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Original Article

Comparison of the Values of Inflammatory Parameters C-Reactive Protein, Neutrophil Lymphocyte Ratio, Platelet Lymphocyte Ratio between Scalp Block Ropivacaine 0.5% and Intravenous Dexmedetomidine in Craniotomy Brain Tumors

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Abstract

Background and Objective: Craniotomy surgery can increase the body's inflammatory response through the neuroendocrine system. neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and C-reactive protein (CRP) are biomarkers of inflammation and immunosuppression. Scalp block using ropivacaine and intravenous dexmedetomidine are commonly used analgesic techniques to attenuate perioperative inflammatory responses. This study aims to determine the comparison of inflammatory marker values between ropivacaine 0.5% scalp block and intravenous dexmedetomidine in brain tumor craniotomy
Subject and Method: This study used a single-blind clinical trial with a two-group posttest-only design. This study collected 36 research subjects who met the inclusion criteria. The subjects were divided into 2 groups, Group A received 0.5% ropivacaine scalp block and Group B received intravenous. Blood tests to assess inflammatory: markers were performed before and 24 hours postoperatively. The data obtained were analysed using SPSS version 20.
Results: The change value of CRP in the scalp block ropivacaine 0.5% group was 24.71± 7.25 mg/l, while the change value of CRP in the dexmedetomidine group was 61.02 ± 17.81mg /l. The change value of PLR in the scalp block ropivacaine 0.5% group was 50.57 ± 57.91 while the change value of PLR in the dexmedetomidine group was 105.26 ± 64.81. There was a significant change of CRP and PLR values in the scalp block group compared to the dexmedetomidine group ($p<0.05$). The change value of NLR in the scalp block group was 9.71 ± 5.75, while the change value of NLR in the demedetomidine group was 13.37 ± 5.55. There was no significant difference in the change value of NLR in the ropivacaine 0.5% scalp block group compared to dexmedetomidine ($p>0.05$).
Conclusion: Scalp block ropivacaine 0.5% has better results than intravenous dexmedetomidine administration in suppressing the inflammatory response in patients undergoing tumour craniotomy surgery.

Keywords: NLR, PLR, CRP, scalp block, ropivacaine, dexmedetomidine, craniotomy

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Introduction

Brain tumours are abnormal cell growth in or around the brain.[1] The most common types of brain tumors include meningioma (36.3%), pituitary tumor (16.2%), and glioblastoma (14.9%).[2] The incidence of brain tumors is recorded at approximately 6.4 per 100,000 people per year, with an overall five-year survival rate of 33.4%.[3] At Adam Malik Hospital Medan, the number of brain tumor patients has increased during the period 2017–2022, with a total of 182 people. In 2017 there were 99 people, 2018 there were 19 people, 2019 there were 7 people, 2020 there were 5 people with 3 of them died, 2021 there were 19 people with 6 died, and

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2022 there were 33 people.[4] Craniotomy can cause significant tissue damage leading to the production of proinflammatory cytokines and chemokines as well as a stress response that activates the hypothalamic-pituitary-adrenal axis and causes the release of cortisol and other stress hormones that modulate the immune response, all of which contribute to the development of inflammation.[5] The extent of the inflammatory reaction in the body can be seen through increased numbers of inflammatory cells, such as neutrophils and platelets, or through elevated levels of inflammatory biomarkers. Neutrophil-to-Lymphocyte Ratio (NLR), Platelet-to-Lymphocyte ratio (PLR), and C-Reactive Protein (CRP) are biomarkers of inflammation and immunosuppression that reflect the cellular immune response, both in trauma and in the acute phase.[5]

