European Journal of Clinical Pharmacy
Print ISSN: 2385-409X **Online ISSN**: Applied Website:
https://farmclin.com/

LACK OF BLOOD-BRAIN BARRIER AT THE AREA POSTREMA. NOVEL IMPLICATIONS

Lourdes de Fátima Ibañez Valdés1, Humberto Foyaca Sibat2
1MD, MSc, FRCP, Sen Lec, Ag Res Inv.
Division of Neurology. Nelson Mandela Academic Hospital. Walter Sisulu University. Mthatha, South Africa.
2 MD, MSc, PhD, Full Professor, FCP, FRCP, FResInv,
Division of Neurology. Nelson Mandela Academic Hospital. Walter Sisulu University. Mthatha, South Africa.
CORRESPONDING AUTHOR: humbertofoyacasibat@gmail.com

ABSTRACT

Introduction:

Objectives: Main goal of this investigation was to search for last information regarding to lack of BBB at the are postrema.

Methods: We performed a searching of the following database PubMed/MEDLINE, Scopus, and Embase databases looking for publications related to novel information on APS including new diagnostic procedures and novel therapeutic approaches.

We searched the literature from January 01, 1989, to January 30th, 2026, following the PRISMA guidelines. We review the following the before cited databases looking for the following issues: "composition of the area postrema" OR " area postrema syndrome " OR " refractory nausea and vomiting " OR " blood-brain barrier " OR " intractable nausea, hiccups and vomiting ", OR " pathophysiology of APS " OR “lack of BBB at the AP”.

Results: After searching literature, we retrieved 149 articles. One hundred and one duplicated publications identified and after reviewing the titles and abstracts removed, forty-eight publications selected. Applying the inclusion/exclusion criteria forty articles excluded; therefore, eight studies mentioned the function of the BBB in their text body, but none comment on the implication of lack of BBB at the AP.

Conclusions: We did not find published investigations about the implications of lack of BBB at the AP. We hypothesized that the free passage of the autoantibodies across the fenestrate CVs may cause neuronal dysfunction in the AP leading to the comorbidity of APS and NMOSD, being it the main implication of lack of BBB at the AP

KEYWORDS: Area postrema, area postrema syndrome, blood-brain-barrier, refractory nausea, and uncontrolled vomiting, neuromyelitis optica spectrum disorder.

How to Cite: Lourdes de Fátima Ibañez Valdés, Humberto Foyaca Sibat, (2026) LACK OF BLOOD-BRAIN BARRIER AT THE AREA POSTREMA. NOVEL IMPLICATIONS, European Journal of Clinical Pharmacy, Vol.8, No.1, pp. 3442-3450.

INTRODUCTION

It is well-known that the blood–brain barrier (BBB) is a well-structured and semipermeable located between the brain tissue and capillary vessels, highly specialized in controlling the passage of toxic substances, nutrients, blood cells, metabolites, drugs, and other molecules into the brain [1].

The BBB participates in central nervous system (CNS) homeostasis and in preserving the brain's healthy network, and its disruption impairs the ability to filter neurotoxic pathogens and harmful molecules, leading to neuroinflammation and neurodegeneration [2-5].

Last year, Gori and La Barbera published an article on therapy against Neurodegenerative Diseases, in which they placed the BBB at the centre of using nanoparticles to pass across the BBB targeting the brain as part of AD therapy [6].

Recently, Li et al. reported a pathogenic relationship between recurrent unprovoked epileptic seizures and gut microbiota via the microbiota-gut-brain axis, after investigating whether Lachnospira eligens could decrease ictal activity by modulating microbiota gut-brain axis (MGBa), focusing on BBB integrity, intestinal barrier ipermeability, and neuroimmune responses. These investigators concluded that L. eligens diminishes the ictal activity by suppressing neuroinflammation and reestablishing intestinal barrier and BBB normal function [7].

Magnus Gustaf Retzius in 1896 described the area postrema (AP) for the first time. He reported it as an anatomical area located at the posterior region of the medulla oblongata, at the caudal aspect of the IV ventricle. The AP is considered as a circumventricular organ overlying the inferior region of vagal trigone while facing the foramen of Magendie and rostral to the obex its main role is modulate regulation nausea and vomiting, supported by its unique BBB permeability and remarkably large connectivity [8].

The AP contains fenestrate capillary vessels (CV) with great permeability and sensory neurons cells able to identify all circulating chemical material travelling through the blood flow and transducing all of them into the neural network. Because of

the anatomical location of AP close to the nucleus of the tractus solitarius of the vagus nerve bilaterally, it work as a sensory transducer, modulating all the blood-to-brain autonomic functions, including the detection of neurohormones involved in the pathophysiology of several neurophysiological function such as hunger, vomiting, thirst, and the control of arterial blood pressure. The AP is linked to a medical condition known as area postrema syndrome (APS), which is characterized by unexplained episodes of intractable singultus, nausea, and vomiting [9].

The AP has the capacity to prevent the mechanism of vomiting as a side effect from many emetic therapeutic drugs; however it has not the same capacity to control vomiting related to motion or afferent inputs from the vagal nerve or secondary to radiation-induced vomiting. On the other hand, AP may trigger nausea and vomiting by sending electrical input to the nucleus tractus solitarius [10]. On the other side, the total amount of blood flow received by the AP is extremely large compared with the nearby cerebral areas. An on top of that the speed of local circulation of its blood components is remarkable slow, allowing to a higher increased capacity to control the passage of almost all circulating elements through the membrane including neurotransmitters and neurohormones [11].

In 2024, other investigators proved that the dorsal vagal complex (DVCX) in the brainstem acts as a modulatory centre for energy and glucose homeostasis by modulating nutrient changes and hormonal elements [12].

