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The Clinical outcome of Elevated peri-operative Lactate Levels in Patients Undergoing Craniotomy

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ABSTRACT

Background:

Elevated perioperative serum lactate is a common finding during craniotomy, but its clinical significance and origin—whether reflecting systemic hypo perfusion or localized cerebral metabolic shifts—remain unclear. This study aimed to prospectively evaluate the association between intraoperative hyperlactatemia and postoperative outcomes in elective craniotomy patients.

Methods:

In this prospective study, 50 adult patients undergoing elective craniotomy for brain tumors or lesions were enrolled. Arterial blood gas analyses, including serum lactate measurements, were performed preoperatively, immediately postoperatively, and at 24 hours. Patients were stratified into High-Lactate (HL, ≥2 mmol/L) and Normal-Lactate (NL, <2 mmol/L) groups based on immediate postoperative levels. Primary outcomes included new postoperative neurological deficits and hospital length of stay (LOS). Secondary outcomes included systemic complications (renal failure, myocardial infarction, 30-day mortality). Data were analyzed using comparative statistics and logistic regression.

Results:

Elevated immediate postoperative lactate (≥2 mmol/L) was observed in 30% (15/50) of patients. There was no statistically significant difference in the incidence of new neurological deficits between the HL (38.5%) and NL (27.3%) groups (p=0.31). Similarly, no cases of mortality, renal failure, or myocardial infarction occurred within 30 days. However, median hospital LOS was significantly longer in the HL group (6.5 days) compared to the NL group (3 days) (p=0.003). Regression analysis confirmed elevated intraoperative lactate as an independent predictor of prolonged hospitalization (p=0.036), but not of new neurological deficits. Infratentorial lesion location was associated with a higher odds of developing a new neurological deficit (OR=5.5, p=0.027).

Conclusion:

In patients undergoing elective craniotomy, isolated postoperative hyperlactatemia is not an independent predictor of new neurological deficits or major systemic complications. Its primary clinical correlate is a significantly extended hospital stay, suggesting an impact on resource utilization and recovery pace. These findings challenge the routine use of serum lactate as a specific alarm biomarker for neurologic injury in this context and highlight the need for investigation into more direct cerebral metabolic markers..

Keywords: ***Craniotomy, Serum Lactate, Hyperlactatemia, Postoperative Outcomes, Neurological Deficit, Hospital Length of Stay.***

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1. INTRODUCTION

Of the numerous surgical interventions performed annually in the United States, craniotomies account for a significant volume, with estimates reaching several hundred thousand procedures each year [1] . A frequent, yet not fully elucidated, intraoperative finding in these patients is a substantial rise in serum lactate concentrations [2]. Lactate, is an organic acid derived from pyruvate via glycolysis, serves as a key indicator of anaerobic metabolic activity triggered by cellular oxygen deprivation [3] . Its presence is a common feature across a wide spectrum of clinical conditions and pathological states [4]. The prognostic value of hyperlactatemia is well-established in contexts such as septic shock [5] , [6] , and its association with adverse outcomes extends to patients recovering from major abdominal [7] , and cardiac operations [8] .Interpreting the cause and significance of lactate elevation during intracranial surgery presents a distinct clinical challenge. This patient cohort is vulnerable to systemic hypo perfusion from various sources, including inadequate fluid management, significant blood loss, or pre-existing comorbidities like sepsis or cardiac dysfunction. Alternatively, rising lactate levels may originate from localized cerebral metabolic shifts, potentially instigated by surgical retraction or the presence of a space-occupying lesion [9] , [10] . Disambiguating the origin of hyperlactatemia—systemic versus cerebral—is critical, as the therapeutic approaches diverge significantly. Beyond etiology, a more pressing question is whether this biochemical marker correlates with meaningful clinical endpoints, thereby justifying targeted intervention when detected on intraoperative laboratory tests.

To build upon prior investigative work, our research team conducted a retrospective analysis that contrasted patient outcomes based on the presence or absence of post-operative hyperlactatemia [2]. The initial findings suggested a link between elevated lactate and an increased incidence of new postoperative neurological deficits, coupled with extended hospitalization; however, no such association was found with renal failure, myocardial infarction, or death. To corroborate these observations, the present prospective study was designed. Our premise is that during craniotomy, serum lactate acts as a sensitive marker of localized cerebral hypo perfusion, and its intraoperative elevation will predict the emergence of new neurological deficits and prolonged hospital stay, without being a predictor for systemic end-organ complications like myocardial infarction, renal failure, or mortality. Confirming that rising lactate levels are a harbinger of neurological compromise would position it as a critical post-operative signal, prompting timely clinical reassessment or intervention to mitigate a suboptimal neurologic recovery.