Ropivacaine is the drug of choice in scalp block due to its long duration compared to lidocaine and lower toxicity to the heart and nervous system compared to bupivacaine.[6] The scalp block procedure blocks C fibers and prevents activation of the inflammatory cascade.[7] Dexmedetomidine (DEX) is a highly selective α2-adrenergic agonist, which has sedative, analgesic and anxiolytic effects. Administration of dexmedetomidine can reduce systemic inflammation by stabilizing the sympathetic nervous system.[8] The goal of anesthesia in brain tumor surgery is to prevent secondary brain injury that may result from increased intracranial pressure, hypotension or hypertension. Brain tumor surgery with low levels of inflammation is usually associated with a better survival prognosis.[7]

Methods

This study used a single-blind clinical trial with a pretest-posttest two-group design. This study collected 36 s ample who met the inclusion criteria. The sample was divided into 2 groups, group A received 0.5% ropivacaine scalp block and group B received intravenous dexmedetomidine. The target population in this study included all patients who underwent brain tumor craniotomy surgery at Adam Malik Hospital Medan, TK II Putri Hijau Hospital Medan, and Haji General Hospital Medan. The study subjects were patients who fulfilled the inclusion and exclusion criteria. The inclusion criteria in this study were: 1) patients undergoing brain tumor craniotomy aged between 18–64 years; 2) Patients with ASA 1-2; 3) duration of surgery < 6 hours. Exclusion criteria in this study are: 1) patients with liver and kidney disorders; 2) diabetes mellitus; 3) systemic/local infection of the scalp; 4) history of steroid treatment in the last 2 weeks; 5) history of ropivacaine or dexmedetomidine allergy. Inflammatory marker screening was first performed prior to general anesthesia. The patient was then prepared for general anesthesia (standard monitor insertion) by recording pulse rate, blood pressure, ECG and oxygen saturation.

General anesthesia was achieved by intravenous administration of fentanyl 1-2 mcg/kg, propofol 1-2 mg/kg and rocuronium 0.6 mg/kg. Intubation with an appropriate ETT, then anesthesia was maintained with 2 vol% sevoflurane and combined oxygen-water bar with 50% fraction and maintenance dose of propofol 25- 100 mcg/kg/min, fentanyl 1 mcg/kg/hour and rocuronium at 0.15 mg/kg/hour via syringe pump. In group A, a scalp block using ropivacaine 0.5% was administered. The block was performed immediately after induction of anesthesia. After asepsis, a 10 ml syringe with a 23G needle was injected subcutaneously. The local anesthetic drug used was ropivacaine 0.5% in 2–3 ml, in each nerve.

Supraorbital nerve: the needle was injected along the orbital margin perpendicular to the skin, approximately 1 cm medial to the supraorbital foramen, supratrochlear nerve: infiltration parallel to the supraorbital nerve (medial to it), auriculotemporal nerve: infiltration along the zygomatic processus with injection 1-1.5 cm anterior to the tragus level, zygomaticotemporal nerve: infiltration at the supraorbital border to the posterior part of the zygomatic arch, large occipital nerve: infiltration about halfway between the occipital protuberance and the processus mastoideus, 2.5 cm lateral to the median nuchal line, small occipital nerve: infiltration along the

superior medial, 2.5 cm lateral from the large occipital. In group B, dexmedetomidine (DEX) was given at a dose of 0.5 mcg/kgBB iv loading dose followed by 0.25 mcg/kgBB/hour syringe pump. Blood pressure, mean arterial pressure and heart rate were recorded during surgery. A sudden increase in blood pressure or heart rate during surgery that exceeded 20% of the initial value was considered dangerous, and a bolus of fentanyl 1 mcg/kg was given as rescue analgesia. The inflammasome markers were re-checked 24 hours postoperatively.