In 2023, Zhang et al. documented that metformin increases kidney GDF15 synthesis, elevating plasma GDF15 levels, and induces GFRAL expression in the AP to diminish weight and feeding rodents [13].

Alexander disease (leukodystrophy) sometimes is associated with intractable vomiting, decreased appetite, poor weight gain and lesions in the AP and adjacent nucleus tractus solitarius (NTS) [14].

Furube and colleagues documented the role played by the neural stem cells (NSCs) in the subgranular and subventricular zones, which exhibit limited regenerative capacity following brain damage. They studied the medio-basal hypothalamic tanycyte, plus the the median eminence, arcuate nucleus, and medulla oblongata. They confirmed the presence of neural progenitor cells (NPCs) and NSCs with gliogenic potential and context-dependent neurogenic in both. Furthermore, they demonstrated that after hypothalamic neuronal damage, the MBH neural circuits have a regenerative capacity to reorganize the neuronal network and reported 2 types of NSCs: tanycyte-like NSCs and astrocyte-like NSCs in the central canal facing the CSF in the AP. The same investigators confirmed active proliferation in astrocyte-like NSCs, and neuronal apoptosis can be induced by monosodium glutamate selectively in the AP. Nevertheless, NPCs can proliferate and differentiate into mature neurons, leading to almost complete restoration of neuronal density.

Demyelination in the AP secondary to experimental autoimmune encephalomyelitis is alleviated by NSCs, which restore oligodendrocyte density. Recent investigations suggest MBH and medulla oblongata show a context-dependent regenerative responses, leading to neuroplasticity with the replacement of neurons and oligodendrocytes following brain damage [15].

Area postrema syndrome typically can be associated with other infectious, inflammatory, autoimmune and demyelinating diseases. Other associations with APS can be negative AQP4, MOG, GFAP antibody related to autoimmune encephalitis [16].

Last year, Muñoz-Zúñiga and collaborators reported a comorbidity of SLE and APS for the first time [17].

Recently, other investigators reported a 63-year-old lady presenting with an APS secondary medullary ischemic stroke leading to intractable nausea, vomiting and hiccups without focal neurological signs [18].

Thapa and collaborators reported an 11-year-old Nepalese girl presenting persistent vomiting, facial asymmetry and progressive limb weakness. Her brain MRI confirmed T2 hyperintensities involving the AP, and serum anti-AQP4 antibody confirmed NMOSD, which responded quite well to Polygam (IVIG) and oral steroids, with progressive physical recovering of her neurological capacities [19].

Courtney et al. reported a case presenting APS with several watershed cerebellar ischemic strokes and a medullary ictus related to giant cell arteritis, responding remarkably well to prednisone, tocilizumab, and IL-6 inhibitor with complete recovery [20].

Recently, Li and Teng reported that APS is a condition related to autoimmune glial fibrillary acidic protein astrocytopathy [21].

While, Koc et al studied a series of forty-six cases presenting mediated by autoantibodies against Aquaporin-4 (AQP4-IgG–positive) NMOSD, assessing the effectiveness and safety of eculizumab, and identifying predictors of disability outcomes. They confirmed that patients presenting with area postrema syndrome (APS) had a better prognosis comparable with cases suffering from spinal attack who had a worse outcome [22].

Just a few weeks ago, Terahara et al. reported a 53-year-old Japanese lady presenting facial nerve palsy and right lateral gaze palsy and, with preservation of convergence and left lateral gaze due to paramedian pontine reticular formation syndrome associated with medial longitudinal fasciculus syndrome and positive antinuclear and anti-AQP4 antibodies, NMOSD without involvement of the AP [23].

METHODOLOGY

We performed a large searching of the literature of the following database PubMed/MEDLINE, Scopus, and Embase databases looking for the implications on lack of BBB at the AP.

We searched for all publications released from 01st, January 1989 to 30th, January of 2026, following the PRISMA guidelines (2020 statement). We investigate the following issues: "area postrema" OR " area postrema syndrome " OR " refractory nausea and vomiting " OR " blood-brain barrier " OR " refractory nausea, hiccups and vomiting ", OR " pathophysiology of APS ", OR “neuromyelitis optica spectrum disorder”

- **Search strategy**

Only articles published in English were selected. While editorials, preclinical studies, and conference proceedings excluded.

- **Study selection criteria**

We reviewed the abstracts and titles of the selected publications and independently reviewed the full-text versions to establish their eligibility. Clinical investigations and therapeutic procedures on AP were selected for inclusion. Moreover, manuscripts without a clear confirmatory diagnosis, duplicate papers, article with poor analysis, with incomplete data were excluded.

- **Criteria for selection**

Articles related to implications on lack of BBB at the AP were chosen with priority.
We applied the next exclusion criteria: (1) Lack of accessibility to the full text; (2) irrelevant clinicopathological information; (3) animal model studies; (4) publication no written in Spanish/Portuguese/English (5) studies without discussing the role of lack of BBB in the AP.

- **and Extraction assessment and quality of date**

We applied the requirements from the National Institutes of Health criteria and the Quality Assessment of Diagnostic Accuracy Studies version 2 (QUADAS-2) evaluation to assess the risk of bias. Both investigators made the quality evaluation independently and when some disagreements raised, we resolved it by scientific discussion until reach a final agreement.

- **Data Collection, Extraction, and Bias Assessment.**

All abstracts and titles with the inclusion criteria were revised by two before cited authors to collect relevant information for the review. For each publication selected in the review, data concerning to author’s name, age, year of publication, country of information’ source. The selected information was introduced into an Excel program.

