JNI Jurnal Neuroanestesi Indonesia

Case Report

Anesthetic Management of Craniotomy for Supratentorial Tumor Resection in a Patient with Femoral-Popliteal Deep Vein Thrombosis: Case Report

Tengku Addi Saputra[*)], **Radian Ahmad Halimi**[*)], **Hamzah**[***)]

[*)]Department of Anesthesiology and Intensive Care, Faculty of Medicine, Universitas Padjadjaran Dr. Hasan Sadikin General Hospital Bandung, [**)]Department of Anesthesiology and Reanimation, Faculty of Medicine, Universitas Airlangga–Dr. Soetomo General Hospital Surabaya

Received: July 03, 2024; Accepted: August 26, 2024; Publish: October 24, 2024

correspondence: addisaputra1980@gmail.com

Abstract

Patients with brain tumors are highly susceptible to venous thromboembolism (VTE), which includes deep vein thrombosis (DVT) and pulmonary embolism (PE). Approximately 20–30% of those with intracranial tumors experience VTE, with factors such as neurological deficits, tissue factor secretion, genetic predispositions, advanced age, and hypertension contributing to the risk. In this case, a 61year woman with Space Occupying Lesion (SOL) Supratentorial at Midfrontal, Hypertension Stage II, Diabetes Mellitus (DM) type 2, and femoral-popliteal DVT undergoes craniotomy in general anesthesia. A thorough preoperative assessment is carried out to increase the success of anesthesia, including Doppler ultrasound assessment, administering anticoagulants up to 24 hours before surgery, and monitoring platelet levels and coagulation profiles. Managing VTE in these patients typically involves anticoagulants, thrombolytics, and thrombectomy tailored to the clinical situation. However, the use of anticoagulants, like heparin, poses a risk of severe bleeding during surgical procedures such as craniotomy. A craniotomy is associated with an increased risk of VTE due to endothelial damage, thromboplastin release, and post-operative immobilization, all contributing to Virchow's Triad (venous stasis, endothelial injury, and hypercoagulability). Anesthesiologists must provide meticulous perioperative care, incorporating preoperative and post-operative anticoagulant prophylaxis and being aware of intraoperative bleeding. While VTE is recognized as a common post-operative complication, its impact during surgery and the strategies needed to mitigate related risks are still underexplored. Understanding and addressing these challenges are essential, particularly in patients undergoing craniotomy for intracranial tumors, to improve surgical outcomes and reduce mortality.

Keywords: Anesthesia management, craniotomy, deep vein thrombosis, meningioma

J. neuroanestesi Indones 2024;13(2): 128–35

I. Introduction

Patients with brain tumors are at high risk for venous thromboembolism (VTE), which includes deep vein thrombosis (DVT) and pulmonary embolism (PE). Various studies have found that a proportion of 20–30% of intracranial tumor patients experience VTE.[1-3] Patients with brain tumors such as meningiomas generally experience neurological deficits in the form of psychomotor disorders.[4] These disorders can lead to physical inactivity, which may cause venous stasis. Additionally, secretion tissue factors and genetic factors also play important roles in increasing VTE risk.[5] Other risk factors that can increase the incidence of VTE in patients with intracranial tumors are age and hypertension. Although the exact pathomechanism is not yet known with

doi: https://doi.org/10.24244/jni.v13i3.606
ISSN (Print): 2088-9674 ISSN (Online): 2460-2302

JNI is accredited as Sinta 2 Journal: https://sinta.kemdikbud.go.id/journals/profile/796

How to cite: Saputra TA, et al, "Anesthetic Management of Craniotomy for Supratentorial Tumor Resection in a Patient with Femoral-Popliteal Deep Vein Thrombosis: Case Report".

certainty, these two factors can elevate the risk of VTE events up to 2-fold.[6,7] Studies have shown that VTE increases the risk of mortality in patients with intracranial tumors by up to 30% within two years.[1] Management of VTE generally involves the use of anticoagulants, thrombolytic agents, and thrombectomy, depending on the patient's clinical condition. Anticoagulants such as heparin are commonly used to prevent the spread of local thrombosis and the formation of emboli. However, caution is necessary with their use, as they can increase the risk of significant bleeding during surgery.[9]