Results

This study looked at the comparison of inflammatory marker values of NLR, PLR and CRP in brain tumor craniotomy patients with intravenous ropivacaine 0.5% and dexmedetomidine scalp block administration who were examined for inflammatory marker values before and 24 hours after surgery. Based on (Table 1), in the group given 0.5% ropivacaine scalp block, there were 14 males (38.8%) and 4 females (11.1%). In the group given dexmedetomidine, there were 8 men (22.22%) and 10 women (27.7%). The average age in the scalp block group was 43.89 ± 14.95 years, while in the dexmedetomidine group the average age was 48.17 ± 10.11 years. For BMI variables, the scalp block group had a value of 26.41 ± 2.57, while the dexmedetomidine group had a value of 25.57 ± 1.82. The average amount of bleeding in the scalp block group was 440 ± 88.05 ml, while in the dexmedetomidine group it was 515 ± 96.6 ml. The duration of surgery in the scalp block group was 251.94 ± 26.24 minutes, while in the dexmedetomidine group 252.22 ± 0.33 minutes. Based on data analysis, gender, age, BMI, amount of bleeding, and duration of surgery in both groups were homogeneous ($p > 0.05$).

Based on (Table 2), the results of the CRP value examination before surgery showed an average of

Table 1. Patient Demographic Characteristics

Variables	Scalp Block Ropivacaine 0.5 %	Dexmedetomidine	p value
Age (years); mean + SD	43.89 ± 14.95	48.17 ± 10.1	0.808*
BMI (kg/m2); mean + SD	26.41 ± 2,57	25.57 ± 1.82	0.219*
Total Haemorrhage (ml); mean+SD	440.00 ± 88,05	515.00 ± 96.60	0.318*
Duration of Operation (minutes); mean + SD	251.94 ± 26.24	252.22 ± 0.33	0.898*
Gender(%)			
Male	14 (38.8%)	8 (22.22%)	0.400**
Female	4 (11.11%)	10 (27.77%)	

BMI: Body Mass Index *Independent T-test, **Chi-Square Test

Table 2. CRP, NLR, and PLR Values in the Ropivacaine 0.5% Scalp Block Group

Inflammatory Response	Scalp Block Ropivacaine 0.5 %		p value*
	Pre Operative (T0)	24 hours Post Operative (T1)	
CRP (mg/L)	2.54 ± 0.77	27.26 ± 7.21	0.0001
NLR	2.08 ± 0.59	11.79 ± 5.86	0.0001
PLR	142.93 ± 33.13	193.51 ± 75.36	0.0001

* Paired T-test (mean ± SD), T0: before surgery, T1: 24 hours after surgery

Table 3. CRP, NLR, and PLR Values in the Dexmedetomidine Group

Inflammatory Response	Dexmedetomidine		p value*
	Pre Operative (T0)	24 hours Post Operative (T1)	
CRP (mg/L)	2.42 ± 0.59	63.44 ± 17.99	0.0001
NLR	2.54 ± 0.47	15.9 ± 5.41	0.0001
PLR	134.01 ± 36.30	239.27 ± 88.29	0.004

* Paired T-test (mean + SD), T0: before surgery, T1: 24 hours after surgery

2.54 ± 0.77 mg/L, while 24 hours after surgery the CRP value increased to 27.26 ± 7.21 mg/L, with a significant increase ($p < 0.05$). For NLR examination, the preoperative value was recorded at an average of 2.08 ± 0.59, while 24 hours postoperatively the NLR value increased to 11.79 ± 5.86, with a significant change ($p < 0.05$). In the PLR examination, the preoperative value was 142.93 ± 33.13 and after 24 hours postoperatively the average PLR increased to 193.51 ± 75.36, which also showed a significant increase ($p < 0.05$).

Based on (Table 3), inflammatory marker examination values in the dexmedetomidine group showed the results before surgery the average CRP value was 2.42 ± 0.59 mg/L and 24 hours after surgery the CRP value increased to 63.44 ± 17.99 mg/L, with a significant increase ($p < 0.05$). For NLR examination before surgery the mean value was 2.54 ± 0.47, while 24 hours postoperatively the NLR value increased to 15.9 ± 5.41, which also showed a significant increase ($p < 0.05$). In the PLR examination before surgery the mean value was 134.01 ± 36.30, while 24 hours after surgery the PLR value increased to 239.27 ± 88.29, with a significant increase ($p < 0.05$).

