JNI Jurnal Neuroanestesi Indonesia

Case Report

Case Report: Postoperative Complication Epidural Haematoma after Brain Tumour Resection

Kenanga Marwan Sikumbang[*)], Ayu Juniarti[**)], Aswin Febria[**)], Arif Budiman Susatya[*)], Oky Susianto[*)]

[*)]Staf Department of Anesthesiology and Therapy Intensive Faculty of Medicine and Health Sciences Lambung Mangkurat University in Banjarmasin, [**)]Resident of Specialist Study Program of Anesthesiology Faculty of Medicine and Health Sciences Lambung Mangkurat University in Banjarmasin

Received: May 22, 2024; Accepted: October 08, 2024; Publish: October 24, 2024

Correspondence: kmsikumbang@ulm.ac.id

Abstract

Postoperative intracranial haemorrhage is one of the most dangerous complications in cranial surgery, especially epidural haematoma although it is very rare with an incidence of 1.0%. The exact mechanism of occurrence is still unknown and with appropriate treatment can result in a good outcome. A 34-year-old female, 63 kg, who lost consciousness after extubation following resection of a meningioma in the parietooccipital region. In the recovery room, the patient regained consciousness and was transferred to the ICU for observation. The patient suddenly lost consciousness after 30 minutes in the ICU, reintubation was performed and a CT scan of the head showed an epidural hematoma after tumour resection. An emergency decompressive craniotomy was performed, with total intravenous anaesthesia (TIVA) combination of remifentanil 0.1 mcg/kg/min and thiopental 2 mg/kg/h, The operation lasted for one hour. The patient was admitted to the intensive care unit (ICU) for seven days under mechanical ventilation. The patient was extubated on the eighth day and transferred to the ward on the following day. There are several causes of epidural hematoma after brain tumour resection, namely sudden decrease in ICP, massive CSF drainage, uneven ICP distribution, coagulopathy factors, and excessive pin fixation. Excessive loss of CSF during surgery causes displacement of the brain and creates negative pressure in the remote area. In this case, it is suspected that the sudden decrease in ICP caused traction on the meningeal blood vessels, so that the negative pressure made the dura pulled and caused extradural haematoma. Conclusion: Postoperative epidural haematoma is a serious and relatively rare complication but if treated promptly, will result in a favourable outcome.

Keywords: Epidural hematoma after surgery, decreased ICP, brain tumour resection

J. neuroanestesi Indones 2024;13(2): 142–52

I. Introduction

Meningioma is the most common primary intracranial tumour. It is slow-growing, well-demarcated (non-infiltrating) and benign. Symptoms depend on the location of the tumour itself, and certain sites are closely associated with a highly explainable symptom complex. Meningiomas account for up to 14.3% – 19% of primary intracranial neoplasms.[1] Postoperative haematoma is a serious complication in cranial surgery, especially epidural haematoma (EDH). The incidence of postoperative EDH is approximately 1%. EDH may occur regionally, contiguously or distantly from the surgical site. There are some reports of EDH occurring after ventriculo-peritoneal shunt surgery or decompressive craniotomy. There are rarer cases of EDH following brain tumour surgery. The exact mechanism of EDH is unclear, but several hypotheses have been proposed. The most common hypothesis is a sudden drop in intracranial pressure that develops due to substantial volume excess loss of cerebrospinal

doi: https://doi.org/10.24244/jni.v13i3.602
ISSN (Print): 2088-9674 ISSN (Online): 2460-2302
This is an open access article under the CC-BY-NC-SA licensed: https://creativecommons.org/licenses/by-nc-sa/4.0/
JNI is accredited as Sinta 2 Journal: https://sinta.kemdikbud.go.id/journals/profile/796
Kenanga Marwan Sikumbang, Ayu Juniarti, Aswin Febria, Arif Budiman Susatya, Oky Susianto Copyright ©2024
How to cite: Sikumbang KM, et al, " Case Report: Postoperative Complication Epidural Haematoma after Brain Tumour Resection".

fluid during surgery. When EDH is suspected postoperatively, early diagnosis and appropriate treatment result in favourable outcomes.[2] We're report an epidural hematoma that occurred after brain tumor resection in woman 34 y.o with meningioma in parietooccipital region.

II. Case Report

History
Mrs NB, 34 years old, 63 kgbw, height 168 cm tall, BMI 22.3 kg/m2, occupation: teacher. Anamnese: chief complaint was headache since 1 year ago. History of illness: headache was felt increasingly aggravated in the last 1 month, she felt pressed in the middle of the head. Headache is relieved by analgetic such as paramex or mefinal. The patient also complained of blurred vision of the right and left eyes since 2 years, the right eye has a blurred vision, the patient went to the ophthalmologist and diagnosed as a cylinder of the right eye (0.5) and left eye (0.25). No complaints of limb weakness or balance disorders. The patient has no previous history of diseases such as hypertension, diabetes mellitus, and asthma.

Physical Examination
B1: Airway clear, snoring (-), gurgling (-), spontaneous breathing, RR 16 bpm, vesicular breath sounds, Rh (-/-), Wh (-/-), no retractions, SpO_2 98% with room air.
B2: Blood pressure 129/85 mmHg, CRT<2 seconds, warm, pulse 88 beats per minute, regular rhythm, murmur (-), gallop (-).
B3: GCS E4 V5 M6, pupils isochore 3 mm/3 mm, light reflexes (+/+), lateralisation (-).
B4: Spontaneous urination.
B5: Supple abdomen, bowel sounds (+) 8-10 times per minute.
B6: Temperature 36.5°C, haemiparesis (-/-), sensory (+|+), atrophy (-).
METs score >4 was obtained.
Cranial nerves examination: N.I +/+; N.II afferent pupil +/+ with a visus of 5/6 | 6/6; N.III efferent pupil +/+ with a narrowed right eye field of view and left eye field of view within normal limits; N.V corneal reflex +/+; N.VII no parese; N.VIII hearing within normal limits and no lateralisation; N.IX-X swallowing +; N.XII SCM (+), trapezius (+); N.XII no deviation.

