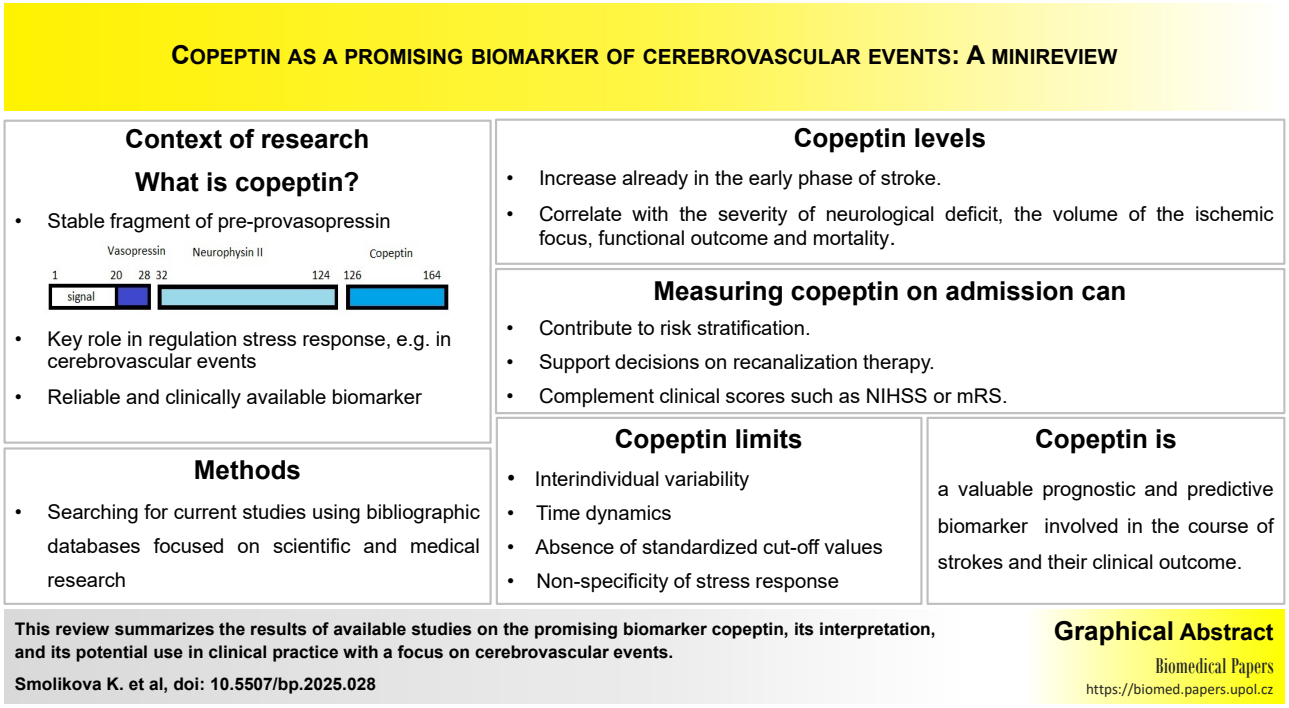
Copeptin as a promising biomarker of cerebrovascular events: A minireview

Kamila Smolikova[1,2], Pavel Potuznik[1,2], Marek Peterka[1,2], Jakub Vejskal[1,2], Martina Pestova[3]

Cerebrovascular events remain a major health issue, and despite significant improvements in diagnosis and treatment in recent years, their high morbidity and mortality mean that increasing attention is now being given to prevention and early prediction. One option for achieving this is to examine various biomarkers. This mini review covers copeptin, a 39-amino-acid glycopeptide, derived from the precursor protein pre-provasopressin. Copeptin shows great potential in the diagnosis and prognosis of diverse diseases, including cerebrovascular events. Compared to arginine vasopressin or cortisol alone, it shows better potential for use in emergency care due to its rapid determination. Understanding the role of copeptin and its clinical applications is essential for advancing patient care and treatment strategies. This mini review presents selected studies on the use of copeptin as a potential biomarker for cerebrovascular events. Its level correlates with the severity of both clinical and radiological impairment after a stroke. A correlation has been demonstrated between copeptin levels and the ability to predict a cerebrovascular event recurrence within 90 days after a transient ischemic attack. This mini review also includes the limits to copeptin, which are influenced by selected vascular risk factors for cerebrovascular events.