- **Literature Search**

Searching literature, we retrieved a total of 149 studies. One hundred and one duplicated publications were removed, and forty-eight publications selected. After applying the inclusion/exclusion criteria then forty articles were finally move out; therefore, eight studies mentioned the function of the BBB in their text body, but no article discussed the implication of lack of BBB at the AP.

To provide an accurate and confident management of this process, we introduced a QUADAS-2 evaluation to identify low/moderate risk of bias.

COMMENTS AND FINAL REMARKS

In this extensive comprehensive review, we did not identify any publication related to the implication of lack of BBB at the AP as is shown in FIGURE 1.

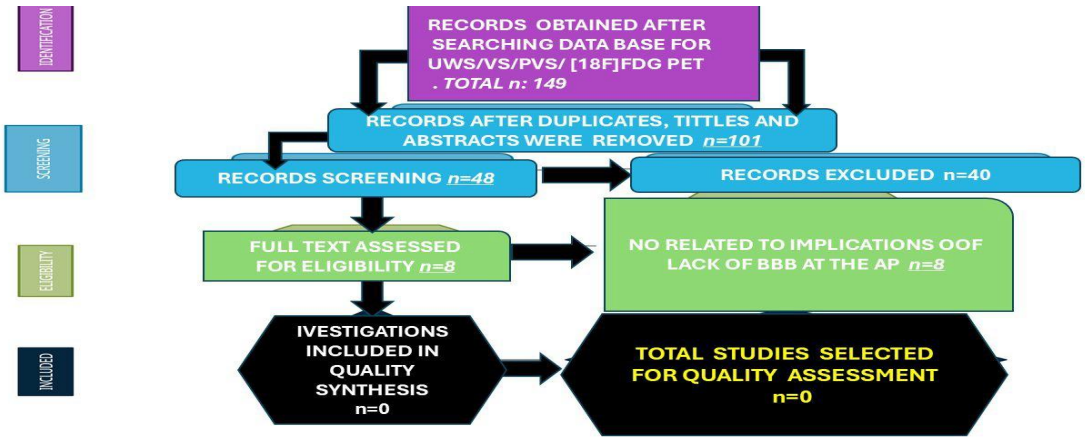

FIGURE 1: PRISMA. Flow diagram with included publications.

Brief comments about the structure and function of AP.

The AP accomplishes its function of sensing baroreceptor (blood-borne) signals from the aorta and the carotid sinus, as well as signals from the liver via osmoreceptors. The lack of TJ between EC, the foot processes of astrocytes, and the composition of fenestrated CV, aid in physiologic signalling with neuron cells [23, 24]. Below,

we show a cross section of the inferior aspect of the brainstem at the level of the AP where it histological composition is observed.

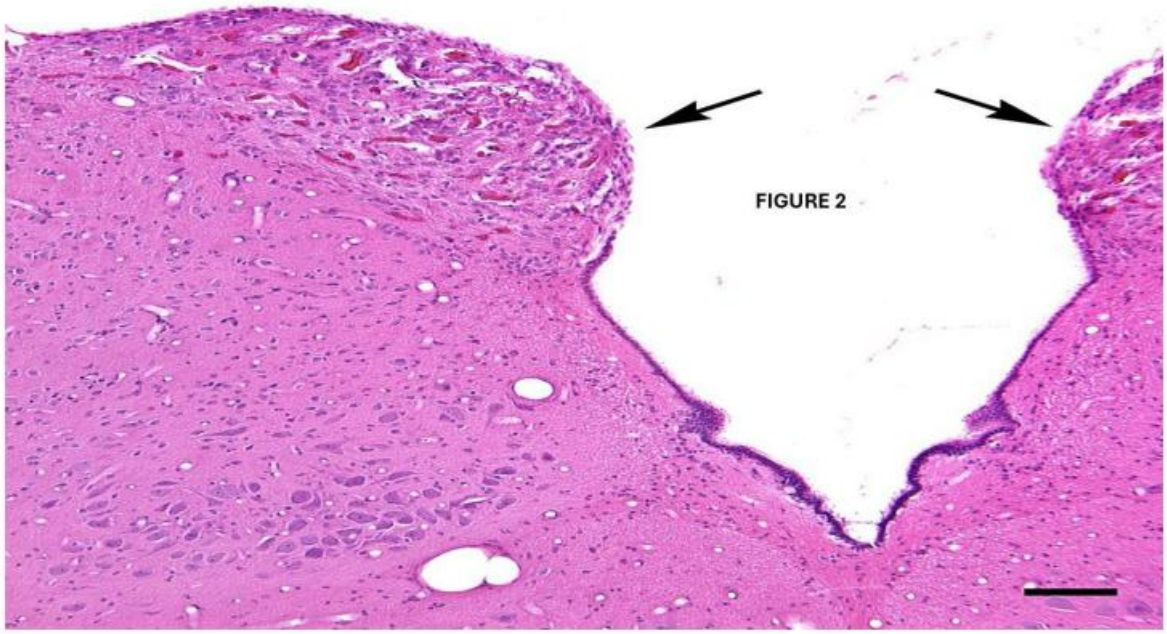

FIGURE 2 Micrograph of the AP in the caudal brainstem of a squirrel monkey (Saimiri sciureus). Haematoxylin and eosin stain. The two black arrows show the AP at the floor of the IV ventricle.

It modulates effect of angiotensin II, increasing BP without affecting HR. Furthermore, neuron cells in the AP receive signal from the stomach through stretch receptors to incorporate mechanical information; it supports somatic growth, which stimulates the physiological mechanism of appetite. [https://www.statpearls.com].