Laboratorium Findings
Haemoglobin 12.5 g/dL and haematocrit 39.0%, leucocytes 6,400/ul, and platelets 324,000/ul. PT 10.3 seconds; aPTT 29.0 seconds; INR 0.93; SGOT 26 U/L and SGPT 45 U/L; ureum 19 mg/dl; creatinine 0.67 mg/dl, blood glucose 87 mg/dL; sodium 140 Meq/L, potassium 4.5 Meq/L; chloride 104 Meq/L.

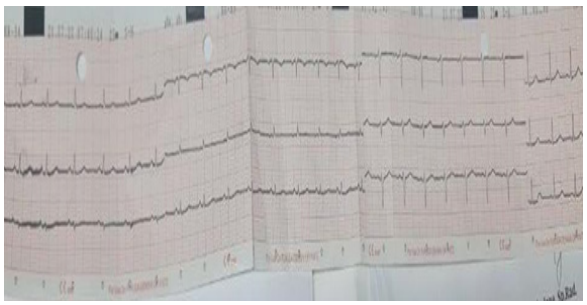

Figure 1. Electrocardiogram

The ECG showed a sinus rhythm with a heart rate of 80 beats per minute with no signs of ischaemia. The results of multi-sliced computed tomography (MSCT) of the head with contrast obtained, showed a supratentorial intraaxial semisolid lesion with a size of 69 mm x 58 mm with perifocal edema in the left basal ganglia to the left centrum semiovale, on post contrast appeared heterogeneous contrast enhancement; midline shift to the right by 10 mm; sulci gyri docked; bilateral lateral ventricles dilated; ventricles III and sisterna narrowed; ventricle IV normal; craniocerebral space does not appear dilated; calvaria intact. In conclusion, (1) supratentorial intraaxial semisolid mass in the left basal ganglia up to the left centrum semi ovale; (2) subfalcine herniation to the right by 10 mm; (3) bilateral lateral ventriculomegaly.

The patient was concluded with physical status ASA III, with medical problems of increased intracranial pressure (ICP) and narrow visual field. Surgical problems, namely, risk of bleeding, risk of infection, risk of prolonged surgery, risk of ventilator associated event (VAE), risk of increased ICP anaesthetic problems,

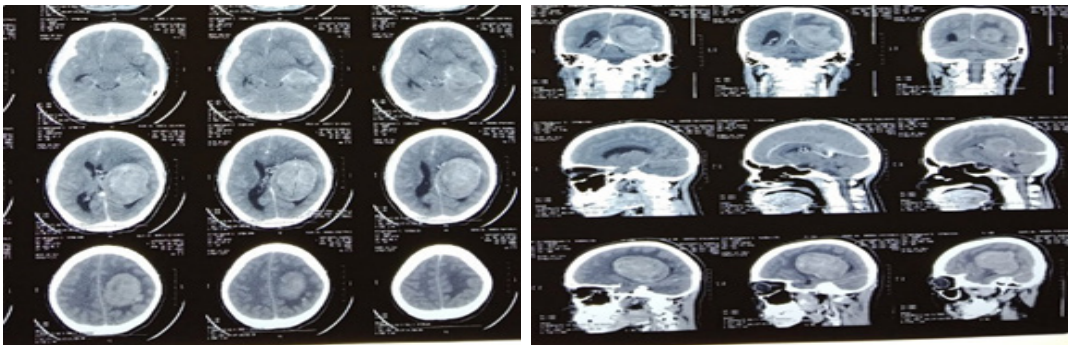

Figure 2. Head MSCT

namely, haemodynamic disorders, decreased functional residual capacity (FRC), risk of postoperative nausea and vomiting (PONV), postoperative pain, and risk of hypothermia.

Preoperative Anesthesia

The patient had six hours fasting before the craniotomy procedure. In the operating table, we applied noninvasive blood pressure (NIBP), pulse oximetry and temperature. Vital signs: blood pressure 128/66 mmHg, pulse 69 bpm, respiratory rate 14 beats per minute and oxygen saturation 99%. Access intravenous in the right arms and we prepared to insert central access after the patient intubate.

Intraoperative Anesthesia

We performed general anaesthesia with smooth intubation techniques with remifentanil loading 1mcg/kgbw for 1 minute and then continued with a maintenance dose of 0.1mcg/kgbw/mnt, thiopental continous at a dose of 2mg/kgbw/hour intraoperatively, and rocurunium at a dose of 0.8mg/kgbw and we combined TIVA technique with sevoflurane 1 vol% with flow oxygen 2 lpm, FiO_2 60%. The patient was operated in prone position.

Postoperative Management of Craniotomy

Tumor resection takes 3 hours long and because there's no massive bleeding and hemodynamic was stabil we planned to extubated and the patient was taken to the recovery room before transferred to the intensive care unit for re-evaluation after the craniotomy. Thirty minute in the ICU the patient became unconscious with GCS score