Craniotomy has become the main surgical approach in cases of intracranial tumors, including parasagittal meningioma.[4] Perioperative anesthetic management of patients with DVT who will undergo craniotomy is a particular challenge for anesthesiologists. This is because craniotomy has been proven to increase the risk of VTE.[7,11,12] The craniotomy procedure causes damage to the endothelium and stimulates the secretion of thromboplastin in the coagulation cascade, leading to increased blood coagulability.[13] Additionally, motor paresis and post-operative immobilization can also lead to venous stasis.[11] Venous stasis, endothelial damage, and blood hypercoagulability are components of Virchow's Triad, which is the main pathomechanism of VTE.[14] Therefore, It can be concluded that patients with intracranial tumors and VTE such as DVT are at high risk of worsening coagulation disorders during and after craniotomy procedures. Consequently, it is very crucial for an anesthesiologist to conduct comprehensive perioperative anesthetic management, including administering preoperative and post-operative prophylactic anticoagulants and anticipating intraoperative bleeding. Various case reports describe VTE as a post-operative impact of neurosurgical procedures.[1,5,7]

However, studies regarding the impact of VTE, such as DVT, on intraoperative procedures and the necessary management to prevent worsening is still very limited. This is important to research because DVT not only acts a role as a complication but also as a contributing factor to the worsening of craniotomy outcomes, particularly in patients with intracranial tumors.

II. Case

History

A female, 61 years old, 70 kg 164 cm (BMI 26 kg/m2), with loss of consciousness and severe headaches for three months before admission to Hasan Sadikin Hospital, was referred from another hospital. A patient with loss of consciousness, severe headache, nausea, vomiting, blurred vision, and left leg swelling. A patient with a history of hypertension with routinely amlodipine 10 mg and DM type 2 with short-acting insulin (s.c).

Physical Examination

A physical examination showed GCS 9 (E2M5V2), blood pressure 160/80 mmHg, pulse rate 78 x/minute, respiratory rate 28 x/minute, temperature 36.5 ^{o}C, SpO_2 99% with 10 liters per minute oxygen through non-rebreathing mask.

Preoperative Laboratory

Hb 12,7 g/dl, PT 12,7 second, INR 0,89 second, APTT 21,7 second, serum glucose 297 mg/ dL (N: 80 – 200 mg/dL), and D-dimer 4,34 µg/ ml (N: <0.55 µg/ml). USG Doppler (Figure 1) preoperative showed DVT of the left femoral vein and left popliteal vein ec. Intraluminal thrombus of the left femoral vein, thickening of the subcutaneous tissue in the left crural region that gives a cobblestone appearance, suggestive of lymphedema. Chest X-ray (Figure 2) showed pneumonia and cardiomegaly. Echocardiography examination showed L.V.E.F. 62 %, average all chambers dimension, normal LV systolic function with normokinetic at rest, normal LV diastolic function, normal anatomy and function of all valves, low probability of PH, normal RV systolic function.

Head CT scan (Figure 3) examination showed Solid extra-axial supratentorial mass located in the right frontal parasagittal measuring 7.64 x 4.44 x 5.95 cm, accompanied by pressing vasogenic edema and narrowing of the sulci of the cortical gyri of the right frontal lobe, and midline shift to the left as far as 2.76 cm which