Key words: copeptin, biomarker, cerebrovascular events, vascular risk factors

Received: May 21, 2025; Revised: September 12, 2025; Accepted: September 22, 2025; Available online: October, 15, 2025
https://doi.org/10.5507/bp.2025.028

[1]*Department of Neurology, University Hospital Pilsen, Pilsen, Czech Republic*
[2]*Faculty of Medicine in Pilsen, Charles University, Pilsen, Czech Republic*
[3]*Department of Immunochemical Diagnostics, University Hospital Pilsen, Pilsen, Czech Republic*
Corresponding author: Kamila Smolikova, e-mail: smolikovak@fnplzen.cz

INTRODUCTION

Arginine vasopressin (AVP, vasopressin, antidiuretic hormone), released from the neurohypophysis, is one of the essential hormones of the hypothalamic-pituitary-adrenal (HPA) axis, which is activated in response to stressful events. Its role in maintaining the balance of the cardiovascular system is important in that it regulates vascular tone and fluid homeostasis. Copeptin itself, as a 39-amino acid peptide, represents the C-terminal portion of pre-

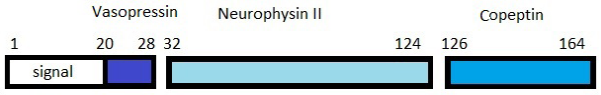


Fig. 1. Illustration of pre-pro-vasopressin[2].
Signal, signal sequence; Vasopressin (dark blue), arginin vasopressin, or AVP; Neurophysin II (pale blue); Copeptin (light blue) as the C-terminal segment of pro-AVP; The numbers represent the number of amino acids in the protein

pro-AVP, which is proteolytically cleaved during axonal transport into vasopressin, neurophysin II, and copeptin (Fig. 1.) (ref.[1]).

AVP causes arteriolar vasoconstriction via the V1 receptor and, via the V2 receptor, has an antidiuretic effect in the kidneys, which can lead to hypovolemic or normovolemic hyponatremia and low plasma osmolality. V3 receptors are mainly found in the central nervous system, and their stimulation regulates corticotropic secretion and thus participates in the secretion of adrenocorticotropic hormone (ACTH) and cortisol[3,4].

Copeptin is then secreted in equimolar proportions with AVP and, in the event of a synergistic effect with corticoliberin on the secretion of adrenocorticotropic hormone (ACTH) and cortisol, the level of copeptin would then also reflect the degree of stress[5,6].

When determining levels, copeptin itself is comparable to AVP and cortisol. It is more stable, with stability at room temperature lasting around 1–2 weeks, and has a longer half-life. In addition, cortisol is adversely affected by circadian rhythms and dynamics during stress responses, and serum AVP is bound to blood platelets by around 90% (ref.[7-9]). The results are available within an hour, making it a useful biomarker in emergency care as a prognostic factor in patients with acute illnesses such as lower respiratory tract infections, cardiovascular diseases, and cerebrovascular events (Table 1) (ref.[2]).

Copeptin in acute ischemic stroke

Stroke remains a major issue, and despite significant improvements in diagnosis and treatment in recent years, more attention is being paid to prognosis and prediction of clinical outcome. Copeptin, as a surrogate for AVP, has become one of the biomarkers with promising potential as a prognostic and predictive biomarker for acute stroke due to the role of AVP in the exacerbation of inflammatory responses, ion and neurotransmitter dysfunction, and thus the development of cerebral edema with a consequent increase in intracranial pressure[11].

Oraby et al. (2021) processed data from 90 individuals, 45 patients with first acute ischemic stroke with onset of symptoms within 24 hours and 45 healthy controls. Significantly higher levels of copeptin were found in patients with acute ischemic stroke (P=0.001) compared to healthy individuals. Significantly elevated copeptin levels were also found in patients with severe stroke assessed according to the NIHSS scale > 16 or mRS 3–6 than in patients with mild to moderate stroke (NIHSS 0–15 or mRS 0–2) (ref.[11]). This finding is supported by other studies by Zenga et al., Dong et al., and Katana et al.[10,16,17].