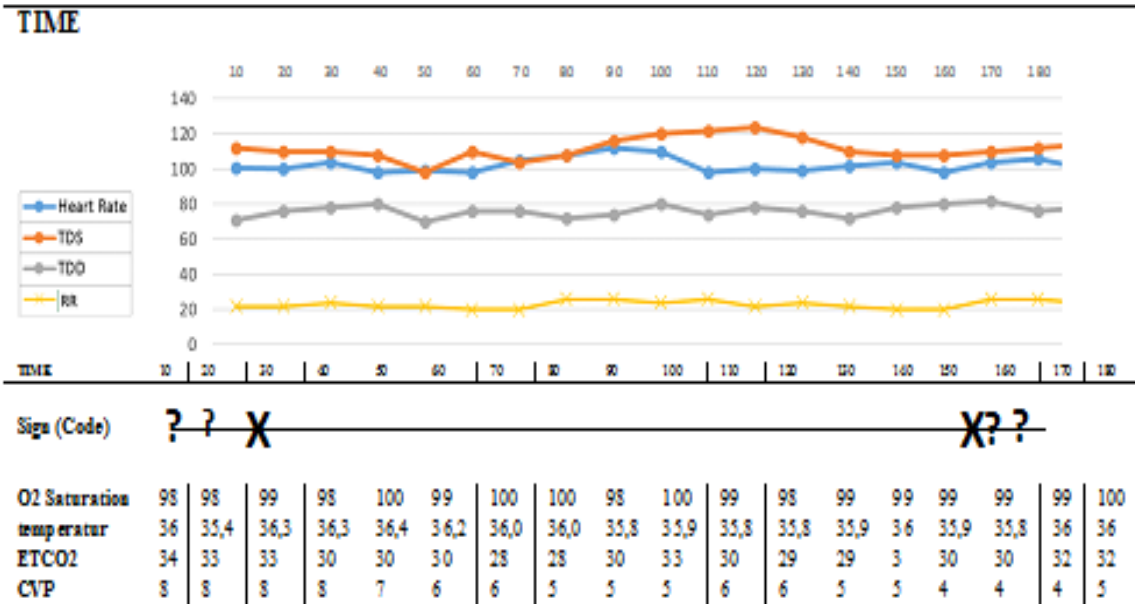

Figure 3. Vital Signs Monitoring Intraoperative

decrease (GCS E2V2M4), pupil anisochore 4mm/3mm, RC +/+, blood pressure 132/70 mmHg, pulse 107 bpm, respiratory rate 23 bpm. The patient was re-intubated with remifentanil, thiopental and rocuronium regiment with ETT 7/20 cm ventilator mode VC-SIMV FiO_2 60% Vt 400 i:e 1:2 rr 16 PEEP 5 Vte 395 – 408 Mve 6.25 with SpO_2 100% and head drain (+) 150 mL. The patient sent immediately to radiologic to perform CT-Scan examination without contrast. After CT-Scan examination, epidural haemorrhage was found and evacuation craniotomy was decided.

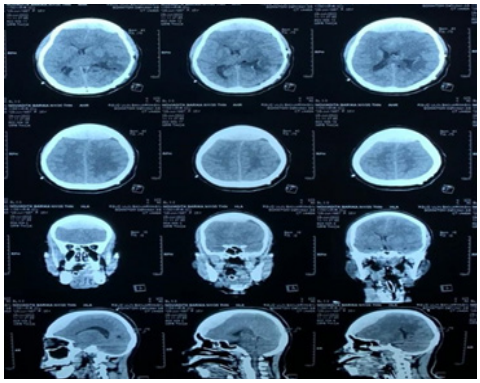

Figure 4. MSCT Examination Postsurgery

Anaesthetic Management for Epidural Hemorrhage Evacuation

The patient was given oxygen supplementation with ETT 7.20 cm, ventilator mode VC-SIMV; FiO_2 60%; Vt 400; i:e 1:2; RR 14; PEEP 5>; Vte 396-411; Mve 5.44; total RR 14x/min without additional breath sounds and SpO_2 100%. Blood pressure 102/68mmHg with MAP 79 mmHg, pulse frequency 78 bpm CVP 5.6 mmHg was measured on the monitor. General anaesthesia technique was performed with continuous total intravenous anaesthesia (TIVA) maintenance dose of 0.1 mcg/kgbw for the duration of surgery, thiopental at a dose of 2mg/kgbw/hour and roculax dose of 0.8 mg/kgbw and in supine position with a duration of 1 hour. Intraoperative bleeding was about 400 ml, with CVP measurement about 2 – 4 mmHg and because this is second operation we resuscitated the patient dan used norepinefrin to maintain cerebral perfusion pressure and to prevent potential hypotension which can occurs intraoperatively. Norepinefrin helps stabilize hemodynamic and prevent exarcebation of elevated ICP by

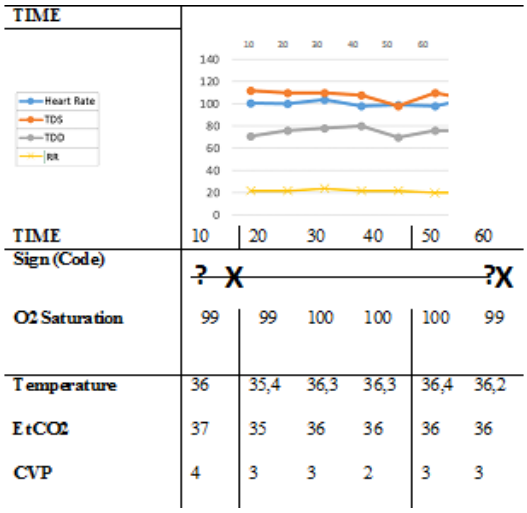

Figure 5. Monitoring Vital Signs Intraoperative during EDH Evacuation

ensuring blood flow to the brain remains optimal.