AP is located close the NTS, and its afferent signal arrives from the cranial nerve IX, X, and nucleus of the hypothalamus. Furthermore, AP also receives afferent information from the carotid sinus and aortic depressor nerves. At the same time, some efferent fibres including those from the NTS, dorsolateral tegmental nuclei, the ventral lateral medulla, parabrachial nucleus, locus coeruleus, and pericentral dorsal tegmental [23, 25].

The AP provide an indirect control on the smooth muscles involved in vomiting, feeding, metabolism, and the heart. Therefore, after sending inputs to NTS, distinct neural outputs are directed to AP for modulation of effector responses of vomiting including cardiovascular, respiration, swallowing, salivation, and gastrointestinal in a structured manner including external intercostal muscles contraction and internal intercostal muscles; muscle contraction of the diaphragm through all abdominal muscles, oesophageal contraction; relaxation of oesophageal sphincter; gastric dysrhythmia; and duodenal retro-peristaltic activity in stomach and duodenum [25]

Brief comments on the BBB

The BBB is present in almost all microvasculature of the CNS, being the largest labyrinthine structure of cyclopean complexity. And its surface is around 350,000 m2 in a 1400 g brain. The main CNS anatomical structures where the BBB is absent listed in FIGURE 3.

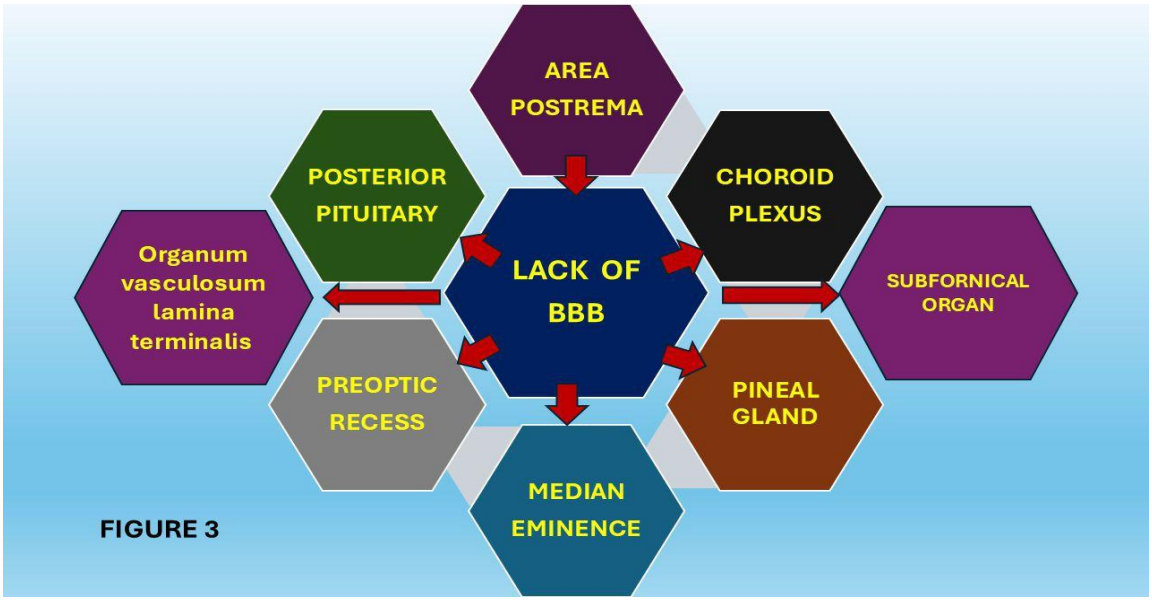

FIGURE 3: Shows area of the brain without BBB.

There are intentional gaps in the BBB, which is characterized by the presence of capillary fenestrations and the absence of TJ. The list of brain areas lacking a BBB commented below:
1.The choroid plexus is CSF-secreting tissue located in the ventricular system. This secretory tissue is composed by a lot of fenestrated CV, which it isolated from the CSF by a layer of cuboidal epithelial cells. 2. Pineal gland is an endocrine organ, secreting melatonin. 3.
Organum vasculosum lamina terminalis that serves as sensory role, detecting changes in sodium concentration and serum osmolality. Its anatomical location is at the ventral aspect of the anterior wall of the third ventricle. 4. The subfornical organ is a sensory organ, which sits at the inferior surface of the fornix; 5. The median eminence is the anatomical region where the hypothalamus secretes its hormone and releasing it into the portal capillary circulation.

The posterior pituitary secretes oxytocin and vasopressin. 6. The preoptic recess of the anterior hypothalamus involved in the coordination of thermoregulation and mating behaviour.

The before cited organs are deep central structures, located in the midline and clustering around the ventricular system, it is why they have grouped as "circumventricular organs". SEE FIGURE 4.

