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Anesthesia Management for Evacuation of Cerebral Abscess in Geriatric Patient with Myasthenia Gravis

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Abstract

A Cerebral abscess is an intracranial focal abscess which is a life-threatening emergency. Myasthenia gravis is an autoimmune disorder caused by antibodies targeting the neuromuscular junction's post-synaptic receptor. A seventy-three-year-old male, with an intra-axial tumor in the frontoparietal region underwent craniotomy for abscess evacuation. The Patient also has a history of hypertension and myasthenia gravis under treatment of dexamethasone and pyridostigmine. Anesthesia induction was performed with thiopental, opioid analgesics with fentanyl, neuromuscular blocking agent (NMBA) with rocuronium, and scalp block. The Patient's depth of neuromuscular block was monitored with a Train-of-Four (TOF). Surgery was performed in a supine position, duration of surgery was 4.5 hours. The Patient was extubated in the operating theatre, monitored in the intensive care unit, and discharged home on the nineteenth day. Anesthetic management in geriatric patients with cerebral abscesses accompanied by myasthenia gravis has become complex due to the interaction of disease state, medical treatment, anesthetic drugs especially neuromuscular blocking agents, and surgical stress. The Patient was at risk for residual paralysis and had high sensitivity to nondepolarizing neuromuscular blocking agents, so the use of train-of-four (TOF) was very helpful for extubating this patient safely.

Keywords: anesthesia, cerebral abscess, geriatric, myasthenia gravis

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I. Introduction

Cerebral abscess is an intracranial focal abscess and is a life-threatening emergency.[1,2] Cerebral abscess appears as a localized cerebritis and later the encapsulated pustular material appears as a mass lesion.[3] Cerebral abscess occurs more frequently in men, two to three times compared to women and the highest morbidity rates occur in the fourth decade of life. Cerebral abscesses have high morbidity, such as seizures, persistent mental status changes and focal motor deficits.[4,5] The cerebral abscesses incidence is around 8% of intracranial masses in developing countries and 1-2% in developed countries.[3,6] Mortality from cerebral abscesses has decreased from around 50% to 20%, due to the avalability of Computerized Tomography (CT) scans which facilitate an early diagnosis and accurate location.[6] Advanced techniques for isolating and identifying microorganisms, better antimicrobials penetration and stereotactic aspiration have reduced the mortality rate of cerebral abscesses to less than 10%. Mortality is affected by older age and severity of general neurological condition, delay in treatment, impaired immunity and poor controlled diabetes mellitus. Decreased consciousness with a Glasgow Coma Scale (GCS) <12 increases the mortality rate and risk of permanent neurological deficits.[7] Myasthenia gravis is a disorder due to antibodies targeting the nicotinic acetylcholine receptor.8 Myasthenia gravis is more frequently found in

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women than men. Women are more commonly affected before fourth decades, and men in the fifth and sixth decades.[9,10] Triggers for myasthenia gravis include infection, emotional or physical stress, changes in thyroid hormone function, general anesthesia, and certain medications. Myasthenia gravis patients usually present with muscle weakness and being tired easily. Ocular symptoms such as ptosis and asymmetric tiredness, with or without double or blurry vision, are typically present. Most of the time, it worsens and resulted in widespread weakening of the muscles of the face, neck, bulbar region, and limbs. Neuromuscular dysphagia, which quickly develops to total loss of swallowing function and accompanied with ventilatory muscle weakness leading to ventilation failure, occurs in myasthenic crisis, which is at the severe end of the illness progression. This is a clinical emergency that has to be managed with intense care.[11,12] Anesthetic management in patients with cerebral abscesses accompanied with myasthenia gravis is complex and potentially increase patient morbidity. Anesthesia problems in myasthenia gravis patients arise from interactions among the disease state, medical treatment, anesthetic drugs especially neuromuscular blocking agent and surgical stress. Patients with myasthenia gravis are highly sensitive to nondepolarizing NMBA and resistant to depolarizing NMBA such as succinylcholine. This patient is also a geriatric patient who is at risk of residual neuromuscular block due to the use of nondepolarizing NMBA. Neuromuscular block monitoring with train-of-four (TOF) provides safer titration of nondepolarizing NMBA thus facilitates early and safe extubation in geriatric patients with myasthenia gravis undergoing craniotomy.