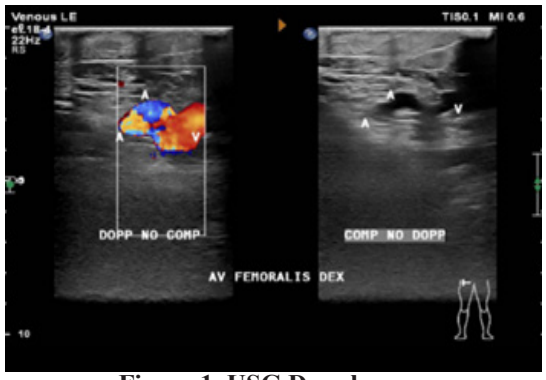

Figure 1. USG Doppler

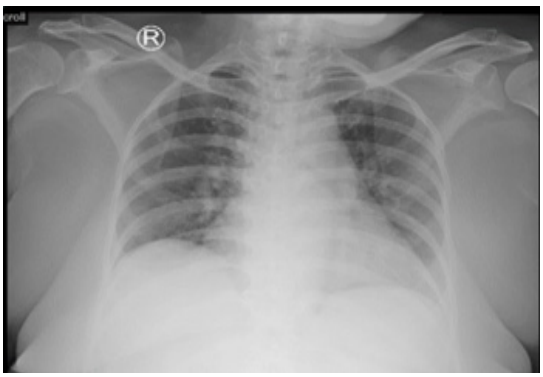

Figure 2. Chest X-ray

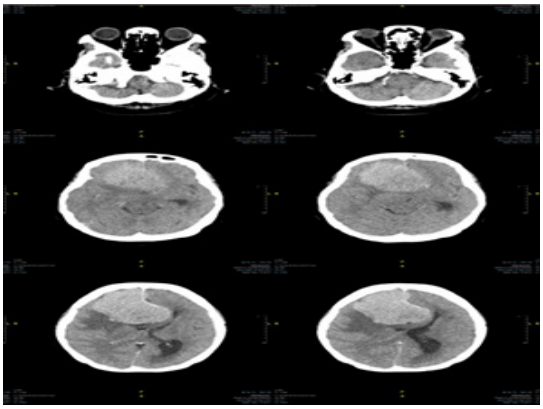

Figure 3. Head CT Scan

on post-contrast scanning gives an image of possibility of homogeneous enhancement. Parasagittal meningioma was the suggestion. A patient diagnosed with SOL Supratentorial at Midfrontal due to Suspect Falx Meningioma with Parasagittal Meningioma + Hypertension Stage II + DM type 2 + femoral-popliteal DVT underwent craniotomy tumor removal. During hospitalization, the patient received enoxaparin therapy for seven days, along with antihypertensive and antidiabetic medications before surgery.

Anesthesia Management

ECG, saturation, $EtCO_2$, CVP, and arterial blood pressure were monitored. Induction was performed with dexmedetomidine 1 mcg/kgBW (70 mcg) iv for 15 minutes, propofol 2 mg/kgBW (140 mg), lidocaine 1.5 mg/kgBW (100 mg) iv, rocuronium 0.8 mg/kgBW (55 mg) iv, xylocaine spray 10% on the oropharynx. ETT was placed, and control ventilation. Dexamethasone 10 mg and mannitol 40 gr was given. Maintenance anesthesia with propofol 50–150 mcg/kgBW/minutes, dexmedetomidine 0.2–0.8 mcg/kgBW/hour and rocuronium 9–12 mcg/kgBW/minute, analgesic intermittent fentanyl 25–50 mcg. The patient had a central venous catheter (CVC) placed at the right subclavian vein. The operation lasted 12 hours. It was bleeding 800 cc and 2500 cc of crystalloid fluid was given, and two units of PRC with a urine output of 1800 cc. During surgery,