One of the more recent studies, Montellano et al. (2024), looked at the possibility of improving the prediction of functional scores after ischemic stroke by adding various biomarkers, including copeptin, to established clinical scores (e.g., ASTRAL, NIHSS). Copeptin was confirmed as a predictor of functional outcome and mortality in patients after ischemic stroke[18].

Oraby et al. also examined the effect of thrombolytic therapy (IVT) using rTPA in acute ischemic stroke on copeptin levels, which were significantly lower in patients treated with IVT (P=0.049). Low copeptin levels are thought to be associated with a lack of worsening or progression of the ischemic lesion and improvement in neurological deficits after IVT treatment[19].

A 2024 publication, (Vasile et al.) found a correlation between copeptin levels and the efficacy of thrombolytic therapy and this could be used to stratify patients before deciding on the treatment[20].

It was also interesting to find a positive correlation between copeptin levels and the initial volume of infarction measured by CT or MRI of the brain, which can be interpreted as a connection between serum copeptin

Table 1. Use of copeptin as a biomarker in emergency care for selected cerebrovascular events (CVE).

Type of cerebrovascular event	Predicted result	Statistical data	Significance of copeptin	Ref.
Acute ischemic stroke	90-day mortality	AUC=0.82	Predictive marker	10
	Functional outcome (mRS ≥ 3)	AUC=0.73	Good prediction of adverse outcome	10
	mRS after 3 months	P<0.001	Significantly higher level in mRS 3–6	11
Hemorrhagic stroke (intracerebral hemorrhage)	NIHSS and mRS correlation	Not quantified	Significant correlation with clinical severity	12
	30-day mortality	AUC=0.88	Predictive marker	13
	90-day functional outcome	AUC=0.68	Predictive marker	13
Subarachnoid hemorrhage	Patients with SAH vs. healthy controls	P<0.001	Diagnostic marker	14
TIA (transient ischemic attack)	Recurrence of stroke within 90 days	AUC=0.73	Higher level in patients with subsequent stroke	15

Inspired by the table by Nickel et al.[2]

levels and cerebral edema, which develops shortly after the onset of focal ischemia[21].

A study by Vasile et al. published this year, analyzed 29 professional articles with the aim of expanding the use of copeptin as a differential diagnostic marker in distinguishing acute stroke from conditions that only mimic it. Although the authors point out that further studies are needed, they confirm the possible role of copeptin in distinguishing between subtypes of stroke and conditions mimicking stroke. This study has added another important role for copeptin in acute neurology[22].

However, it is important to comment on the timing of blood sampling for the determination of copeptin and thus its diagnostic and prognostic value. According to Spagnolello et al., copeptin levels are highest on admission of patients with acute ischemic stroke (median 50.71 pmol/L) and decrease significantly during the following 24 h. To avoid false negative results in late sampling, the authors recommend sampling as early as possible, ideally in the early window, to capture the maximum prognostic value of the biomarker[23].

This dynamic supports the use of copeptin as an early biomarker in acute conditions. If serial measurements were considered, they could be useful in cases of unclear time of onset of stroke symptoms, monitoring the development of stress response, or complications.

Copeptin measurement can significantly influence treatment algorithms in patients with acute stroke. According to Vasile et al., it can not only predict the occurrence of ischemic stroke itself, but also help predict the effectiveness of recanalization therapy and identify patients at higher risk of complications[20].

Karatzetzou et al. recommend its inclusion in decision-making algorithms for individualized care, as in previous studies[24].

In an emergency setting, according to Nickel et al., it can serve as a tool for rapid risk stratification, where copeptin measurement can speed up decision-making in acute conditions such as stroke[2].

Copeptin as a predictive biomarker for recurrent cerebrovascular events

Katan et al. conducted a prospective study involving 107 patients with transient ischemic attack. At the beginning of the study, all patients were assigned an ABCD2 score and their copeptin and cortisol levels were measured in order to identify a more reliable prognostic biomarker for stratifying the risk of recurrent cerebrovascular events (TIA or ischemic stroke) within 90 days of the first event. This study demonstrated the importance of copeptin as a prognostic marker, unlike cortisol, as nine percent of patients with elevated baseline copeptin levels experienced recurrence. Cortisol was not a more suitable prognostic marker. Copeptin showed a higher area under the curve (AUC) for predicting re-events compared to the ABCD2 score (AUC 0.73 vs 0.43; $P<0.01$) and showed better prognostic accuracy (AUC of the combined model 0.77; $P=0.002$) (ref.[15]).