II. Case

History

A Male, 73 years old, presented with weakness in the left side of his body approximately 2 weeks before admission to the hospital. Initially the patient was still able to lift his arms, legs and walk slowly, then later he became weaker and eventually he was unable to move. The patient also complained of slurred speech but was still able to swallow soft food. The patient denied pain in the upper and lower extremities. History of nausea, vomiting, seizure, and fainting were also denied. Eating, drinking and defecation were not disturbed. Urination was a bit obstructed, but urine was still coming out. A history of hypertension had been known for 10 years with treatment with amlodipine 10 mg once a day. This patient had a known history of myasthenia gravis since 2019, currently being treated with prednisone 10 mg twice a day and a history of receiving azathioprine 20 mg once a day but this had been stopped for 2 weeks. The patient was receiving pyridostigmine 60 mg four times a day. Patient declared of no history of diabetes mellitus, asthma, heart disease and other systemic diseases. He also stated that there wasn't prior surgical history either. The patient is a grandfather whom before his illness was still carrying out mild to moderate activities without any complaints any shortness of breath.

Physical Examination

The patient's weight was 60 kg, height 170 cm, body mass index of 18.7 kg/m2. The patient was fully conscious with GCS E4V5M6. Axillary temperature 36.5oC, Numerical rating scale (NRS) score 0-1/10, Metabolic Equivalent of Task (METs) score 5-6. Chest wall movement was symmetrical, respiratory frequency was 18-21 times per-minute, vesicular sounds in both lung fields, without any rales and wheezing, peripheral oxygen saturation was 96-97% and Sabrazes test was 24-25 seconds. Blood pressure 130/80 mmHg, pulse rate 82 beats per minute, heart sounds was normal and regular, without additional heart sounds. The patient's teeth were not intact, there were no dentures. Abdominal examination revealed normal positive bowel sounds. Musculoskeletal examination revealed limited neck flexion, warm extremity, and no ptosis. Neurological examination found no signs of meningeal irritation, there was paresis of the right VII nerve, the impression of lateralization with right spasticity grade >3, there was a suggestion of lateralization to the left, left supranuclear paresis of the VII and XII nerves, left flaccid hemiparesis grade 1/2. Pathological reflexes were not found, and the Wartenberg test was normal.

Laboratory and Imaging Results

A complete blood count found leukocytes 11.37 x 103/µL (4.1-11), hemoglobin 13.40 g/dL (12-16), hematocrit 40.90% (36-46), platelets 224 x 103µL (140-440). Hemostatic profile PT 9.7 seconds (10-12.7), aPTT 20.7 seconds (23-34.7), INR 0.84 (0.9-1.1). Clinical chemistry examination found Natrium 142 mmol/L (136-145), Kalium 3.70 mmol/L (3.5-5.1), Chloride 104.7 mmol/L (94-110), BUN 12.90 mg/dL (8.00-23.00), Creatinine 0.69 mg/dL (0.72-1.25); SGOT 18.7 U/L (5-34); SGPT 25.10 U/L (11.0-34.00). Electrocardiography (ECG) examination showed normal sinus rhythm, pulse rate 82 beats per minute, without ST-T segment changes. Thoracic X-Ray examination showed that the heart and lungs were within normal limits with a cardio-thoracic ratio of 56% and a visible thoracic spondylosis. Magnetic Resonance Imaging (MRI) examination of the head revealed multiple cystic lesions in the right frontoparietal lobe with