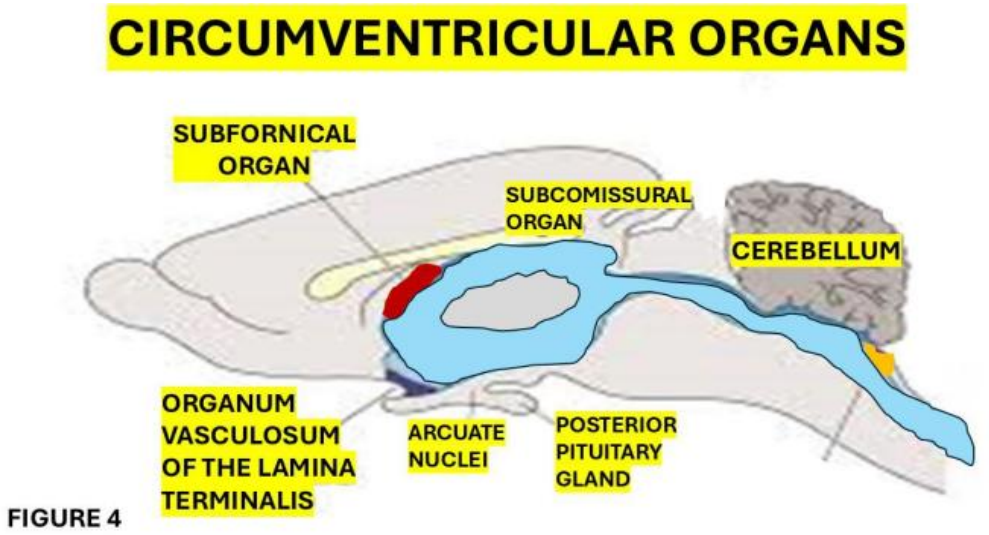

FIGURE 4: Graphical representation of the circumventricular organs.

However, the most common components of the BBB are cellular and ultrastructural elements which can be seen across all levels of the CNS.

Permeability and organization of the BBB are quite dynamic. Notice that it histological structure are not present in all capillaries vessels at all over the brain and spinal cord, as previously cited. Therefore, it has discontinuities at the different locations along specific vascular beds. The graphical representation of the BBB is in FIGURE 5.

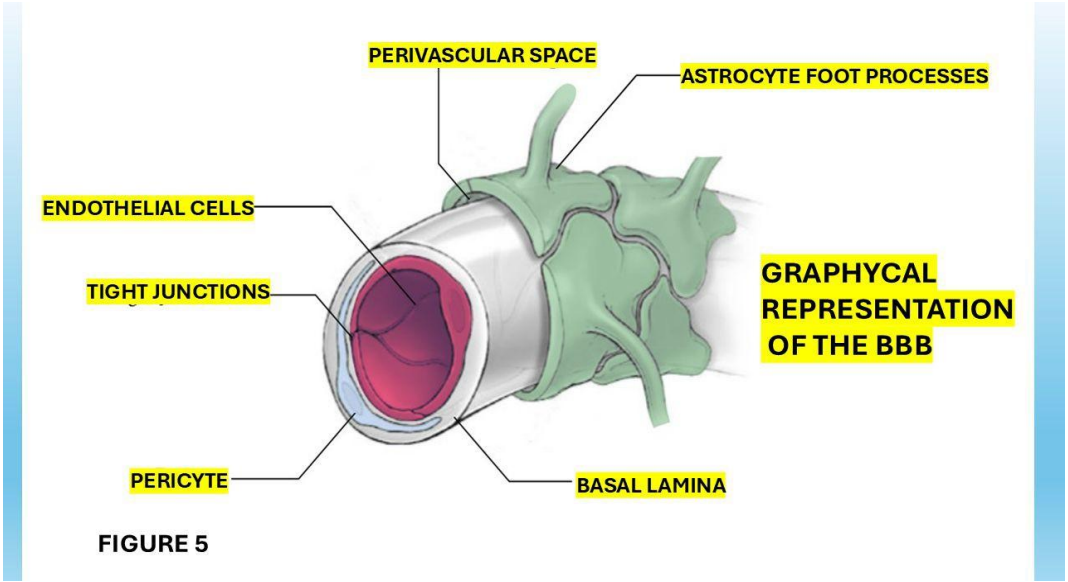

FIGURE 5: Cellular and physical components of the blood brain barrier: Endothelial cells (tight junctions, no fenestrations), Basement membrane (20-30nm), 3 Pericytes, 4. Perivascular fluid space (Virchow-Robin space), 5. Astrocyte foot processes.

The BBB's endothelial (EC) cells differ morphologically from those of microcirculatory beds, lacking fenestrations and having a continuous lipid bilayer, thereby controlling the passage of water-soluble substances and large molecules.

Tight junctions (TJ) located between these EC made almost impenetrable to other molecules and water. Therefore, these TJ are composed of proteins with a sticky label, such as "occludin" and "adherin," which are involved in the histological integrity of these TJ.

BBB's basement membrane (BM) lies beneath the EC and is like BMs in other tissues. It is main functions are as a scaffold, maintenance structure, and support for the health and integrity of the BBB.

The Rouget cells best known as pericytes are supporting mural cells arising from connective tissue that are located within the basal membrane, and their main roles include regulation of brain circulation, angiogenesis, maintenance of the BBB, and control of development of BV [27][26].

As mentioned before, astrocyte foot processes and glial cells are components of the BBB, at least contribute to it and remain intact after administration of immunotoxins.[28]

Brief comments on barrier functions.
To reduce extension of this manuscript we represented the three main functions of the BBB in FIGURE 6.