Postoperative Management of Evacuated Epidural Hemorrhage

After the epidural haemorrhage evacuation process was completed, we didn't extubated the patient and sent to intensive care unit; Blood pressure 102/68 mmHg with MAP 79 mmHg, pulse 78 bpm with norepinephrine 0.05 mg/kgBB/min CVP 5.6 mmHg. Ramsay score 5 on thiopental 125mg/hour, isochore pupils 2mm/2mm, RC +/+. Urine output 350cc / hour ~ 78 cc / hour, head drainage (+) 100 mL. The patient received NaCl 0.9% fluid therapy 2000 mL/24 hours, remifentanil 0.1mcg/ kgbw/min, paracetamol 3 times 1000mg, thiopental 125 mg/hour, head-up 30°, ulcer prophylaxis with omeprazole 2x40 mg IV, blood sugar (BG) 149 mg/dL, antibiotics ceftriaxone 2x40mg IV, phenytoin 3x100mg IV, and atorvastatin 1 time 40 mg per NGT. The patient was planned to maintain MAP >70 mmHg with the help of norepinephrine, maintain normothermy, perform complete blood tests, renal function, hepatic function, serum electrolytes, and blood gas analysis, and lactic acid. The patient was also monitored for vital signs, observed for increased intracranial pressure, fluid balance with a target urine output of 1.0 mL/kgbw/hour, and drainage production. The patient received treatment in the intensive care unit (ICU) for 10 days, overall there was a improvement in clinical

Table 1a. Follow up in ICU

Day 1	B1	B2	B3	B4	B5	B6
1	Airway on ETT 7/20cm, ventilator mode VC-SIMV; FiO_2 60%; Vt 400; i:e 1:2; RR 16; PEEP 5>; Vte 395-408; Mve 6.25; Rh (-) Wh (-) SpO_2 100%	BP 97/64 mmHg (73), HR 84x/min, murmur (-), gallop (-), CVP 9.24 mmHg	GCS Ramsay 5 on thiopental 2mg/ kgbw/ hour, pupils isochore 3mm/3mm, Light reflex (LR) (+/+), lateralisation (-)	UO 140cc/3h ~ 46cc/h, FB +612cc/3h	Soefl, Bowel sound (+), NGT residue (-)	Warm acral, T:36.7^{O} C, head drainage (+) 150 mL
2	Airway on ETT 7/20cm, ventilator mode VC-SIMV; FiO_2 60%; Vt 400; i:e 1:2; RR 16; PEEP 5>; Vte 389-447; Mve 3.8-4.6; ; Rh (-) Wh (-) SpO_2 100%	BP 95/65 mmHg (75), HR 77x/min, murmur (-), gallop (-), CVP 5.95 mmHg	GCS Ramsay 5 on thiopental 2mg/ kgbw/ hour, pupils isochore 3mm/3mm, Light reflex (LR) (+/+), lateralisation (-)	UO 2200cc/24 hours~91.6cc/ hour, FB-24.5cc/3 hours	Soefl, Bowel sound (+), NGT residue (+) clear	Warm acral, T:35.8^{O} C, head drainage (+) 10 mL/24 hours
3	Airway on ETT 7/20cm, ventilator mode VC-SIMV; FiO_2 40%; Vt 420; i:e 1:2; RR 16; PEEP 5>; Vte 450-574; Mve 5.57; Rh (-) Wh (-) SpO_2 100%	BP 124/70 mmHg (81), HR 90x/min, murmur (-), gallop (-), CVP 5.95 mmHg	GCS Ramsay 5 on thiopental 2mg/ kgbw/ hour, pupils isochore 3mm/3mm, Light reflex (LR) (+/+), lateralisation (-)	UO 2315cc/24h ~ 96cc/h, FB-175cc/24h	Soefl, Bowel sound (+), NGT residue (+) clear	Warm acral, T:35.8^{O} C, head drainage (+) 50
4	Airway on ETT 7/20cm, ventilator mode VC-SIMV; FiO_2 40%; Vt 420; i:e 1:2; RR 16; PEEP 5>; Vte 308-740; Mve 9.18; Rh (-) Wh (-) SpO_2 100%	BP 111/60 mmHg (79), HR 93x/min, murmur (-), gallop (-), CVP 5.95 mmHg	GCS Ramsay 5 on thiopental 2mg/ kgbw/ hour, pupils isochore 3mm/3mm, Light reflex (LR) (+/+), lateralisation (-)	UO 2300cc/24h ~ 97cc/h, FB-665cc/24h	Soefl, Bowel sound (+), NGT residue cloudy	Warm acral, T:35.8^{O} C, oedema +/+
5	Airway on ETT 7/20cm, ventilator mode VC-SIMV; FiO_2 40%; Vt 420; i:e 1:2; RR 16; PEEP 5>; Vte 308-740; Mve 9.18; Rh (-) Wh (-) SpO_2 100%	BP 111/60 mmHg (79), HR 93x/min, murmur (-), gallop (-), CVP 5.95 mmHg	GCS E2VxM4 on ETT, pupils isochore 2mm/2mm, LR (+/+), lateralisation (-)	2350cc/24h ~ 97cc/h, BC-665cc/24h	Soefl, , bowel sound (+), NGT residue cloudy	Warm acral, T:36.7^{O} C, oedema +/+