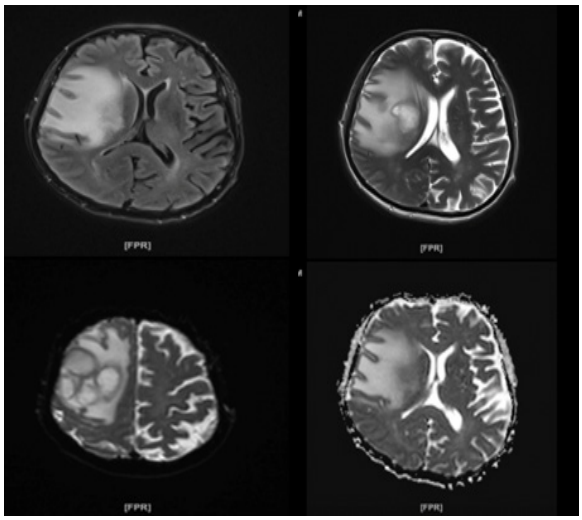

Figure 1. MRI examination of the head with contrast revealed multiple cystic lesions in the right frontoparietal lobe with daughter cysts and surrounding vasogenic edema, suggesting cerebral abscess

daughter cysts and surrounding vasogenic edema compressing the right lateral ventricle causing a midline shift to the left, suggesting a cerebral abscess. The patient was concluded having an intra-axial tumor in the frontoparietal region which was cerebral abscess with myasthenia gravis and planned for craniotomy cerebral abscess evacuation. The patient's classified as American Society of Anesthesiologists (ASA) III.

Preoperative Management

The patient was positioned head up 30º. Patient received antibiotic ceftriaxone, metronidazole, dexamethasone, omeprazole, amlodipine, valsartan, acetylcysteine, betahistine and chest physiotherapy.

Anesthetic Management

Arterial line was put in place under local anesthesia lidocaine 2% after an Allen test was performed. Anesthesia was induced with thiopental 250 mg (3-5 mg/kg) and analgetics fentanyl 150 mcg i.v (2-3 mcg/kg). Before intubation, the patient received rocuronium 15 mg i.v (0.3 mg/kg titration) with train-of-four

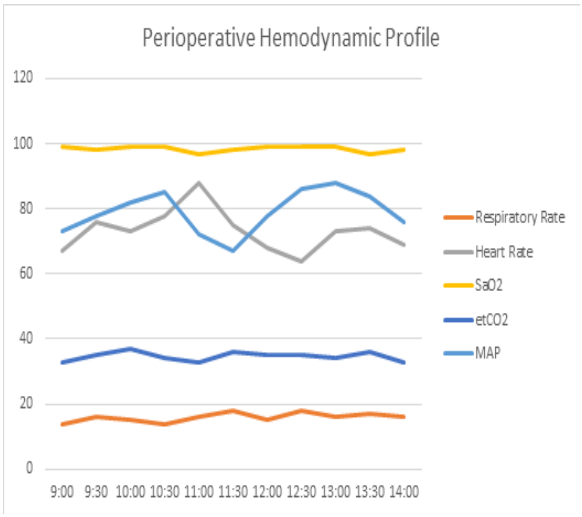

Figure 2. Perioperative Hemodynamic Profile

monitoring. Laryngoscopy was performed and endotracheal intubation with tube number 7. Scalp block was performed with infiltration of the Supratrochlear nerve, Supraorbital nerve, Zygomaticotemporal nerve, Auricotemporal nerve, Occipital Major nerve, and Occipital Minor nerve with 2% lidocaine, 2–3 ml on each nerve before the patient was positioned and a headpin was installed. Maintenance of anesthesia done by sevoflurane 0.4 MAC, thiopental 1–3 mg/kg/hour i.v, dexmedetomidine 0.3–0.7 mcg/

kg/h, intermittent fentanyl 0.25 mcg/kg i.v. Other medications received by the patient were paracetamol 1000 mg, tranexamic acid 1000 mg i.v, mannitol 0.5mg/kg, and ondansetron 8 mg i.v.