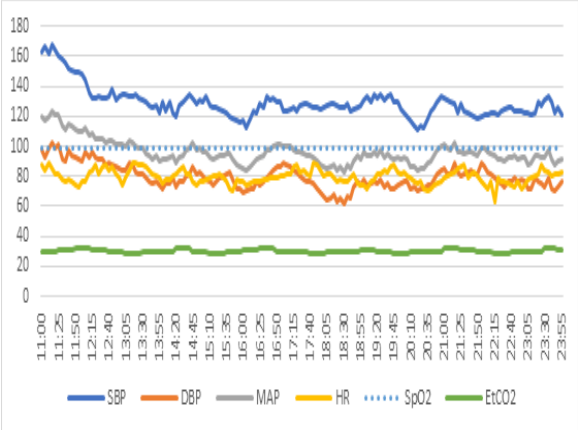

Figure 4. Haemodynamic Intraoperative

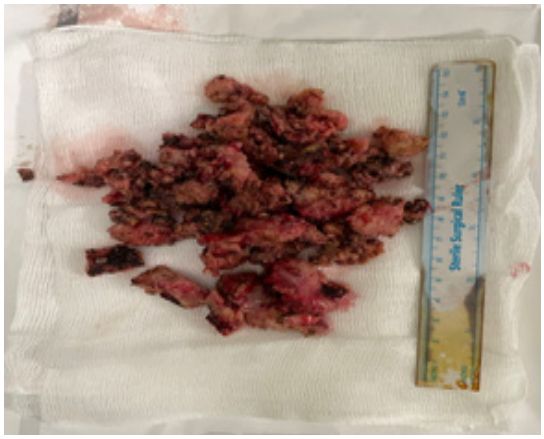

Figure 5. Intraoperative Finding

the hemodynamics were stable (Figure 4), and no complications were observed. Intraoperatively, the tumor was resectable at about 95% (Figure 5). The patient was transferred to the ICU and mechanically ventilated.

Post-operative Management in the ICU

The patient was treated in the ICU. The patient was given a ventilator with tidal volume 6mL/kgBW, respiratory rate 14 times per minute, and PEEP 5, and gradual weaning was performed. The patient was still sedated using propofol 25 – 100 mcg/kgBW/minute, dexmedetomidine 0.2 – 0.4 mcg/kg/hour, and paracetamol 1gr / 6 hour, ceftriaxone 1gr/12 hour, methylprednisolone 125mg/12 hour, citicoline 100mg/12 hours, mannitol 150 mL/6 hour, phenytoin 100 mg/8 hour, insulin 0.5 units/hour, enoxaparin 2x0.6 mL (s.c) (after 24 hours post-surgery) and KCl correction 25 meq target K >3.5. Blood gas analysis showed pH 7,36 pCO_2 38 PO_2 173 BE -2 HCO_3 23,4 SaO_2 99% and $etCO_2$ with 30–35 mmHg. The patient was extubated the next day, and enoxaparin therapy was resumed. Post-operative in ICU laboratory examination was performed, and Hb 12,2 g/dL was found. Serum glucose 82 mg/dL, D-dimer 1,66 µg/ml, and Kalium 2,8 meq/L. USG doppler after surgery was performed to evaluate DVT in the ICU. The hemodynamics were stable, and GCS 10 E4M4V2 and analgesics were continued. The patient was transferred to HCU three days post-operative.

III. Discussion

The term thrombosis refers to the formation of a blood clot (thrombus) in a blood vessel, and an embolus is a fragment of a thrombus that breaks off and travels in the blood until it occludes the narrowed blood vessel. Deep vein thrombosis (DVT) is caused by venous stasis (decreased blood flow), hypercoagulability, and blood vessel damage. These three factors are known as Virchow's triad. DVT generally occurs in veins in the lower extremities but can form emboli that block pulmonary blood vessels (PE). PE is a manifestation of the same disease entity as DVT and can collectively be referred to as venous thromboembolism (VTE).[15] A case study reported a 3.2 to 23% incidence of DVT in neurooncology patients, with a PE incidence of 1.8%. PE is a significant cause of death. It is estimated that the mortality rate for PE reaches 15 – 30%.16 Various patient-related factors have been proven to significantly increase the risk of developing DVT, including female gender, advanced age (over 60 years), hypertension, obesity, physical inactivity, and diabetes mellitus.5 The patient in this case is a 61-year-old woman with a history of uncontrolled grade II hypertension and type 2 diabetes mellitus. The patient complained of experiencing decreased consciousness for the last three months, which indicates a state of physical inactivity. DVT is generally treated by administering anticoagulants, thrombolysis, or installing an inferior vena cava (IVC) filter.8 In most patients with DVT occurring in