A prospective multicenter study involving 302 patients with TIA from three stroke centers in Switzerland and Germany, similar to previous studies, investigated copeptin as a predictive factor for recurrent cerebrovascular events within three months after TIA. It also focused on the correlation between copeptin levels and ABCD2 and ABCD3-I scores. The association with recurrent cerebrovascular events (TIA or ischemic stroke) 3 months after the first event was not significant, but significantly higher copeptin levels were measured in patients with stroke within 3 months of TIA. After adding copeptin to the ABCD2 score, the area under the ABCD2 score curve improved from 0.60 to 0.74, thereby improving the prognostic value of the ABCD2 score for predicting stroke[25].

A meta-analysis by Xu et al. involving patients with acute stroke and TIA also demonstrated that higher levels of copeptin are significantly associated with stroke recurrence within 90 days ($P<0.001$), independently of other risk factors[26].

Copeptin limits

Like most biomarkers, copeptin is beset with issues of interpretation.

In the following studies, copeptin levels are reported either in molar units (pmol/L) or in mass units (pg/mL), with 1 pmol/L corresponding to approximately 5 pg/mL, based on the molecular weight of copeptin reported in the scientific literature, which is 5 kDa (ref.[27]).

In healthy individuals, serum copeptin levels are determined to be between 1 and 12 pmol/L (ref.[28]). Blek et al., found the average level of copeptin in patients with CVE was 19.8 ± 17.4 pmol/L, compared to 9.7 ± 6.6 pmol/L in healthy controls. The study also reports the copepin level in patients with unfavorable CVE progression (mRS 3–6), which was 29.4 ± 14.5 pmol/L, while in patients with favorable progression (mRS 0–2) it was 12.0 ± 3.6 pmol/L (ref.[29]).

Zhou et al. report that copeptin levels in patients with stroke correlate with NIHSS and mRS clinical scores, with values as high as 44.66 ng/mL (ref.[12]).

The cited study by Oraby et al. reports values in patients with acute ischemic stroke of 120.52 ± 45.7 pg/mL, compared to healthy controls, in whom lower values of 76.51 ± 32.8 pg/mL were recorded. In patients with severe stroke (NIHSS > 16, mRS 3-6), the average value was 139.45 ± 38.7 pg/mL, while in patients with mild to moderate stroke (NIHSS 0-15 or mRS 0-2) 95.47 ± 40.3 pg/mL (ref.[11]).

Copeptin levels may also signal various complications of stroke, such as pneumonia, early neurological deterioration, or delayed cerebral ischemia[24].

However, there is still no uniform reference value for copepin levels in patients with stroke, which limits its application in practice.

Copeptin levels vary between the genders, with higher levels in men than in women. Reduced copeptin clearance may be caused by reduced glomerular filtration rate in the male population[30].

Higher levels of copeptin were also found in patients with arterial hypertension, diabetes mellitus (especially type 2), dyslipidemia, and higher BMI. Studies differ on the effect of age on copeptin levels. Studies by Molnar et

al. and Oraby et al. show a statistically significant positive correlation with patient age[31,11]. On the other hand, Wang and his colleagues found no correlation between copeptin levels and different age groups[32].

Available studies do not indicate that copeptin levels are affected by food intake, but even small amounts of oral fluid intake can significantly reduce its levels[33].

These interindividual variations may complicate the use of copeptin as a universal prognostic tool. Furthermore, it is important to note that copeptin reflects activation of the stress axis, meaning that its levels may also be elevated in other acute conditions, which may reduce its specificity for CVE.

CONCLUSION

The stability of copeptin, its rapid response to stress, and its correlation with disease severity make it an important biomarker in medicine, particularly in the field of cerebrovascular disease. Its presence in individuals exposed to significant or prolonged stress can be assessed as a risk factor, and primary prevention can focus on procedures that reduce stress levels. In an existing stroke, it can be used as one of the prognostic and predictive biomarkers involved in the course of the stroke and its clinical outcome.