Postoperative Management

After surgery, patient was extubated in the operating theatre and closely monitored for one day in the intensive care unit. Post operative analgesic were fentanyl 350 mcg i.v in 24 hours and paracetamol i.v.

III. Discussion

Cerebral Abscess

Cerebral abscess can originate from: First spread of infection from neighboring pericranial foci such as sinus and middle ear. Second, spread from distant foci of infection by hematogenous route such as lung abscess and bacterial endocarditis. Third, from direct inoculation such as head trauma or neurosurgery.[12,13]

The four main signs of a cerebral abscess were localized mass enlargement, widespread damage, elevated intracranial pressure, and focal neurological impairments. The location, size, number, and particular brain regions affected, together with any environmental structural changes affecting the ventricles, dural venous sinuses, and degree of subsequent cerebral damage, all influence of how an intracranial abscess presents clinically. Patients frequently showed signs of focal neurological impairments, fever (which may be absent in 30–76% of cases), and elevated intracranial pressure (which can cause headaches, nausea, vomiting, and mental status abnormalities).[14-16] The most typical clinical manifestations are headache, fever, and stiff neck. The symptoms might last anywhere from one day to eight weeks.[2] In this case, the patient presented with weakness in the limbs. On neurological examination, the patient did not complaints any fever, headache and neck stiffness with negative meningeal signs. There was impression of lateralization of right spasticity grade > 3 and positive lateralization to the left. Left supranuclear paresis of cranial nerves VII and XII. MRI examination revealed the impression of a cerebral abscess in the patient. Focal neurological symptoms may appear due to increased ICP (Intracranial Pressure) and local compression by the cerebral abscess.[16]

Myasthenia Gravis

The dominant clinical symptom of myasthenia gravis is muscle weakness, which usually gets worse after muscle activity, so patients usually state that the symptom was in the morning, followed by weakness that gets worse in the afternoon or evening.[17] Around 85% patients suffer generalized myasthenia gravis and approximately 15% of patients have only ocular symptoms.[11] Typical ocular symptoms of ptosis and diplopia, usually asymmetrical, due to extraocular muscles are often affected,.18 Respiratory muscle weakness may occur rarely, leading to ventilatory failure and needing ventilatory support. Most myasthenia gravis patients are stable with adequate medical treatment, suffering mild muscle weakness and are fully able to perform daily functions.[19] Myasthenia gravis is due to autoantibodies that bind to critical functional receptors on the postsynaptic membrane at the neuromuscular junction. Antibodies against the acetylcholine receptor is detectable in 85% of patients with myasthenia gravis. Small proportion of myasthenia gravis patients have antibodies against muscle-specific kinase (MuSK) or lipoprotein-related protein 4 (LRP4).[17] Antibodies are not detectable in 10–15% of patients with generalized myasthenia gravis.[20] Myasthenia gravis is induced by thymoma in 10% of patients. In this case there was no history of thymoma nor thymectomy surgery. This patient was diagnosed with Myasthenia gravis since 2019 with current treatment: dexamethasone 5 mg i.v every 12 hours and pyridostigmine 60 mg orally every 6 hours.

Neuro-Anesthesia in Geriatric

There are several alterations in the central nervous system brought on by both normal and pathological aging, most of which make patients more vulnerable to anesthesia and surgical site problems. In older people, several medications have unfavorable side effect profiles, such as benzodiazepines, diphenhydramine, scopolamine, metoclopramide, meperidine, and nonsteroidal

anti-inflammatory drugs (NSAIDs).[18] The volume and synapses of the brain decrease in the elderly population. Memory loss, cognitive decline, sleep difficulties, delirium, depression, and impaired neuroplasticity are some of the functional declines that can resulted from volume reduction and synapses of the brain. Dementia diagnosis becomes pertinent when cognitive and memory impairments become so severe as to interfere with everyday activities. Cognitive impairment and post-operative delirium are two risks closely associated with dementia. Maintaining cerebral autoregulation during anesthesia is essential for optimal cerebral perfusion and oxygenation, particularly in individuals with dementia who are more likely to experience cognitive problems after the surgery.[18]