Table 1b. Follow up in ICU

Day 1	B1	B2	B3	B4	B5	B6
6	Airway on ETT 7/20cm, T-Pierce observed for 15 minutes and extubation performed	BP 127/63 mmHg (88), HR 105x/ min, murmur (-), gallop (-)	GCS E3VxM5 on ETT, pupils isochore 2mm/2mm, LR (+/+), lateralisation (-)	UO 1500cc/7h ~ 97cc/h, FB-897cc/7h	Soefl, Bowel sound (+) NGT attached residue (+) clear	Warm acral, T:37.4^{O} C, oedema +/+
7	RR 18-20x/ min, Rh (-) Wh (-) SpO_2 100% on NC 2 lpm	BP 115/70 mmHg (80), HR 95x/min, murmur (-), gallop (-)	GCS E3V5M5, pupils isochore 2mm/2mm, LR (+/+), lateralisation (-)	UO 2210cc/11h ~ 200cc/hour, FB-1180cc/11h	Soefl, Bowel sound (+), NGT residue (+) clear	Warm acral, T:37.5^{O} C, oedema +/+
8	RR 17-20x/min, Rh (-) Wh (-) SpO_2 100% on NC 2 lpm	BP 116/77 mmHg (88), HR 96x/min, murmur (-), gallop (-)	GCS E3V5M5, pupils isochore 2mm/2mm, LR (+/+), lateralisation (-)	UO 4615cc/24h ~ 192cc/h, FB-1391cc/24h	Soefl, Bowel sound (+), NGT residue clear	Warm acral, T:37.2^{O} C, oedema +/+
9	RR 17-20x/ min, Rh (-) Wh (-) SpO_2 100% on NC 2 lpm	BP 118/87 mmHg (96), HR 72x/min, murmur (-), gallop (-)	GCS E3V5M5, pupils isochore 2mm/2mm, LR (+/+), lateralisation (-)	UO 2260cc/24h ~ 95cc/h, FB-298cc/24h	Soefl, Bowel sound (+) NGT residue clear	Warm acral, T:37.0^{O} C, oedema +/+

Notes: BP= blood pressure; ETT= endotracheal tube; FB= fluid balance, GCS= glasgow coma scale; HR= heart rate; LR= light reflex; NC= nasal canule; NGT= nasogastric tube; RR= respiratory rate; UO= urine output

condition every day. Weaning from ventilator was carried out on the 7th day and extubated the next day. The patient's vital signs during the treatment process were always stable without any signs of shock or hypertension and we stopped nor epinefrin as soon we saw the hemodynamic was stabilize. Level of consciousness increase every day from Ramsay 5 on thiopental 4mg/ kgbw/ hour until on day 5 it could be evaluated to GCS E1VxM4, then on day 5 E2VxM4, on day 7 E3VxM5, on day 8 E3V5M5, and on day 10 GCS became compos mentis E4V5M6. We‘re preserve Nasogastric tube for enteral nutrition and oral medication like atorvastatin. Monitoring 24 hours include vital signs, fluid balance, head drainage production, sign of increase intracranial pressure and GCS score.

III. Discussion

Meningioma is the most common primary intracranial tumour. This type of tumour is slow-growing, well-demarcated (non-infiltrating) and benign.[1] The incidence of malignancy histologically is only around 1.7% of the total meningioma cases, but there are lesion similarities between haemangiopericytoma which is also fast-growing.[3] The symptoms depend on the

Table 2a. Laboratory Examination Results

Day 1	Laboratory	Therapy	Planning
1	Hb 10.2/ Leu 8400/ HCT 31%/ PLT 240,000	IVFD NaCL 0.9% 2000mL/24 hours, Remifentanil 0.1mcg/kgbw/ minute, Paracetamol 1gr/iv/18hr, Thiopental 125mg/hour, head up 30o, IV Omeprazole 40mg/iv/12 hr, Ceftriaxone 100mg/iv/8 hr, Phenytoin 100mg/8hr/iv, Atorvastatin 1x40mg	Maintain MAP >70 mmHg, maintain temperature 35-36^{o} C, check DL, OT/PT, Ur/Cr, SE, AGD, lactic acid, observe vital signs, increase intracranial pressure, fluid balance, observe drainage.
2	Hb 10.3/ Leu 8500/ HCT 32.2%/ PLT 164,000 OT/PT: 47/29 Ur/Cr: 22/0.63 Lactic acid: 1.5 BGA: Ph 7.360/ pCO_2 50.5/ pO_2 178/ HCO_3 28.6/ BE 7/ FiO_2 40/ PF Ratio 445	IVFD NaCL 0.9%: Asering (1:1 mL/24hrs), Entramix 6x150mL, Remifentanil 0.1mcg/kgbw/min, Thiopental 1mg/kgbw.hr, head up 30o, Omeprazole 40mg/iv/12hr, IV Ceftriaxone 100mg/8hr/iv, Phenytoin 100mg/8hr/iv, Dexamethasone 3x10mg, IV NB 5000/hr, IV Citicoline 3x500mg, Per NGT Atorvastatin 1x40mg.	Maintain MAP >70 mmHg, maintain temperature 35-36^{o} C, ventilator settings VC-SIMV; FiO_2 40%; VT 400; i:e 1:2; RR 20x/min PEEP 5, gut feeding 6x150mL, observe vital signs, increased intracranial pressure, fluid balance.observe drainage
3	Hb 10.4/ Leu 7800/ HCT 31.6%/ PLT 250,000 OT/PT: 91/83 Ur/Cr: 26/0.59 Lactic acid: 1.1 BGA: Ph 7.390/ pCO_2 52/ pO2 178/ HCO_3 31.5/ BE 7/ FiO_2 40/ PF Ratio 445	IIVFD NaCL 0.9%: Asering (1:1 mL/24hrs), Entramix 6x150mL, Remifentanil 0.1mcg/kgbw/min, Thiopental 1mg/kgbw.hr, head up 30o, Omeprazole 40mg/iv/12hr, IV Ceftriaxone 100mg/8hr/iv, Phenytoin 100mg/8hr/iv, Dexamethasone 10mg/8hr/iv, Citicoline 3x500mg, Per NGT Atorvastatin 1x40mg	Maintain MAP >70 mmHg, maintain temperature 35-36^{o} C, thiopental lowered 0.5mg/ kgbw/hour until tapering off while evaluating consciousness, increasing ventilator settings until observation of spontaneous breathing, observation of vital signs, increased intracranial pressure, fluid balance
4	Hb 10.4/ Leu 7800/ HCT 31.6%/ PLT 250,000 OT/PT: 91/83 Ur/Cr: 26/0.59 Lactic acid: 1.0 BGA: Ph 7.441/ pCO_2 46.2/ pO_2 184/ HCO_3 31.6/ BE 7/ FiO_2 40/ PF Ratio 460	IVFD NaCL 0.9%: Asering (1:1 mL/24hrs), Entramix 6x150mL, Remifentanil 0.1mcg/kgbw/min, Thiopental 1mg/kgbw.hr, head up 30o, Omeprazole 40mg/iv/12hr, IV Ceftriaxone 100mg/8hr/iv, Phenytoin 100 mg/8hr/iv, Dexamethasone 10mg/8hr/iv, Citicoline 500mg/8hr/iv, via NGT (Atorvastatin 1x40, Curcuma 3x1, VIP Albumin 2x1, KSR 3x1mmHg).	Tapering down remifentanil to 0.05 mcg/kgbw/min, weaning ventilator gradually by weaning RR to 12->10>8 if spontaneous is sufficient and tidal volume and minute volume are achieved until smooth extubating, maintain MAP >70 mmHg, maintain temperature 35-37^{o} C, observe vital signs, increased intracranial pressure, and fluid balance with a target UO of 1mL/kgbw/hour
5	Hb 10.4/ Leu 7800/ HCT 31.6%/ PLT 250,000 OT/PT: 91/83 Ur/Cr: 26/0.59 Lactic acid: 1.0 BGA: Ph 7.441/ pCO_2 46.2/ pO_2 184/ HCO_3 531.6/ BE 7/ FiO_2 40/ PF Ratio 460	IVFD NaCL 0.9%: Asering (1:1 mL/24hrs), Entramix 6x150mL, Remifentanil 0.1mcg/kgbw/min, Thiopental 1mg/kgbw.hr, head up 30o, Omeprazole 40mg/iv/12hr, IV Ceftriaxone 100mg/8hr/iv, Phenytoin 100 mg/8hr/iv, Dexamethasone 10mg/8hr/iv, Citicoline 500mg/8hr/ iv. Per NGT (Atorvastatin 1x40, Curcuma 3x1, VIP Albumin 2x1, KSR 3x1mmHg).	Tappering down remifentanil to 0.05 mcg/kgbw/min, weaning ventilator gradually by weaning RR to 12->10>8 if spontaneous is sufficient and tidal volume and minute volume are achieved until smooth extubation, maintain MAP >70 mmHg, maintain temperature 35-37^{o}C, observe vital signs, increased intracranial pressure, and fluid balance with a target UO of 1mL/kgbw/hour