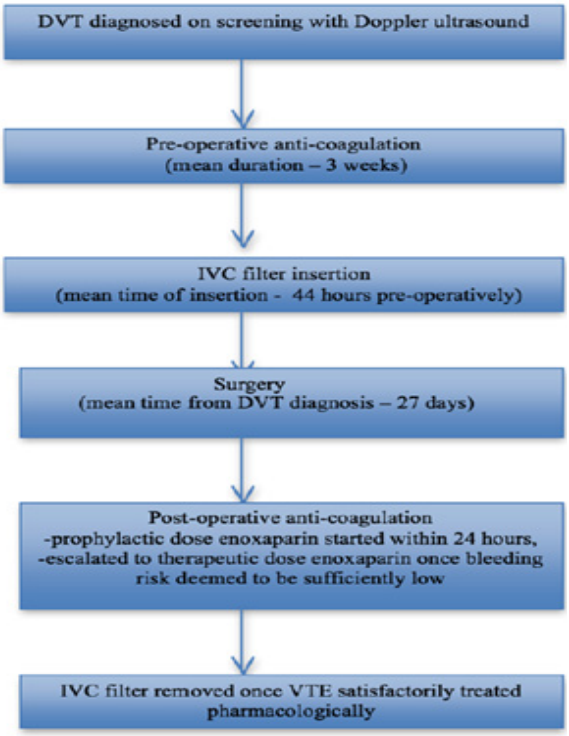

Figure 6. Perioperative Management Protocol with Preoperative DVT Findings

proximal veins, single anticoagulation therapy is recommended. Thrombolysis may be considered in patients with DVT limb-threatening (phlegmasia cerulea dolens) and in young patients who have a low risk of bleeding with DVT involving the common iliac and femoral veins.

Table 1. Wells Modified Score

Clinical Variable	Score
Clinical symptoms of DVT (leg swelling, pain on palpation)	3.0
Other diagnosis less likely than PE	3.0
Heart rate > 100	1.5
Immobilization (≥3 days) or surgery in the past 4 weeks	1.5
Previous DVT/PE	1.5
Hemoptysis	1.0
Malignancy	1.0
Modified Wells criteria interpretation	
PE likely	>4.0
PE unlikely	≤ 4.0

Thrombolytic therapy is also recommended in patients with PE accompanied by hemodynamic disturbances. IVC filters are beneficial in patients with DVT in the proximal veins and significant cardiopulmonary disease, as well as in patients with PE and hemodynamic compromise who have contraindications to anticoagulant therapy.[7] The patient was given an enoxaparin 2x60 mg 7 days before surgery (Figure 6). Patient with hypercoagulopathy (D-dimer 4,64 mcg) and significant loss of consciousness, emergency craniotomy tumor surgery was performed. So, enoxaparine stopped 24 hours before surgery.

Preoperative Management

Preoperative evaluation of patients undergoing craniotomy must be able to assess the presence or absence of intracranial hypertension. CT scans and MRI examinations can be used to assess whether there is brain edema, midline shift >0.5 cm, ventricular shift, or compression. Dexamethasone administration can be used to reduce the mass effect. Anticonvulsant and diuretic treatment was given as indicated. Neurological examinations such as level of consciousness and sensory and motor functions should be performed. Laboratory examination is also important to exclude hyperglycemia due to corticosteroids, electrolyte disturbances due to diuretics, or abnormal secretion of the hormone ADH. The intravenous fluid used is glucose-free isotonic crystalloid fluid. Hyperglycemia is often found in neurosurgical patients and is associated with brain tissue ischemia. In addition, hyperglycemia conditions must be corrected in the preoperative period.[17]

Before surgery, it is crucial to screen neuro-oncology patients for suspected DVT. A study shows that the proportion of DVT in neurooncology patients is 6%, and around 71% of them are asymptomatic. Screening is generally carried out by examining D-dimer levels. However, due to the relatively low specificity of this test, it should be followed by Doppler ultrasonography, which has a higher specificity. Ultrasound examinations are performed periodically before surgery to rule out further development of the already formed DVT as well as the formation of new thrombosis.[18]