Search strategy and selection criteria

The search for professional studies published between 1983 and 2025 was conducted from December 2024 to July 2025 primarily in international online bibliographic databases supporting scientific and medical research, such as Pubmed, BBC Medicine, and Science Direct. The Czech portal AIM Journal was an exception. Specifically, the terms "copeptin," "cerebrovascular events," "vascular risk factors," and "biomarker" were searched. The aim of this short review was to familiarize readers with a promising prognostic and predictive biomarker of cerebrovascular accidents – the glycopeptide copeptin – with a view to improving primary prevention and prediction of cerebrovascular accidents. Its physiology, role in cerebrovascular accidents, limitations, and influence of vascular risk factors were discussed.

ABBREVIATIONS

ABCD2 score, Prognostic scoring system for estimating the risk of CVA recurrence after TIA; ABCD3-I score, Extended version of the ABCD2 score including imaging findings and history of recurrent TIAs, thereby increasing the accuracy of prediction; ACTH, Adrenocorticotropic hormone; ASTRAL score, A tool used to predict the outcome of acute ischemic stroke; AVP, Arginine vasopressin, vasopressin; AUC, Area under the curve; BMI, Body Mass Index; CVE, Cerebrovascular events; GCS, Glagow Coma Scale; HPA, Hypothalamic-pituitary-adrenal axis; IVT, Intravenous thrombolysis; mRS, Modified Rankin Scale; NIHSS, National Institute of Health Stroke Scale; *P,* statistical data, the probability that the observed difference could have arisen by chance alone; rTPA, Recombinant tissue plasminogen activator; TIA, Transient ischemic attack.

Acknowledgement: Supported by the grant of Ministry of Health of the Czech Republic – Conceptual Development of Research Organization (Faculty Hospital Pilsen – FNPl, 00669806).

Author contribution: All authors contributed equally to preparing the manuscript.

Conflict of interest statement: None declared.

REFERENCES

1. Repaske DR, Medlej R, Gültekin EK, Krishnamani MR, Halaby G, Findling JW, Phillips JA. Heterogenity in clinical manifestation of autosomal dominant neurohypophyseal diabetes insipidus caused by a mutation encoding Ala-1-->Val in the signal peptide of the arginine vasopressin/neurophysin II/copeptin precursor. JCEM 1997;82(1):51-6. doi: 10.1210/jcem.82.1.3660
2. Nickel CH, Bingisser R, Morgenthaler NG. The role of copeptin as a diagnostic and prognostic biomarker for risk stratification in the emergency department. BMC Med 2012;10:7. doi: 10.1186/1741-7015-10-7
3. Thibonnier M, Preston JA, Dulin N, Wilkins PL, Berti-Mattera LN, Mattera R. The human V3 pituitary vasopressin receptor: ligand binding profile and density-dependent signaling pathways. Endocrinology 1997;138(10):4109-22. doi: 10.1210/en.138.10.4109
4. Balík M, Černý V, Šrámek V, Matějovič M, Kunstýř J. Vasopresin a jeho role v anesteziologii a intenzivní medicíně. Anest Intensiv Med 2015;26(4):223-7. (In Czech)
5. Rivier C, Vale W. Interaction of corticotropin-releasing factor and arginine vasopressin on adrenocorticotropin secretion in vivo. Endocrinology 1983;113(3):939-42. doi: 10.1210/endo-113-3-939
6. Milsom SR, Conaglen JV, Donald RA, Espiner EA, Nicholls MG, Livesey JH. Augmentation of the response to CRF in man: relative contributions of endogenous angiotensin and vasopressin. Clin Endocrinol (Oxford) 1985;22(5):623-9. doi: 10.1111/j.1365-2265.1985.tb02998.x
7. Katan M, Morgenthaler N, Widmer I, Puder JJ, Konig C, Muller B, Christ-Crain M. Copeptin, a stable peptide derived from the vasopressin precursor, correlates with the individual stress level. Neuro Endocrinol Lett 2008;29(3):341-6.cited 12/2024.PMID:18580851
8. Morgenthaler NG, Struck J, Alonso C, Bergmann A. Assay for the measurement of copeptin, a stable peptide derived from the precursor of vasopressin. Clin Chem 2006;52(1):112-9. doi: 10.1373/clinchem.2005.060038
9. Abdelmageed M, Güzelgül F. Copeptin: Up-to-date diagnostic and prognostic role highlight. Anal Biochem 2023;673:115181. doi: 10.1016/j.ab.2023.115181
10. Katan M, Fluri F, Morgenthaler NG, Schuetz P, Zweifel C, Bingisser R, Muller K, Meckel S, Gass A, Kappos L, Steck AJ, Engelter ST, Muller B, Christ-Crain M. Copeptin: a novel, independent prognostic marker in patients with ischemic stroke. Ann Neurol 2009;66(6):799-808. doi: 10.1002/ana.21783
11. Oraby MI, Soliman RH, Abd Elkareem RM, Mohammed AI. Copeptin: a potential blood biomarker for acute ischemic stroke. Egypt J Neurol Psychiatry Neurosurg 2021;57:40. doi: 10.1186/s41983-021-00393-2
12. Zhou Z, Sun F, Jiang J, Dou Z, Lv Y, Zhang Z, Zhou X, Zhao X, Qin Y, Huang B. Establishment and Clinical Application in Stroke of a Serum Copeptin Time-Resolved Fluorescence Immunoassay. J Fluoresc 2024;34:2349-58. doi: 10.1007/s10895-023-03441-7
13. Zweifel C, Katan M, Schuetz P, Siegemund M, Morgenthaler NG, Merlo A, Mueller B, Christ-Crain M. Copeptin is associated with mortality and outcome in patients with acute intracerebral hemorrhage. BMC Neurol 2010;10:34. doi: 10.1186/1471-2377-10-34
14. Ozkan S, Baykan N, Salt O, Yakar S, Sen A, Durukan P. The role of plasma copeptin level in determining the severity and mortality of