Based on (Table 4), the change in CRP value in the scalp block group was 24.71 ± 7.25 mg/L, while in the dexmedetomidine group the change in CRP was 61.02 ± 17.81 mg/L. There was a significant difference in the increase in CRP response before and after surgery between the two groups ($p < 0.05$). The change in NLR

Table 4. Changes in Inflammatory Response Values of CRP, NLR, and PLR in Both Groups

Inflammatory Response	**Scalp Block Ropivacaine 0.5 %**	**Dexmedetomidine**	**p value***
Δ(CRP(T1)-CRP(T0) (mg/L)	24.71 ± 7.25	61.02 ± 17.81	0.001
ΔNLR(T1)-NLR(T0)	9.71 ± 5.75	13.37 ± 5.55	0.060
ΔPLR(T1)- PLR(T0)	50.57 ± 57.91	105.26 ± 64.81	0.012

*Unpaired t test, T0: before surgery, T1: 24 hours after surgery

value in the scalp block group was 9.71 ± 5.75, while in the dexmedetomidine group it was 13.37 ± 5.55. There was no significant difference in the change of NLR value between the scalp block and dexmedetomidine groups ($p > 0.05$). The change in PLR value in the scalp block group was 50.57 ± 57.91, while in the dexmedetomidine group it was 105.26 ± 64.81. There was a significant difference in the change of PLR value between the two groups ($p < 0.05$)

Discussion

Tissue damage caused by craniotomy triggers the release of a substance by sensory nerve endings to produce inflammation in the target tissue, which will then become a neurogenic inflammatory response. the key to neurogenic inflammation is the activation of the main afferent nerve. Blocking the nerve through local anesthesia can reduce the release of substance P and block nerve transmission in the tissue causing the injury, thereby reducing the inflammatory response.[9] The results of this study showed a significant increase in CRP, NLR, and PLR values in

the group that received 0.5% ropivacaine scalp block preoperatively compared to 24 hours postoperatively. This suggests that the craniotomy surgical procedure increases the inflammatory response despite the intervention of 0.5% ropivacaine scalp block. Hartawan's study showed the use of scalp block with 0.25% levobupivacaine solution decreased CRP, NLR and PLR in glioblastoma patients undergoing craniotomy. Changes in CRP, NLR and PLR in both groups were significant at 72 hours post craniotomy ($p < 0.001$).[10] A study on patients undergoing elective craniotomy with and without scalp block (SB) using 0.5% ropivacaine showed that NLR levels increased over time and reached a peak at 24 hours postoperatively in both groups. The NLR values were 6.80 ± 1.17 in the SB group and 7.33 (5.83–8.09) in the non-SB group.[9]

Preoperative NLR and PLR levels were slightly higher in patients without scalp block (SB) compared to those with SB, with NLR values of 3.77 (2.44–5.17) versus 2.15 (1.74–4.84) and PLR values of 159 (119.2–201.2) versus 147.4 (114.2–187.7), respectively; however, the differences were not statistically significant.[11] Both groups showed increased NLR and PLR postoperatively, but patients with SB showed lower NLR and PLR levels on the third postoperative day (median NLR: 7.55 [2.87-13.79] and median PLR: 169 [117.4-253.4]) compared to patients without SB NLR: 11.85 [8.84-15.62] and median PLR: 244 [185.8-322.6], $P < 0.001$).