The anticoagulant used must have a short duration of action to allow time for planning surgery. Anticoagulants such as enoxaparin can be given at a dose of 40 mg/day SC 24 hours before surgery, along with the use of anti-embolic stockings (AES). If anticoagulants are indicated, they may be administered low-molecular-weight Heparin (LMWH) and AES. AES is not recommended for patients with peripheral arterial disease, peripheral neuropathy of the feet, ulcers on the feet, severe foot deformity, allergies to AES ingredients, cellulitis, and massive edema of the feet.[19] The duration of enoxaparin administration varies based on the urgency and type of surgery. In patients with stable meningiomas, anticoagulants may be given for 1–3 months before surgery. Patients with more aggressive tumors are given anticoagulants for a shorter duration, namely around 1–3 weeks before surgery (Figure 6). Therapeutic doses of anticoagulants are administered up to 24 hours before surgery to minimize the risk of

thrombosis as well as intraoperative bleeding. The platelet count and coagulation profile were closely monitored in the preoperative period. On the other hand, for patients with intracranial tumors such as glioblastoma who are indicated to undergo surgery quickly (within 24 hours), anticoagulants are given after the operation is completed.[20] After consideration and discussion with the anesthesia and neurosurgery teams, it was decided that this patient would be treated with enoxaparin therapy for seven days, based on the consideration that the patient experienced a progressive loss of consciousness.

Wells-modified score DVT (Table 1) in the lower extremities is associated with an increased risk of PE. However, the symptoms may vary from patient to patient and are often invisible and nonspecific. The primary preoperative management anesthetist is very important for the early detection of PE and the prevention of mortality and morbidity in patients. Research designed to evaluate the utility of age-adjusted D-dimer limit levels combined with the modified Wells score as a clinical pre-test probability assessment (PTP) to predict PE in patients at risk of DVT.[10] In this patient, the Wells score was > 4, so the risk of PE could occur during surgery and after surgery.

Intraoperative Management

Premedication can be done using sedative agents or opioids. Administration of corticosteroids and anticonvulsants can be continued intra-operatively as indicated. In addition to monitoring using a standard monitor, monitoring of intra-arterial pressure and urinary catheterization is required. Sudden changes in blood pressure during anesthesia procedures, patient positioning, and surgical manipulations can be addressed with the help of continuous invasive blood pressure measurements. The arterial pressure transducer was calibrated at the level of the external auditory meatus or parallel to the circle of Willis rather than at the level of the right atrium to facilitate the measurement of cerebral perfusion pressure (CPP). In addition, it is necessary to carry out blood gas analysis to strictly regulate $PaCO_2$. Placement of central venous access (CVC) should be considered in patients requiring vasoactive agents. Intracranial pressure (ICP) monitoring can be done by placing transducers in various places, such as the ventricle, intraparenchymal, and subdural by a neurosurgeon. These transducers are also calibrated at the same level as arterial pressure transducers.[17]

Induction and intubation can cause an increase in ICP. The most frequently used induction technique is propofol. Ventilation is controlled until the induction agent is injected. Muscle relaxants are given to facilitate ventilation and prevent coughing, which can increase ICP. Esmolol 0.5 – 1 mcg/kg can be used to prevent tachycardia. Hypertension during induction can be managed by administering a beta-1 blocker or by deepening the anesthesia with additional propofol. Sevoflurane is effective in maintaining the autoregulation of cerebrospinal fluid (CSF) flow and limiting vasodilation. Hypotension is treated with phenylephrine. Frontal, temporal, and parietooccipital craniotomy procedures were performed in the supine position. The head is elevated 15–30 degrees to facilitate venous and CSF drainage. Maintenance of anesthesia can be carried out by administering inhalation anesthesia, intravenous anesthesia, or a combination of both. Normocarbic conditions must be maintained. $PaCO_2$ low levels can cause cerebral ischemia and disrupt the dissociation of oxygen from hemoglobin. Breathing patterns that cause an increase in airway pressure should be avoided because they can increase central venous pressure, leading to increased ICP and pulmonary disorders. Ventilation with a tidal volume ≤6 mL/kg is recommended.[17] In this case, the patient was intraoperative with $etCO_2$ at around 30 – 35 mmHg and $PaCO_2$ at about 32–37 mmHg. During the operation, a sign of pulmonary embolism was not found, such as a sudden drop of SpO_2, hypotension, arrhythmia, or decreased $etCO_2$.