subarachnoid hemorrhage. Acta Clinica Croatica 2023;62:595-604. doi: 10.20471/acc.2023.62.04.3
15. Katan M, Nigro N, Fluri F, Schuetz P, Morgenthaler NG, Jax F, Meckel S, Gass A, Bingisser R, Steck A, Kappos L, Engelter S, Muller B, Christ-Crain M. Stress hormones predict cerebrovascular re-events after transient ischemic attacks. Neurology 2011;76(6):563-6. doi: 10.1212/WNL.0b013e31820b75e6
16. Zeng X, Deng A, Ding Y, Ni Y, Xu T, Liu J, Yang B, Goa M, Ma G, Ding W, Mu L. Copeptin and NT-proBNP as prognostic markers for recurrent ischemic cerebrovascular events in ischemic stroke patients. J Am Coll Cardiol 2016;68(24):2710-11. doi: 10.1016/j.jacc.2016.09.954
17. Dong X, Tao DB, Wang YX, Cao H, Xu YS, Wang QY. Plasma copeptin levels in Chinese patients with acute ischemic stroke: a preliminary study. Neurol Sci 2013;34(9):1591-5. doi: 10.1007/s10072-013-1291-2
18. Montellano FA, Rücer V, Ungethüm K, Penalba A, Hotter B, Giralt M, Wiedmann S, Mackenrodt D, Morbach C, Frantz S, Störk S, Whiteley WN, Kleinschnitz C, Meisel A, Montaner J, Haeusler KG, Heuschmann PU. Biomarkers to improve functional outcome prediction after ischemic stroke: Results from the SICFAIL, STRAWINSKI, and PREDICT studies. Eur Stroke J 2024;9(4):968-80. doi: 10.1177/23969873241250272
19. Furlanis G, G, Ajčević M, Buoite Stella A, Cillotto T, Caruso P, Ridolfi M, Cova MA, Naccarato M, Manganotti P. Wake-up stroke: thrombolysis reduces ischemic lesion volume and neurological deficit. J Neurol 2020;267(3):666-73. doi: 10.1007/s00415-019-09603-7
20. Vasile AI, Tiu C, Badiu C. Copeptin as biomarker for acute ischemic stroke prognosis and revascularization treatment efficacy. Front Neurol 2024;15:1447355. doi: 10.3389/fneur.2024.1447355
21. Choi KS, Kim HJ, Chun HJ, Kim JM, Yi HJ, Cheong JH, Kim ChH, Oh SJ, Ko Y, Kim YS, Bak KH, Ryu JI, Kim W, Lim T, Ahn HS, Ahn IM, Lee SH. Prognostic role of copeptin after stroke: a systematic review and meta-analysis of observational studies. Sci Rep 2015;5:11665. doi: 10.1038/srep11665
22. Vasile AI, Tută S, Tiu C, Badiu C. Copeptin as a new blood-based marker for differential diagnosis in stroke patients. Int J Mol Sci 2025;26(11):5328. doi:10.3390/ijms26115328
23. Spagnolello O, De Michele M, Lorenzano S, Cerulli Irelli E, Naitana F, Falcou A, Letteri F, Bachetoni A, Collepardo D, Bertazzoni G, Toni D. Copeptin Kinetics in Acute Ischemic Stroke May Differ According To Revascularization Strategies, Pilot Data. Stroke 2019;50:3632-5. doi: 10.1161/STROKEAHA.119.025433