As we aged, fewer neurotransmitters and neuroreceptors are available. This decrease frequently leads to a longer length of neuromuscular blockade and raises the possibility of reintubation and postoperative respiratory problems. In treating the elderly, neuromuscular blocking agent should be avoided or administered more cautiously. Furthermore, in the presence of residual neuromuscular block, elderly patients are more vulnerable to aspiration pneumonia due to weaker pharyngeal muscles and reflexes; therefore, full reversal should be confirmed prior to extubation.[18]

Surgical Management

The effective treatment of brain abscess requires a comprehensive strategy. This strategy involves the use of antibiotics, surgical intervention, neuroradiological examination, and removal of the main infection focus. The development of an intracranial abscess is a direct result of the human immune system and the pathogenic microorganism's pathogenicity.

Early neurosurgical intervention is advised for cerebral abscesses, since they typically require drainage in addition to the proper antimicrobial treatment.[15] Despite the fact that brain abscesses are essentially a surgical disease, some research suggest that the patient should choose the course of treatment that works best for them. The best candidates for medical treatment are those with a small abscess (less than 2.5 cm), good initial clinical condition (GCS >12), and known etiology (microorganisms isolated from non-abscess material); or in cases of multiple abscesses, following surgery for abscesses larger than 2.5 cm or those that result in mass effect; or in patients who pose a serious risk to themselves during surgery, though in this situation the final decision must be taken into account that each case has a poor prognosis.[17]

Anesthetic Management

Anesthetic management of myasthenia gravis patients must be tailored to the severity grade of the disease, in response to medical treatment and extend of surgery. Whenever regional anesthesia is employed, the dose of local anesthetic should be reduced. Myasthenia gravis patients under anticholinesterase therapy should be given reduced dose of ester local anesthetics. General anesthesia can be performed safely, after optimal preparation of the myasthenia gravis patients and neuromuscular function were monitored throughout anesthesia. In this case, general anesthesia combined with regional scalp block anesthesia was performed and neuromuscular function was monitored using train-of-four.[18]

Preoperative evaluation of myasthenic patients should be done carefully. Preanesthetic accurate assessment of respiratory muscle and bulbar function is important for base line post operative evaluation. Respiratory reserve should be monitored by measuring serial forced vital capacity (FVC) with spirometry examination.19 In this patient, a CT-Scan of the chest with contrast was performed to track the presence of thymoma and the results showed that no lung or mediastinal mass was visualized, but no lung function test through spirometry was done yet. The patient's condition was optimized by administering pyridostigmine until the morning before surgery. Discontinuing pyridostigmine the night before surgery made patients more susceptible to muscle weakness while awaiting surgery and patients would show significant increased sensitivity to nondepolarizing NMBA. In the preoperative periode the patient should

have continued the treatment that had been given, but in this case the patient had stopped taking prednisone 10 mg twice a day for 2 weeks. In this case we only gave premedication dexamethasone 10 mg i.v. Giving corticosteroids would have a better long-term impact.[18]

General anesthesia in myasthenic patients could be performed without using NMBA, because of the marked sensitivity to non-depolarizing NMBA and the unpredictable response to depolarizing NMBA. When performing laryngoscopy, tracheal intubation and maintenance of anesthesia, combination of general anesthetics and short acting opioid are sufficient. Desflurane and sevoflurane may offer some advantages, due to their low blood solubility.[19] When possible, the anesthesiologist can ultilize regional anesthetic techniques. Combining regional anesthesia with general anesthesia potentially reduce or eliminate the need for NMBA in surgery. General anesthesia in myasthenic patients could also be performed with non- depolarizing NMBA in cautious manner, because this group of patients is extremely sensitive to nondepolarizing NMBA. Nondepolarizing NMBA should be admistered with significant dose reduction, titrated to effect and guided by using neuromuscular monitoring train of four.

