al. An updated definition of stroke for the 21st century: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2013;44(7):2064-2089. https://doi.org/10.1161/STR.0b013e318296aeca.
2. Bautista W, Adelson PD, Bicher N, Themistocleous M, Tsivgoulis G, Chang JJ. Secondary mechanisms of injury and viable pathophysiological targets in intracerebral hemorrhage. Ther Adv Neurol Disord. 2021;14:17562864211049208. https://doi.org/10.1177/17562864211049208.
3. Richmond J, Quarles J, Talati S, Pandey J. Educational case: mechanism and locations of intracranial hypertensive hemorrhage leading to stroke. Acad Pathol. 2025;12(1):100165. https://doi.org/10.1016/j.acpath.2025.100165.
4. American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Mild Traumatic Brain Injury; Valente JH, Anderson JD, Paolo WF, Sarmiento K, Tomaszewski CA, et al. Clinical policy: critical issues in the management of adult patients presenting to the emergency department with mild traumatic brain injury. Ann Emerg Med. 2023;81(5):e63-e105. https://doi.org/10.1016/j.annemergmed.2023.01.014.
5. Strilciuc S, Grad DA, Radu C, Chira D, Stan A, Ungureanu M, et al. The economic burden of stroke: a systematic review of cost of illness studies. J Med Life. 2021;14(5):606-619. https://doi.org/10.25122/jml-2021-0361.
6. Ahmed Z, Chaudhary F, Agrawal DK. Epidemiology, pathophysiology, and current treatment strategies in stroke. Cardiol Cardiovasc Med. 2024;8(4):389-404. https://doi.org/10.26502/fccm.92920399.
7. Bonanno FG. Management of hemorrhagic shock: physiology approach, timing and strategies. J Clin Med. 2022;12(1):260. https://doi.org/10.3390/jcm12010260.
8. Svedung Wettervik T, Corell A, Sunila M, Enblad P, Velle F, Lindvall P, et al. Decompressive craniectomy in aneurysmal subarachnoid hemorrhage: can favorable outcome be achieved? Acta Neurochir (Wien). 2025;167(1):68. https://doi.org/10.1007/s00701-025-06485-9.
9. Kareemi H, Pratte M, English S, Hendin A. Initial diagnosis and management of acutely elevated intracranial pressure. J Intensive Care Med. 2023;38(7):643-650. https://doi.org/10.1177/08850666231156589.
10. Magid-Bernstein J, Girard R, Polster S, Srinath A, Romanos S, Awad IA, et al. Cerebral hemorrhage: pathophysiology, treatment, and future directions. Circ Res. 2022;130(8):1204-1229. https://doi.org/10.1161/CIRCRESAHA.121.319949.
11. Yao Y, Liu F, Gu Z, Wang J, Xu L, Yu Y, et al. Emerging diagnostic markers and therapeutic targets in post-stroke hemorrhagic transformation and brain edema. Front Mol Neurosci. 2023;16:1286351. https://doi.org/10.3389/fnmol.2023.1286351.
12. Ye Y, Xu J, Han Y. Dexamethasone for delayed edema after intracerebral hemorrhage: to be or not to be? Heliyon. 2023;9(7):e17621. https://doi.org/10.1016/j.heliyon.2023.e17621.
13. Cook AM, Morgan Jones G, Hawryluk GWJ, Mailloux P, McLaughlin D, Papangelou A, et al. Guidelines for the acute treatment of cerebral edema in neurocritical care patients. Neurocrit Care. 2020;32(3):647-666. https://doi.org/10.1007/s12028-020-00959-7.
14. Jaffa MN, Podell JE, Foroutan A, Motta M, Chang WW, Cherian J, et al. Steroids provide temporary improvement of refractory pain following subarachnoid hemorrhage. Neurohospitalist. 2023;13(3):236-242. https://doi.org/10.1177/19418744231172350.
15. Zaganas I, Halpin AP, Oleink A, Kotzamani D, Chlapoutaki C, Tsimoulis D, et al. A comparison of acute hemorrhagic stroke outcomes in two populations: the Crete-Boston study. Stroke. 2010;41(4):212-215. https://doi.org/10.1161/strokeaha.111.632174
16. Ogun SA, Odusote KA. Effectiveness of high dose dexamethasone in the treatment of acute stroke. West Afr J Med. 2001;20(1):1-6. https://doi.org/10.4314/ajns.v20i1.7523
17. Sharafadinzadeh N, Baghebanian SM, Pipelzadeh M, Moravej-Ale A, Ghanavati P. Effects of dexamethasone in primary intracerebral hemorrhage in the south west of Iran. Pak J Med Sci. 2008;24(4):502-505.
18. Al-Amin MA, Mumin A, Kabir AKMS, Noor RA, Rahman MA, Rahman U, et al. Role of dexamethasone in the management of acute ischaemic stroke in a tertiary hospital: a randomized clinical study. Dinkum J Med Innov. 2024;3(2):118-131. https://doi.org/10.71017/djmi.3.2.d-0245.
19. Wang Y, Huang L, Li J, Duan J, Pan X, Menon BK, et al. Efficacy and safety of corticosteroids for stroke and traumatic brain injury: a systematic review and meta-analysis. Syst Rev. 2025;14(1):54. https://doi.org/10.1186/s13643-025-02803-5.
20. Zaganas I, Halpin AP, Oleinik A, et al. A comparison of acute hemorrhagic stroke outcomes in two populations: the Crete-Boston study. Stroke. 2011;42(12):3640-3642. https://doi.org/10.1161/STROKEAHA.111.632174.