The risk of thromboembolic events is further increased by other factors, such as the use of vasopressors, dehydration, and motor impairments both before and after surgery. Lastly, hypercoagulability brought on by malignant tumors or subarachnoid hemorrhage is

another condition that may raise the chance of a thromboembolic event. During the operation, there was bleeding of around 800 cc, and a transfusion of 2 units of PRC was carried out, taking into account that the bleeding was approaching 20% Estimated blood Volume (EBV). We managed the status volume of the patient intraoperatively to prevent the risk of thromboembolic events.

Post-operative Management

All patients, both without and with a history of DVT, were given a prophylactic dose of enoxaparin within 24 hours after surgery unless there were contraindications such as intraoperative bleeding. In particular, in patients with a history of DVT, the dose of enoxaparin can be increased to a therapeutic dose if the risk of intracranial bleeding is low. This is generally done up to 1 week after surgery. The use of AES was continued postoperatively until the patient was discharged.[20] Monitoring hemodynamics, increased intracranial pressure, and complications of DVT were performed in the ICU after surgery. USG Doppler after surgery was performed to evaluate DVT in the ICU. The patient was treated in the ICU for three days, then moved to a semi-intensive room with GCS 10 E4M4V2 with dexmedetomidine 0.1 mcg/hour, blood pressure 162/69 mmHg, pulse rate 93 x/minute, respiratory rate 20 x/minute, SpO_2 100% with nasal cannula 3 liters per minute.

IV. Conclusion

Perioperative management of patients with extensive brain tumors complicated by deep vein thrombosis requires a comprehensive and multidisciplinaryapproach. Bycarefully balancing the risks of anticoagulation with the potential for intraoperative bleeding, anesthesiologists can significantly minimize the risk of venous thromboembolism and enable patients to undergo necessary surgical interventions, including emergency craniotomy safely. In this high-risk patient population, proactive and attentive treatment during the perioperative phase is critical to improving outcomes.

References

1. Simanek R, Vormittag R, Hassler M, Roessler K, Schwarz M, Zielinski C, et al. Venous thromboembolism and survival in patients with high-grade glioma. Neuro Oncol. 2007; 1;9(2):89–95. Doi: 10.1215/15228517-2006-035

2. Silvani A, Gaviani P, Lamperti E, Botturi A, Ferrari D, Simonetti G, et al. Metabolic, electrolytes disorders and thromboembolic risk in malignant glioma patients. Neurol Sci. 2011;32(S2):229–31. Doi: 10.1007/s10072-011-0793-z

3. Semrad TJ, O'Donnell R, Wun T, Chew H, Harvey D, Zhou H, et al. Epidemiology of venous thromboembolism in 9489 patients with malignant glioma. J Neurosurg. 2007;106(4):601–8. Doi: 10.3171/jns.2007.106.4.601

4. Mathiesen T. Parasagittal meningiomas. Handb Clin Neurol. 2020;170: 93–100. Doi: 10.1016/B978-0-12-822198-3.00031-8

5. Riedl J, Ay C. Venous Thromboembolism in brain tumors: risk factors, molecular mechanisms, and clinical challenges. Semin Thromb Hemost. 2019;45(04):334–41. Doi: 10.1055/s-0039-1688493

6. Kaewborisutsakul A, Tunthanathip T, Yuwakosol P, Inkate S, Pattharachayakul S. Incidence and risk factors for venous thromboembolism following craniotomy for intracranial tumors: A cohort study. Asian J Neurosurg. 2020;15(01):31–8. Doi: 10.4103/ajns.AJNS_351_19