In this case, induction of anesthesia and nondepolarizing NMBA facilitated intubation were performed with a dose reduction from the usual dose of rocuronium (0.3 mg/kg) with monitoring of neuromuscular function with train-of-four. Maintenance of anesthesia in this case used a combination of the volatile anesthetic sevoflurane and intravenous thiopental and dexmedetomidine without further additional NMBA. In this case, we used thiopental to perform anesthesia induction. A study comparing the use of thiopental with propofol in myasthenia gravis patients explained that both drugs had the same recovery rate and extubation time. No significant hemodynamic changes were found. Propofol has the advantage of a short duration of action.[17] The opioid fentanyl which used in this case can reduce cerebral blood flow (CBF), reduce ICP, maintain cerebral perfusion pressure and cerebral metabolic oxygen rate (CMRO2). We ultilized topical airway anesthesia to prevent marked changes in hemodynamic parameters and changes in intracranial pressure during the intubation and maintenance phases.[17] Complete reversal after using rocuronium or vecuronium is possible with sugammadex while avoiding the disadvantages of neostigmine administration in myasthenia gravis patients. The anesthesiologist must closely monitor the patients for any possible residual neuromuscular blockade.[19] Postoperative management such as ventilation function should be monitored carefully. Adequate spontaneous ventilation and respiratory muscle function must be ensured before tracheal extubation. Myasthenic patients has an increased risk of postoperative respiratory problem. Slower recovery with intensive care support should always be considered by the anesthesiologist and surgical team. Extubation after patient fully awake with adequate ventilatory function is ideal in myasthenia gravis patients undergoing general anesthesia.[20]

IV. Conclusion

Anesthesia problems in geriatric patients with cerebral abscess and myasthenia gravis is complex. This complexity includes interactions between disease, treatment, and drugs used for anesthesia, especially NMBA. Myasthenia gravis patients are very sensitive to nondepolarizing NMBA and resistant to depolarizing NMBA such as succinylcholine. This patient is also a geriatric patient who is at risk of residual neuromuscular block due to the use of nondepolarizing NMBA.
. Successful anesthetic management of geriatric patients with myasthenia gravis undergoing craniotomy requires close monitoring, especially neuromuscular function with train-of-four.

References

1. Alvis Miranda H, Castellar-Leones SM, Elzain MA, Moscote-Salazar LR. Brain abscess: Current management. J Neurosci Rural Pract 2013;4(Suppl 1):67-81. Doi: https://doi.org/10.4103/0976-3147.116472

2. Muzumdar D, Jhawar S, Goel A. Brain abscess: an overview. Int J Surg. 2011;9(2):136–44. Doi:10.1016/j.ijsu.2010.11.005

3. Gilhus NE. Myasthenia Gravis. N Engl J Med [Internet]. 2016 ;375(26):2570–81. Available from: https://doi.org/10.1056/NEJMra1602678

4. Gilhus NE, Tzartos S, Evoli A, Palace J, Burns TM, Verschuuren JJGM. Myasthenia gravis. Nat Rev Dis Prim [Internet]. 2019;5(1):30. Available from: https://doi.org/10.1038/s41572-019-0079-y

5. Evoli A. Myasthenia gravis: new developments in research and treatment. Curr Opin Neurol [Internet]. 2017;30(5). Available from: https://journals.lww.com/co-neurology/Fulltext/2017/10000/Myasthenia_gravis__new_developments_in_research.5.aspx

6. Ciafaloni E. Myasthenia gravis and congenital myasthenic syndromes. Continuum (Minneap Minn). 2019;25(6):1767–84. Doi: https://doi.org/10.1212/CON.0000000000000800

7. Fang W, Li Y, Mo R, Wang J, Qiu L, Ou C, et al. Hospital and healthcare insurance system record-based epidemiological study of myasthenia gravis in southern and northern China. Neurol Sci. 2020;41(5):1211–23. Doi: https://doi.org/10.1007/s10072-019-04146-1