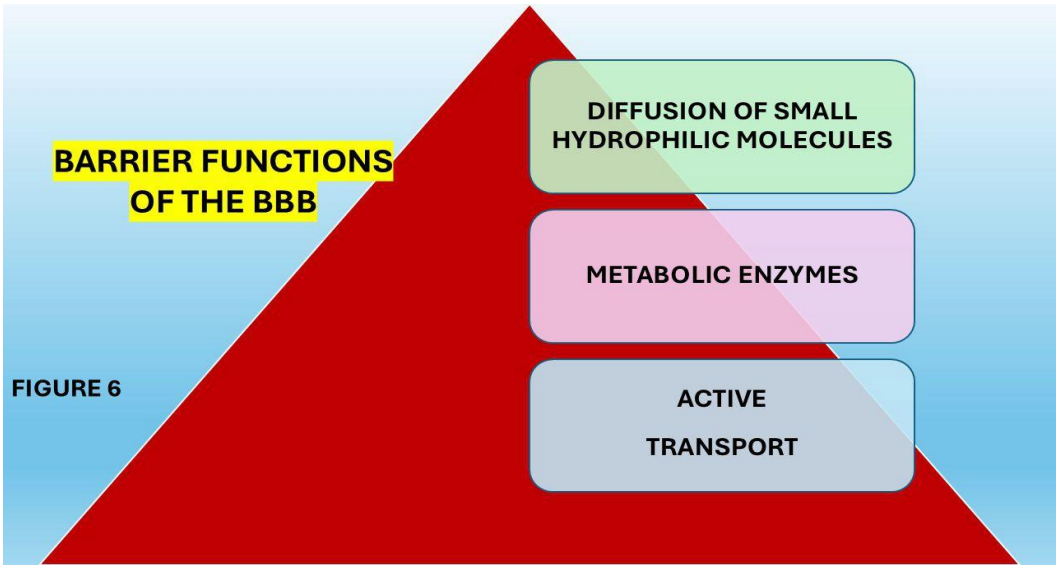

FIGURE 6: Shows the main functions of the BBB according to its components: Tight junctions prevent paracellular passage of small hydrophilic elements; Active transport mechanisms determine which substances can pass.

In Figure 7, we represented the most important elements of transportation through the BBB.

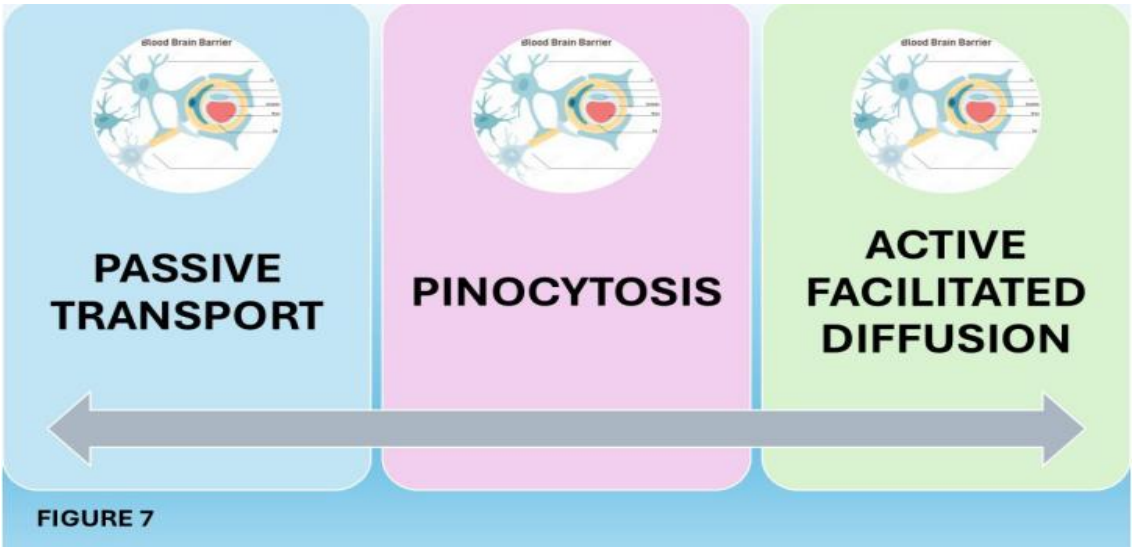

FIGURE 7: The main components of transport across the BBB are represented here and their substrates are listed here.

The physical barrier function is composed by lipid-soluble material, the TJ between the epithelial cells, and the lack of fenestrations. Therefore, molecules can move through lipid bilayers on their way to target neurons/supporting cells. The active transport process modulates the transport barrier, either its presence or its absence. The EC support the transport of specific molecules, which receive the green light to pass through, such as glucose transporters. Metabolic barriers are controlled by EC and astrocyte metabolism of actively transported molecules on the luminal side.

Brief comments on the characteristics of drugs which can cross the BBB.

To pass through BBB, drugs should have certain properties, which we summarized in FIGURE 8

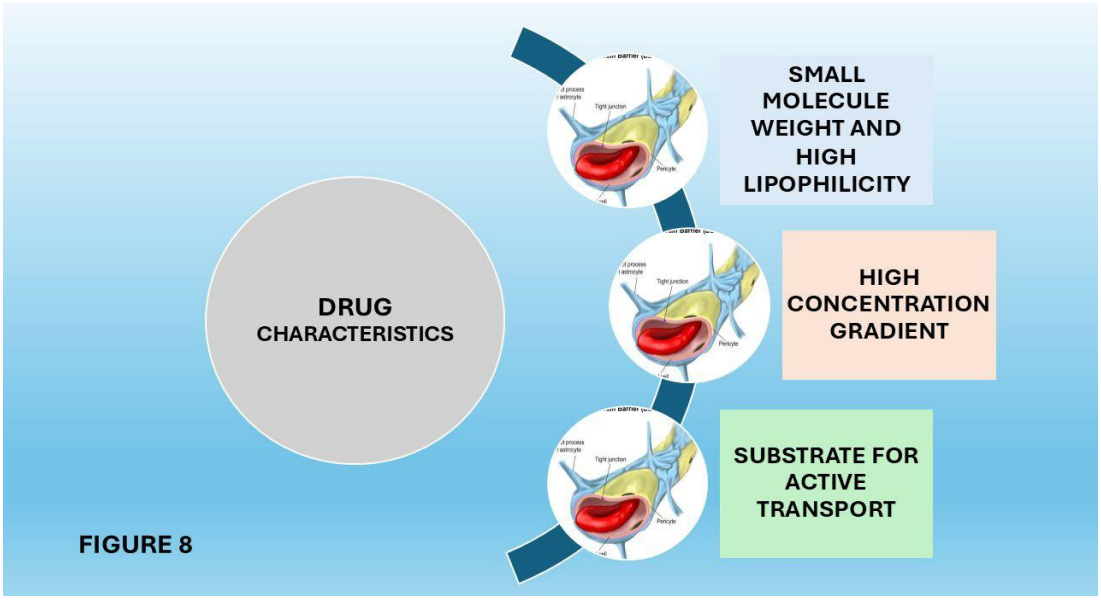

FIGURE 8: The features of drug to support their penetration of the BBB such as: mall molecular weight, high lipophilicity, High concentration gradient (low protein binding, small volume of distribution, low potency of drug i.e. large concentration of drug), Substrate for active transport (resemble endogenous ligand) are represented in this figure.