24. Karatzetzou S, Tsiptsios D, Sousanidou A, Fotiadou S, Christidi F, Kokkotis C, Gkantzios A, Stefas E, Vlotinou P, Kaltsatou A, Aggelousis N, Vadikolias K. Copeptin implementation in stroke prognosis. Neurol Int 2023;15(1):83-99. doi: 10.3390/neurolint15010008
25. De Marchis GM, Weck A, Audebert H, Benik S, Foerch C, Buhl D, Schuetz P, Jung S, Seiler M, Morgenthaler NG, Mattle HP, Mueller B, Chris-Crain M, Arnold M, Katan M. Copeptin for the prediction of recurrent cerebrovascular events after transient ischemic attack: results from the CoRisk study. Stroke 2014;45(10):2918-23. doi: 10.1161/STROKEAHA.114.005584
26. Xu Q, Tian Y, Peng H, Li H. Copeptin as a biomarker for prediction of prognosis of acute ischemic stroke and transient ischemic attack: a meta-analysis. Hypertens Res 2017;40:465-71. doi: 10.1038/hr.2016.165
27. Suh SH, Kim SW. A novel role of copeptin as a biomarker for the prediction of treatment response in hyponatremia. Kidney Res Clin Pract 2021;40(3):329-31. doi: 10.23876/j.krcp.21.000
28. Morgenthaler NG, Struck J, Jochberger S, Dünser MW. Copeptin: clinical use of a new biomarker. Trends Endocrinol Metab 2008;19(2):43-9. doi: 10.1016/j.tem.2007.11.001
29. Blek N, Szwed P, Putowska P, Nowicka A, Drela WL, Gasecka A, Ladny JR, Merza Y, Jaguszewski MJ, Szarpak L. The diagnostic and prognostic value of copeptin in patients with acute ischemic stroke and transient ischemic attack: A systematic review and meta-analysis. Cardiol J 2022;29(4):610-18. doi: 10.5603/CJ.a2022.004
30. Bhandari SS, Loke I, Davies JE, Squire IB, Struck J, Ng LL. Gender and renal function influence plasma levels of copeptin in healthy individuals. Clin Sci (Lond) 2009;116(3):257-63. doi: 10.1042/CS20080140
31. Molnár AH, Varga C, Berkó A, Rojik I, Párducz A, László F, László FA. Inhibitory effect of vasopressin receptor antagonist OPC-31260 on experimental brain oedema induced by global cerebral ischaemia. Acta Neurochir (Wien) 2008;150(3):265-71. doi: 10.1007/s00701-007-1400-1
32. Wang CB, Zong M, Lu SQ, Tian Z. Plasma copeptin and functional outcome in patients with ischemic stroke and type 2 diabetes. J Diabetes Complications 2016;30(8):1532-6. doi: 10.1016/j.jdiacomp.2016.07.030
33. Choy KW, Wijeratne N, Chiang C, Don-Wauchope A. Copeptin as a surrogate marker for arginine vasopressin: analytical insights, current utility, and emerging applications. Crit Rev Clin Lab Sci 2024;62(1):24-44. doi: 10.1080/10408363.2024.2383899