8. Sonneville R, Ruimy R, Benzonana N, Riffaud L, Carsin A, Tadié JM, et al. An update on bacterial brain abscess in immunocompetent patients. Clin Microbiol Infect [Internet]. 2017;23(9):614–20. Available from: https://www.sciencedirect.com/science/article/pii/S1198743X1730259

9. Dresser L, Wlodarski R, Rezania K, Soliven B. Myasthenia Gravis: Epidemiology, Pathophysiology and Clinical Manifestations. J Clin Med 2021;10(11). Doi: https://doi.org/10.3390/jcm10112235

10. Hendricks TM, Bhatti MT, Hodge DO, Chen JJ. Incidence, epidemiology, and transformation of ocular myasthenia gravis: A population-based study. Am J Ophthalmol. 2019;205:99–105. Doi:https://doi.org/10.1016/j.ajo.2019.04.017.

11. Gilhus NE, Skeie GO, Romi F, Lazaridis K, Zisimopoulou P, Tzartos S. Myasthenia gravis - autoantibody characteristics and their implications for therapy. Nat Rev Neurol. 2016;12(5):259–68. Doi: https://doi.org/10.1038/nrneurol.2016.44.

12. Gilhus NE, Verschuuren JJ. Myasthenia gravis: subgroup classification and therapeutic strategies. Lancet Neurol. 2015;14(10):1023–36. Doi: https://doi.org/10.1016/S1474-4422(15)00145-3.

13. Andersen JB, Gilhus NE, Sanders DB. Factors affecting outcome in myasthenia gravis. Muscle Nerve. 2016;54(6):1041–049. Doi:https://doi.org/ 10.1002/mus.25205

14. Marx A, Pfister F, Schalke B, Saruhan-Direskeneli G, Melms A, Ströbel P. The different roles of the thymus in the pathogenesis of the various myasthenia gravis subtypes. Autoimmun Rev. 2013;12(9):875–84. Doi: https://doi.org/10.1016/j.autrev.2013.03.007

15. Chauvet D, Sainte-Rose C, Boch AL. [The mystery of prehistoric trepanations: Is neurosurgery the world eldest profession?]. Neurochirurgie. 2010;56(5):420–5. Doi: https://doi.org/10.1016/j.neuchi.2010.07.019

16. Arlotti M, Grossi P, Pea F, Tomei G, Vullo V, De Rosa FG, et al. Consensus document on controversial issues for the treatment of infections of the central nervous system: bacterial brain abscesses. Int J Infect Dis IJID Off Publ Int Soc Infect Dis. 2010;14 Suppl 4:S79-92. Doi: https://doi.org/10.1016/j.ijid.2010.05.010

17. Froese L, Dian J, Batson C, Gomez A, Unger B, Zeiler FA. Cerebrovascular response

to propofol, fentanyl, and midazolam in moderate/severe traumatic brain injury: A scoping systematic review of the human and animal literature. Neurotrauma rep. 2020;1(1):100–12. Doi: https://doi.org/10.1089/neur.2020.0040

18. Partridge JS, Harari D, Martin FC, Dhesi JK. The impact of pre-operative comprehensive geriatric assessment on postoperative outcomes in older patients undergoing scheduled surgery: a systematic review. Anaesthesia. 2014;69 Suppl 1:8-16. Doi:https://doi.org/10.1111/anae.12494

19. Fernandes HDS, Ximenes JLS, Nunes DI, Ashmawi HA, Vieira JE. Failure of reversion of neuromuscular block with sugammadex in patient with myasthenia gravis: Case report and brief review of literature. BMC Anesthesiol. 2019;19(1):160. Doi: https://doi.org/10.1186/s12871-019-0829-0

20. Chen L, Xie W, Zheng D, Wang S, Wang G, Sun J, et al. Early extubation after thymectomy is good for the patients with myasthenia gravis. Neurol Sci 2019; 40(10):2125–32. Doi: https://doi.org/10.1007/s10072-019-03941-0