They would need to either actively or passively transported based on the following requirements such as: For all types of drugs:
Small molecular mass, to facilitate diffusion (ethanol).
Small amount of distribution (leading to a large amount of drug is available in the bloodstream).
High lipid solubility (propofol),

1. Great concentration along to the bloodstream.
2. Low protein binding.
3. Enough molecular similarity with another actively transported substrate like lithium, Valproate, and monoclonal antibodies.

The perivascular space (Virchow-Robin) is a cavity filled of fluids which separates the foot processes of astrocytes from the medial layer of arterioles.

Brief comments on microbiotas and AP

The relationship between AP and neuromyelitis optica spectrum disorder has been reported previously [29]. In 2024, we delivered evidence on the involvement of dysbiosis on the pathogenesis of NMOSD via the microbiota-gut-brain axis [30] and its implications of the free passage of certain substances across capillary vessels (CVs).

Now, we hypothesize that the free passage of autoantibodies across the fenestrate CVs may cause neuronal dysfunction in the AP, leading to the comorbidity of APS and NMOSD, a main implication of the lack of a BBB at the AP. As far as we know, this is the first publication on the implications of the absence of the BBB at the AP.

ACKNOWLEDGMENT

We want to thanks to Prof Thozama Dubula HOD on Internal Medicine and Therapeutic for his support.

Statement on ethic:

This investigation does not qualify for Ethical Approval based on its design

Patient consent:

To ensure anonymity all identifying information from the series was removed.

Conflicts of Interest:

Investigator and collaborators of this manuscripts did not report conflicts of interest.

REFERENCES

1. Obermeier B., Daneman R., Ransohoff R.M. Development, maintenance and disruption of the blood-brain barrier. Nat. Med. 2013; 19:1584–1596. doi: 10.1038/nm.3407].
2. Xiao M., Xiao Z.J., Yang B., Lan Z., Fang F. Blood-Brain Barrier: More contributor to disruption of central nervous system homeostasis than victim in neurological disorders. Front. Neurosci. 2020; 6:764. doi: 10.3389/fnins.2020.00764.
3. Ferrari I., Limiti E., Giannitelli S.M., Trombetta M., Rainer A., D'Amelio M., La Barbera L., Gori M. Microfluidic-Based Technologies for Crossing the Blood-Brain Barrier Against Alzheimer's Disease: Novel Strategies and Challenges. Int. J. Mol. Sci. 2025; 26:9478. doi: 10.3390/ijms26199478.
4. Che J., Sun Y., Deng Y., Zhang J. Blood-brain barrier disruption: A culprit of cognitive decline? Fluids Barriers CNS. 2024; 21:63. doi: 10.1186/s12987-024-00563-3.
5. Chagnot A., Montagne A. The blood-brain barrier. Curr. Biol. 2025; 35:R1010–R1015. doi: 10.1016/j.cub.2025.06.061.
6. Manuele Gori, Livia La Barbera. Special Issue "Advances in Biomedical and Tissue Engineering Strategies to Cross the Blood–Brain Barrier for Next-Generation Treatments Against Neurodegenerative Diseases." Int J Mol Sci. 2025 Dec 20;27(1):63.doi: 10.3390/ijms27010063.
7. Huifeng Li, Ruili Niu, Wenzhen He, Huanling Lai, Shangnan Zou, Qihang Zou, Yue Gui, Tengyue Zhang, Guoyun Feng, Yue Xing, Dehai Gou 2, Xiaofeng Yang. Lachnospira eligens attenuates epileptogenesis via gut-brain axis regulation of blood-brain barrier integrity and neuroinflammation. Theranostics. 2026 Jan 1;16(2):1045–1062.doi: 10.7150/thno.116959.
8. Navari R.M., Aapro M. Antiemetic Prophylaxis for Chemotherapy-Induced Nausea and Vomiting. N. Engl. J. Med. 2016; 374:1356–1367. doi: 10.1056/NEJMra1515442.
9. McKinley, Michael J.; Denton, Derek A.; Ryan, Philip J.; Yao, Song T.; Stefanidis, Aneta; Oldfield, Brian J. "From sensory circumventricular organs to cerebral cortex: Neural pathways controlling thirst and hunger". Journal of Neuroendocrinology. 2029;31 (3) e12689. doi:10.1111/jne.12689
10. Scorza K., Williams A., Phillips J.D., Shaw J. Evaluation of Nausea and Vomiting. Am. Fam. Physician. 2007; 76:76–84.
11. Gross, Paul M.; Wall, Katharine M.; Wainman, Dan S.; Shaver, Steven W. "Subregional topography of capillaries in the dorsal vagal complex of rats: II. Physiological properties". The Journal of Comparative Neurology.1991; 306 (1): 83–94. doi:10.1002/cne.903060107
12. Kyla Bruce, Ameth N Garrido, Song-Yang Zhang, Tony KT Lam. Regulation of Energy and Glucose Homeostasis by the Nucleus of the Solitary Tract and the Area Postrema. Endocrinol Metab (Seoul). 2024 Aug 1;39(4):559–568. doi: 10.3803/EnM.2024.2025.
13. Song-Yang Zhang, Kyla Bruce, Zahra Danaei, Rosa JW Li, Daniel R Barros, Rachel Kuah, Yu-Mi Lim, Laura H Mariani, David Z Cherney, Jennifer FM Chiu, Heather N Reich, Tony KT Lam. Metformin triggers a kidney GDF15-dependent area postrema axis to regulate food intake and body weight. Cell Metab. 2023 Apr 14;35(5):875–886.e5. doi: 10.1016/j.cmet.2023.03.014
14. Tracy L Hagemann, Michelle M Sonsalla, Cora Luzinski, Fernando Zacahua, David A Harris, Dudley W Lamming, Albee Messing. Growth Differentiation Factor 15 Elevation in the Central Nervous System Is Associated With Failure to Thrive in Alexander Disease. Ann Clin Transl Neurol. 2025 Sep 29;13(1):144–156. doi: 10.1002/acn3.70209.
15. Eriko Furube, Rena Fujii, Yuri Nambu, Daishi Hiratsuka, Ryoichi Yoshimura, Seiji Miyata. Region-Specific Adult Neural Stem Cell Niches of the Mediobasal Hypothalamus and Medulla Oblongata. Stem Cell Rev Rep. 2025 Oct 21;22(1):222–248. doi: 10.1007/s12015-025-10995-5.