Patients with SB showed a lower inflammatory response both in systemic NLR and PLR and in the operative area compared to patients without SB.[11] Yang et al. found scalp nerve block with 0.75% ropivacaine showed a preventive effect on postoperative inflammation, as evidenced by lower plasma IL-6 concentrations within 6 hours after craniotomy for cerebral aneurysm and decreased CRP levels and increased IL-10 levels within 12 and 24 hours postoperatively.[12] Elimination of local anesthesia is followed by the return of nerve conduction mechanisms. In regional anesthesia, there is an anti-inflammatory effect due to blockade of C nerve fibers, thereby decreasing cytokine production and decreasing sympathetic nerve activity. Pain after surgery is caused mainly by local inflammation and activation of C nerve fibers which can be inhibited by decreasing cytokine production thereby limiting the inflammatory response after tissue trauma.[10] This study found a significant increase in inflammatory responses, both CRP, NLR and PLR, in the group given dexmedetomidine preoperatively compared with 24 hours postoperatively. This suggests that craniotomy surgical procedure increases the inflammatory response despite intravenous dexmedetomidine intervention.

The use of dexmedetomidine (DEX) at doses of 0.1–0.7 mcg/kg/hour showed that the highest CRP levels occurred on postoperative day 2 in both the DEX group (20.3 mg/dL) and non-DEX group (21.1 mg/dL). However, CRP levels during the 14-day observation period were significantly lower in the DEX group (5.6–20.3 mg/dL) compared to the non-DEX group (8.3–21.1 mg/dL) ($P = 0.03$). Dexmedetomidine administration was also associated with reduced plasma inflammatory cytokine concentrations.[13] Chen et al. found that postoperative NLR in both groups increased to a peak on postoperative day (POD 0) and then decreased gradually. In the dexmedetomidine (DEX) group at a dose of 0.3 μg/kg/hour, the overall postoperative estimated marginal mean (EMM) NLR was lower than the normal saline (NS) group [NS vs. DEX, 15.48 (13.22- 17.75) vs. 13.29 (11.68-14.90), $P = 0.122$].[14] The use of DEX may reduce serum levels of inflammatory factors in patients undergoing surgery. In addition, DEX improves immunosuppression resulting in better postoperative cognitive function outcomes, effective perioperative analgesia, and less opioid use.[15]

Intravenous administration of dexmedetomidine in postoperative patients can also reduce the inflammatory response by activating the cholinergic anti-inflammatory pathway, which in turn suppresses the proinflammatory response. This finding is consistent with previous studies showing a significant difference between patients receiving dexmedetomidine and the control group

in individuals undergoing craniotomy surgery.[16]

In this study, changes in NLR values preoperatively and 24 hours postoperatively (ΔNLR: NLR T1 - NLR T0) showed no significant difference between the ropivacaine 0.5% scalp block group and the dexmedetomidine group ($p > 0.05$). This result is in line with Desiree et al.'s study, which found no significant change in NLR values ΔT0-T1 and ΔT0-T2 (T0 = time after induction of anesthesia, T1 = 6 hours after incision, T2 = 24 hours after surgery). This non-significant result may be due to the blood sampling that was only done once after surgery without serial examination.[9] However, in CRP and PLR, there were significant changes in CRP (ΔCRP: CRP T1 - CRP T0) and PLR (ΔPLR: PLR T1 - PLR T0) values preoperatively and 24 hours postoperatively between the two groups ($p < 0.05$).

These results suggest that ropivacaine 0.5% has better results than dexmedetomidine in suppressing the inflammatory response. The inflammatory response generated by the craniotomy process is an important component of surgery. This response is necessary to restore normal physiology, but excessive response can lead to secondary brain damage and systemic inflammatory response syndrome (SIRS).[5] Excessive inflammation not only has adverse effects on wound healing, but is also thought to cause a range of complications, such as postoperative pain, fatigue, atrial fibrillation and cognitive dysfunction.[16]

Conclusion

There were significant changes in the inflammatory marker values of CRP and PLR in tumor craniotomy patients who received intravenous dexmedetomidine compared to the 0.5% ropivacaine scalp block group. Scalp block ropivacaine 0.5% showed better results than intravenous dexmedetomidine in suppressing the increased inflammatory response in brain tumor craniotomy patients.

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