Table 2b. Laboratory Examination Results

Day 1	Laboratory	Therapy	Planning
6	BGA: Ph 7.453/ pCO_2 46.4/ pO_2 199/ HCO_3 32.3/ BE 9/ FiO_2 40/ PF Ratio 487	IVFD NaCL 0.9%: Asering (1:1 mL/24hrs), Entramix 6x150mL, Remifentanil 0.1mcg/kgbw/min, Thiopental 1mg/kgbw. hr, head up 30o, Omeprazole 40mg/iv/12hr, IV Ceftriaxone 100mg/8hr/iv, Phenytoin 100 mg/8hr/iv, Dexamethasone 10mg/8hr/iv, Citicoline 500mg/8hr/iv, via NGT (Atorvastatin 1x40, Curcuma 1x1, VIP Albumin 2x1, KSR 3x1)	Extubation planning with NRM
7	BGA: Ph 7.453/ pCO_2 46.4/ pO_2 199/ HCO_3 32.3/ BE 9/ FiO_2 40/ PF Ratio 487	IVFD NaCL 0.9%: Asering (1:1 mL/24hrs), Entramix 6x150mL, Remifentanil 0.1mcg/kgbw/min, Thiopental 1mg/kgbw. hr, head up 30o, Omeprazole 40mg/iv/12hr, IV Ceftriaxone 100mg/8hr/iv, Phenytoin 100 mg/8hr/iv, Dexamethasone 10mg/8hr/iv, Citicoline 500mg/8hr/iv, via NGT (Atorvastatin 1x40, Curcuma 1x1, VIP Albumin 2x1, KSR 3x1)	Extubation with lidocaine 80mg and post-extubation observation of the patient with NC 2-4 lpm
8	Lactic Acid 1.5 BGA: Ph 7.437/ pCO_2 46.7/ pO_2 70/ HCO_3 31.6/ BE 7/ FiO_2 45/ PF Ratio 155	IVFD NaCL 0.9%: Asering (1:1 mL/24hrs), Entramix 6x150mL, Remifentanil 0.1mcg/kgbw/min, Thiopental 1mg/kgbw. hr, head up 30o, Omeprazole 40mg/iv/12hr, IV Ceftriaxone 100mg/8hr/iv, Phenytoin 100 mg/8hr/iv, Dexamethasone 10 mg/8hr/iv, Citicoline 500mg/8hr/iv, via NGT (Atorvastatin 1x40, Curcuma 1x1, VIP Albumin 2x1, KSR 3x1)	Administration of entramix 3x200 mL, filtered porridge 3x200 mL, observation of vital signs, administration of fluids with NaCl 0.9% 500 cc, laboratory tests, observation of increased intracranial pressure, observation of fluid balance with a target UO of 1mL/kgbw/hour
9	Hb 10.9/ Leu 9200/ HCT 32.8%/ PLT 473,000	IVFD Aminofluid 1000mL/24 hours, Drip KCL 25 mcg finished in 24 hours, Entramix 3x200 mL via NGT, enteral 3x200 mL via NGT, head up 30o, via NGT (Atorvastatin 1x20, Curcuma 3x1, VIP Albumin 2x1, HP Pro 3x1)	Observation of vital signs, observation of increased intracranial pressure, observation of fluid balance with a target UO of 1mL/kgbw/hour

Notes: Hb=hemoglobin; Leu=leucocyte; Hct: haematocrite, NGT= nasogastric tube; Plt=platelet; UO=urine output

location of the tumour itself, and certain locations are closely associated with complex symptoms that are highly explainable. Seizures may occur in patients with supratentorial meningiomas as a result of irritation of the cerebral cortex.[4] Meningiomas are fragile and bleed easily. Pre-operative embolisation and autologous blood donation may be helpful for operative management of the tumour.