7. Chaichana KL, Pendleton C, Jackson C, Martinez-Gutierrez JC, Diaz-Stransky A, Aguayo J, et al. Deep venous thrombosis and pulmonary embolisms in adult patients undergoing craniotomy for brain tumors. Neurol Res. 2013;35(2):206–11. Doi: 10.1179/1743132812Y.0000000126

8. Ortel TL, Neumann I, Ageno W, Beyth R, Clark NP, Cuker A, et all. American

Society of Hematology 2020 guidelines for management of venous thromboembolism: treatment of deep vein thrombosis and pulmonary embolism. Blood Adv. 2020; 4(19): 4693-4738. Doi: 10.1182/bloodadvances.2020001830

9. Wang ZY, Wan YD, Liu XZ, Wang H, Jiang GY, Yang B. A single-center, randomized, double-blind study of 94 patients undergoing surgery for cerebral glioma to compare post-operative thromboprophylaxis with and without rivaroxaban. Med Sci Mon. 2021;28:1-7. Doi: 10.12659/MSM.934341

10. Singh S, Goel A. A study of modified Wells score for pulmonary embolism and age-adjusted D-dimer values in patients at risk for deep venous thrombosis. J Family Med Prim Care. 2023;12(9):2020–023. Doi: 10.4103/jfmpc.jfmpc_2455_22

11. Rinaldo L, Brown DA, Bhargav AG, Rusheen AE, Naylor RM, Gilder HE, et al. Venous thromboembolic events in patients undergoing craniotomy for tumor resection: incidence, predictors, and review of literature. J Neurosurg. 2020;132(1):10–21. doi: https://doi.org/10.3171/2018.7.JNS181175

12. Smith TR, Nanney AD, Lall RR, Graham RB, McClendon Jr J, Lall RR, et al. Development of venous thromboembolism (VTE) in patients undergoing surgery for brain tumors: Results from a single center over a 10year period. J Clin Neurosci. 2015;22(3):519–25. Doi: 10.1016/j.jocn.2014.10.003

13. Eisenring CV, Neidert MC, Bové DS, Held L, Sarnthein J, Krayenbühl N. reduction of thromboembolic events in meningioma surgery: a cohort study of 724 consecutive patients. PLoS One. 2013;8(11):e79170. Doi: 10.1371/journal.pone.0079170

14. Bagot CN, Arya R. Virchow and his triad: a question of attribution. Br J Haematol. 2008 ;143(2):180–90. Doi: 10.1111/j.1365-2141.2008.07323.x

15. Reyes N, Abe K. Deep vein thrombosis & pulmonary embolism. Centers for Disease Control and Prevention. 2023. Available from: https://wwwnc.cdc.gov/travel/yellowbook/2024/air-land-sea/deep-vein-thrombosis-and-pulmonary-embolism

16. Pandey A, Thakur B, Hogg F, Brogna C, Logan J, Arya R, et al. The role of preoperative deep vein thrombosis screening in neurooncology. J Neurosurg. 2018;130(1):38–43. Doi: 10.3171/2017.9.JNS17176

17. Butterworth JF, Mackey DC, Wasnick JD. Morgan & Mikhail's Clinical Anesthesiology. 7th ed. New York: McGraw Hill Education. 2018.

18. Tatarano S, Enokida H, Yonemori M, Eura R, Yoshino H, Nishimura H, et al. Significance of preoperative screening of deep vein thrombosis and its indications for patients undergoing urological surgery. Investig Clin Urol. 2021;62(2):166-71. doi: https://doi.org/10.4111/icu.20200300

19. Greater Glasgow and Clyde. Thromboprophylaxis for medical and surgical patients. National HealthService.2021.Availablefrom:https://handbook.ggcmedicines.org.uk/guidelines/cardiovascular-system/thromboprophylaxis-for-medical-and-surgical-patients

20. Gupta A, Dhiman D. Perioperative management of patient with pre-existing deep vein thrombosis for laparoscopic peritoneal dialysis catheter insertion: a case report. Ain-Shams J Anesthesiology. 2023;15(6): 1-4. doi: https://doi.org/10.1186/s42077-023-00301-1