16. David Andrew Prentice, Ravi Ambati, Lay K Kho, Thomas Jenkins, Paul M Parizel. Seronegative Autoimmune Encephalomyelitis with Area Postrema Symptoms. Case Rep Neurol. 2025 Mar 26;17(1):50–56. doi: 10.1159/000545402
17. Jose Ricardo Muñoz-Zúñiga, Andrea del Mar Tamayo-Delgado, Alberto Masaru Shinchi-Tanaka, Juan Camilo Márquez, Alex Echeverri-García, Santiago Lopez Garcia. Postrema area syndrome in the context of systemic lupus erythematosus: Case report. Heliyon. 2024 Nov 21;11(1):e40614. doi: 10.1016/j.heliyon.2024.e40614
18. Patrick Stancu, Nicolae Sanda, Karl-Olof Lovblad, Nils Guinand, Andreas Kleinschmidt, José Bernardo Escribano Paredes. Area postrema syndrome as the only sign of medullary infarction adjacent to area postrema. eNeurologicalSci. 2025 Mar 14;39:100563. doi: 10.1016/j.ensci.2025.100563.
19. Caitlin Courtney, Catherine Franconi, Ferry Dharsono, David Prentice. Giant Cell Arteritis Manifesting as Area Postrema Syndrome: A Case Report. Cureus. 2025 Sep 29;17(9):e93470. doi: 10.7759/cureus.93470.
20. Qingchen Li, Junfang Teng. Area postrema syndrome in patients with autoimmune glial fibrillary acidic protein astrocytopathy. Front Neurol. 2025 Jan 30;16:1538602. doi: 10.3389/fneur.2025.1538602.
21. Emine Rabia Koc, Mehmet Fatih Yetkin, Furkan Saridas, Omer Faruk Turan, Serhan Sevim, Murat Terzi, Sedat Sen. et al. Real-world effectiveness and safety of eculizumab in AQP4-IgG-positive neuromyelitis optica spectrum disorder. J Neurol. 2026 Jan 8;273(1):67. doi: 10.1007/s00415-025-13608-w.
22. Manami Terahara, Takahiro Hobara, Yujiro Higuchi, Shota Hirakata, Yutaka Noguchi, Satoshi Nozuma, Hiroshi Takashima. Isolated Paramedian Pontine Reticular Formation (PPRF) syndrome in AQP4 antibody–positive neuromyelitis optica spectrum disorder: a case report. BMC Neurol. 2026 Jan 29; 26:132. doi: 10.1186/s12883-026-04670-6.
23. Mariam Mirza; Joe M. Das. Neuroanatomy, Area Postrema. Author Information and Affiliations
24. https://www.ncbi.nlm.nih.gov/books/NBK544249/
25. https://physiome.org/jsim/models/webmodel/NSR/Crone63_BTEX/Crone1963_doc.pdf
26. Lourdes de Fátima Ibañez Valdés. The Role of Rouget Cells in Neurocysticercosis. Journal of Drug and Alcohol Research Vol. 13 (2024), Article ID 236289. DOI: 10.4303/JDAR/236289.
27. Lourdes de Fátima Ibañez Valdés* Humberto Foyaca Sibat. Meningeal lymphatic vessels and glymphatic system in neurocysticercosis. A systematic review and novel hypotheses. Clin Schizophr Relat Psychoses 17 (2023). Doi: 10.3371/CSRP.DLHF.011023.
28. Prakash Thapa, Sriram Kc, Bulbul Pradhan, Inesh Khanal, Aakripa R Shrestha. Neuromyelitis optica spectrum disorder in an 11-year-old: a case report in a paediatric patient from Nepal. Ann Med Surg. (Lond). 2026 Jan 20;88(2):2061–2066. doi: 10.1097/MS9.0000000000004760.
29. Lourdes de Fatima Ibanez Valdes, Foyaca-Sibat Humberto. Impact of Gut Dysbiosis on Neuromyelitis Optica Spectrum Disorder: Implications for Alcohol and Substance Use. Journal of Drug and Alcohol Research Vol. 13 (2024), Article ID 236418, 07 pages DOI: 10.4303/JDAR/236418.