The general principles of surgery on meningiomas are:[5,6] Early interruption of the blood supply to the tumour, Internal decompression (using ultrasonic aspirator or loop cautery), dissection of the tumour capsule from the brain by cutting and coagulating the vascular and arachnoid attachments while folding the tumour into the decompression area with minimal retraction of the adjacent brain, removal of attached bone and dura with tumour when possible. Certain operative positions are required to facilitate the operator and reduce complications during surgery, i.e. the head should be elevated ≈ 30° above the right atrium.

There are some exceptions in meningiomas which involving the anatomical superior sagittal

sinus (SSS), namely: [6]

- For tumors affecting the anterior third of the SSS, the position is semi sitting position until supine.
- For tumors of the middle third of the SSS, the position is lateral with the tumourside down and the neck tilted 45° over the shoulder.
- For tumors of the posterior third of the SSS, prone position

In some studies, it has been suggested that perioperative anaesthetic procedures have an important role in the incidence of metastasis and recurrence related to cancer cell signalling, immune response, and modulation of neuroendocrine stress response. Anaesthesia and analgesics can directly suppress antitumour immune function, although systemic stress responses due to surgery and perioperative pain can activate neuroendocrine cascades that inhibit natural killer cell (NKC) activity during the perioperative period. The main goal of intraoperative care is to maintain the physiological state of the brain, facilitate surgery, and correct any adverse effects of surgery and comorbidities to maintain overall patient homeostasis. This requires adequate monitoring of the patient during surgery, cardiorespiratory support, fluid therapy management, and application of knowledge regarding anaesthetic agents to brain physiology.[7]

Intracranial haematoma is the most dangerous complication in brain surgery, especially epidural haematoma. Epidural haematoma is a serious complication that can occur at the site of intracranial surgery. Epidural haematoma in postoperative patients is a rare case. The reported incidence of postoperative EDH is around 1%. EDH may develop regionally, adjacent to, or distant from the operated part of the brain. There are few reports of EDH occurring after VP-shunt surgery or decompressive craniotomy. EDH after brain surgery is most commonly reported in the posterior fossa of the surgery. The exact mechanism of EDH is still uncertain, but some hypotheses suggest that there is a sudden drop in intracranial pressure that develops due to cerebrospinal fluid volume loss during surgery.[2]

In general, the selection of anaesthetic drugs is based on their effects on the cardiovascular system, however, in neurosurgical patients, the effects on cerebral blood flow, cerebral blood volume, intracranial pressure, cerebrospinal fluid production and absorption, autoregulation, response to CO_2, and so on should be considered. The qualification of drugs for neuroanesthesia must be easy to control, for example, fast start of action, short duration of action, fast awakening from anaesthesia, so the term Fast-tract neuroanesthesia is known by using drugs that are Short Acting Fast Emergence (SAFE). In addition, the drug must also have a stable haemodynamic effect and intracranial homeostasis, not affecting neurophysiological monitoirng and having antinociception and brain protection effects.

Drugs that reduce intracranial pressure and cerebral blood flow from the intravenous induction drug class are the most reducing pentotal, then etomidate, propofol, midazolam, so the main choice is pentotal. All intravenous anaesthetic drugs decrease cerebral blood flow with the exception of ketamine which has the effect of increasing cerebral blood flow. Barbiturates decrease Ca influx, block sodium channels, inhibit free radical formation, decrease lactate, extracellular, glutamate and aspartate. Propofol increases glutamate excitotoxicity, increases neuronal damage and can cause propofol infuse syndrome (PRIS). In supratentorial tumour surgery, it is expected that the patient will wake up immediately and be extubated at the end of surgery in order to evaluate the surgical outcome and postoperative neurological function. The advantages and disadvantages of waking up immediately and allowing the patient to sleep post-surgery are still debated. Factors other than anaesthetic drugs that cause prolonged consciousness are large intracranial tumours, preoperative loss of consciousness, surgical complications (seizures, cerebral oedema, haematoma, pnemocephalus, vascular occlusion/ ischemia), electrolyte disturbances and hypothermia.

Post-surgery, patients can be admitted to the ward or to the ICU/Neuro ICU. In general, whether

or not a patient is admitted to the ICU depends on their preoperative GCS level, tumour size and location, and the presence of midline shift. All patients should be head up 30 degrees in a neutral position i.e. not tilted to the left or right, not hyperextended or hyperflexed to improve cerebral venous drainage. If necessary ventilate, maintain normocapnia. $PaCO_2$ <35 mmHg should be avoided for the first 24 hours after head injury. Control blood pressure within autoregulatory limits. Systolic should not be less than 90 mmHg. In therapeutic head injury when mean arterial pressure >130 mmHg Infuse with 0.9% NaCl, limit RL administration, colloids may be given. Haematocrit should be maintained at 33%. If Hb <10 g% give blood, in patients with cerebral ischaemia, the target Hb is 10 g%. Usually in healthy patients (no cerebral abnormalities) transfusion is given when Hb <8 g%. For seizure control, phenytoin can be given 10–15 mg/kg at a rate of 50 mg/minute. If a seizure occurs while giving phenytoin, give diazepam 5–10 mg intravenously (0.3 mg/kgbw) slowly for 1-2 minutes. Waking up from anaesthesia after supratentorial surgery should be smooth and gentle. The decision as to whether the patient should be awakened and extubated depends on the degree of preoperative consciousness, the site of surgery, the extent of cerebral oedema, and the amount of drugs administered.

In this case, after the craniotomy, the patient was found to have Ramsay score 5, i.e. the patient showed slow response to light glabellar tapping or loud auditory stimulus, 4mm/3mm anisochore pupils, LR +/+, blood pressure 132/70 mmHg, pulse frequency 107 beats per minute, respiratory frequency 23 beats per minute. These signs were then suspected as brain herniation. The patient was then proposed for a CT-Scan examination without contrast, and epidural haemorrhage was found. The bleeding condition was thought to be the cause of the brain herniation symptoms. Spontaneous bleeding after craniotomy can be influenced by several factors. Among them, based on several previous studies, it was found that position changes such as changing from supine to prone position during surgery can increase intracranial pressure. In a study conducted on patients with traumatic brain injury attached to a ventilator in the ICU, there was an increase in intracranial pressure to > 22 mmHg with a change of about 11 mmHg after various position changes were made. The study also found a decrease in cerebral perfusion pressure (CPP) to below 60 mmHg. Although the resulting changes in ICP and CPP are often transient, repeated acts of position change can be cumulative to intracranial hypertension and the potential for cerebral hypoperfusion to occur.[8]

The patient presented with epidural haemorrhage in the frontal region, with the surgical site based on the preoperative CT scan being a supratentorial intraaxial semisolid mass in the left basal ganglia to the left centrum semi ovale. The parietal and frontal lobes are the most common locations for EDH, and EDH at these locations may be caused by a loose dural fixation to the inner cranium. EDH does not always occur in the same location at the surgical site. Complications of EDH generally occur a few hours after surgery, but according to some literature, it can occur within hours to 3 days after surgery. Symptoms generally appear quickly in acute EDH, but if the haemorrhage is slow, the patient may be asymptomatic and the diagnosis may be delayed, leading to adverse outcomes. This patient was found to have decreased consciousness after 30 minutes in the ICU postoperatively.[9]

Epidural haematoma in postoperative patients is a rare case. The reported incidence of postoperative EDH is about 1%. EDH may develop regionally, contiguously, or distantly from the operated part of the brain. The period of occurrence of EDH after surgery can occur after 14 days of surgery, with an average lucid interval of 16 hours. Several hypotheses have been proposed in explaining the mechanism of delayed EDH, one of which is the loss of tamponade effect of the bleeding source. Rapid drainage of the cerebrospinal fluid during surgery is also thought to cause rapid epidural haematoma due to the separation of the dura from the calvarium and cause postoperative EDH at a site adjacent to the previous surgical site. High pressure from the suction drain in the epidural space may also play

a role in the mechanism of postoperative EDH.[10]

IV. Conclusion

A case report of postoperative epidural hematoma following tumour resection removal has been reported. Although the exact mechanism of remote postoperative EDH is still debated, we assume that multiple loose sutures and bleeding during surgery were possible causes in our case. Postoperative epidural haematoma is a serious and relatively rare complication. We report a remote case of EDH after brain tumour surgery. The patient was treated promptly and had an excellent outcome.

References

1. Ogasawara C, Philbrick BD, Adamson DC. Meningioma: A review of epidemiology, pathology, diagnosis, treatment, and future directions. Biomedicines. 2021;9(3): 1-23. Doi: https://doi.org/10.3390/biomedicines9030319.

2. Chung HJ, Park JS, Park JH, Jeun SS. Remote postoperative epidural hematoma after brain tumor surgery. Brain Tumor Res Treat. 2015;3(2): 132-37. Doi: 10.14791/btrt.2015.3.2.132

3. Maggio I, Franceschi E, Tosoni A, Di Nunno V, Gatto L, Lodi R, et al. Meningioma: not always a benign tumor. A review of advances in the treatment of meningiomas. CNS Oncol. 2021; 10(2): 1-16. Doi: 10.2217/cns-2021-0003
4. Huntoon K, Toland AMS, Dahiya S. Meningioma: A review of clinicopathological and molecular aspects. Front Oncol.2020; 2: 1–14. Doi: https://doi.org/10.3389/fonc.2020.579599.

5. Trobe JD. Comprehensive neurosurgery board review. J Neuro-Ophthalmology. 2006; 26, 74–75. Doi: 10.1097/01.wno.0000206248.72904.d3

6. Hanna Jr C, Willman M, Cole D, Mehkri Y, Liu S, Willman J, et al. Review of meningioma diagnosis and management. Egyptian J Neurosurg. 2023; 38(16): 1-9. Doi: https://doi.org/10.1186/s41984-023-00195-z

7. Siegmuller, C. 'Put a damper on' - rectify arterial line underdamping with an alternative to the air bubble injection method. Anaesthesia. 2021; 76(12): 1671-674. Doi: https://doi.org/10.1111/anae.15573

8. Harrois A, Anstey JR, Deane AM, Craig S, Udy AA, Mcnamara R, et al. Effects of routine position chnages and tracheal suctioning on intracranial pressure in traumatic brain injury patients. J Neurotrauma. 2020;37(20). Available from: https://www.liebertpub.com/doi/10.1089/neu.2019.6873

9. Yakar F, Dogan I, Bahadir B, Ozates MO, Ozgural O, Attar A, et al. Epidural hematoma: postoperative complication. Int Surg J. 2017;4(6): 2058. Doi: 10.18203/2349-2902.isj20172412

10. Laaidi A, Rafiq A, Tahrir Y, Hilmani S, Lakhdar A. Contralateral epidural hematoma after decompressive surgery: case report and systematic literature review. Ann Med Surg. 2022; 74: 1–5. Doi: 10.1016/j.amsu.2021.103233