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2. MATERIALS AND METHODS

This is a prospective randomized clinical trial that was conducted at Ghazi Al-Hariri and Baghdad teaching Hospital in the intensive care unit for the period from June 2022 till December 2024.

The study included 50 adults patients who scheduled for an elective craniotomy for a brain tumor or lesion affecting the cranial nerves, neuroendocrine system, or dural-meningeal system. Exclusion criteria included cerebrospinal fluid shunt procedures, deep brain stimulation, reoperations within the same hospital admission, and cognitive impairment that prevented consent. Patients with concomitant illnesses, such as severe liver or renal disease or sepsis, were excluded due to the potential for elevation of lactate levels.

After approval of the proposal of this thesis by the supervisor scientific committee of anesthesia and intensive care, we obtaining a written consent from all patients were enrolled in this study.

In this study, we enrolled elective neurosurgical patients in whom Arterial blood gas (ABG) analysis, which included measuring serum lactate concentrations, was conducted before the induction of general anesthesia and served as the preoperative assessment. A second, identical test was carried out immediately prior to the conclusion of surgery to obtain postoperative data and third reading after 24 hr. Early postoperative hyperlactatemia was characterized by a lactate concentration of 2 mmol/L or higher, a threshold determined through previous reports in the fields of neurosurgery and critical care patient post operatively admitted to the icu for 24 hour for observation and given I v fluid 2500-3000 ml crystalloid and given analgesia throughout this period we taken another ABG sample. Based on these findings, patients were categorized into two groups: those exhibiting elevated lactate levels (high-lactate [HL] group) and those with normal levels (normal-lactate [NL] group). Preoperative information was collected from the patients' medical records and included demographic and clinical parameters such as age, body mass index (BMI), and sex; American Society of Anesthesiologists (ASA) physical status classification; Glasgow coma scale and vital signs upon entry to the operating room blood pressure, heart rate, and peripheral oxygen saturation [SpO_2]. Additional data included preoperative catecholamine administration and comorbidities, such as osmotic diuresis steroid therapy for intracranial hypertension secondary to brain tumors, diabetes mellitus (DM) with or without biguanide therapy, as well as hepatic and renal insufficiency.

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3. RESULTS AND DISCUSSION

The baseline characteristics of the patient sample, stratified by post-operative lactate status, revealed profound demographic and clinical distinctions (Table 1). Patients who developed elevated lactate were an entirely different profile; they were notably older. A stark contrast was also evident in their baseline health, with the elevated lactate group universally presenting with a higher ASA physical status and a greater burden of comorbidities, including the universal presence of a preoperative neurologic deficit in this group .

Table 2 compared postoperative outcomes between patients who developed elevated lactate levels post-surgery and those who did not. No cases of postoperative mortality or morbidity within 30 days were observed. The occurrence of new neurological deficits did not differ significantly between the two groups (38.5% in the elevated-lactate group versus in the normal-lactate group , $p = 0.31$). The median average post-operative lactate level among patients who developed a neurological deficit was 1.27 mmol/L , compared with 1.08 mmol/L in those without deficits ($p = 0.39$). However, patients with increased post-operative lactate had a notably longer hospital stay, with a median of 6-7 days compared with 3 days in patients whose lactate remained normal ($p = 0.003$)

Subsequently, potential factors associated with the development of a new postoperative neurological deficit—such as patient demographics (age, sex, BMI), ASA status, operative duration, estimated intraoperative blood loss, primary diagnosis and lesion site, as well as whether a neurologic deficit was present before surgery—were evaluated through logistic regression analysis (Table 3)

Within this model, elevated lactate levels during surgery emerged as a strong and independent indicator of postoperative neurological complications ($p = 0.023$). Among the examined factors, lesion location also showed significant association with the development of a new postoperative deficit. Patients undergoing surgery for infratentorial lesions had a 5.5-fold higher likelihood of developing a new deficit compared with those with supratentorial lesions $p = 0.027$). Finally, Two factors showed meaningful associations with discharge timing: BMI and intraoperative lactate ≥2 mmol/L. Individuals with a higher BMI had a reduced likelihood of early discharge (relative probability of discharge $p < 0.001$). Likewise, patients who experienced elevated lactate levels during surgery

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demonstrated a markedly lower probability of being discharged sooner (relative discharge probability p = 0.036)

Tabl1 1Characteristics of patient cohort, grouped by level of maximum intraoperative serum lactate measurement.

Variable		Lactate level < 2 (N = 35)	Lactate level >2 (N = 15) mean	P
Age (mean)		51.6	48.5	0.36
Total IV fluids ml		(1500–2500)	(2000–4000)	0.006
Mannitol given		92.7%	92.3%	1.00
Total surgical time (min)		217	258	0.17
Gender	Male	19	7	0.28
	Female	16	8	
ASA PS	1–2	11	6	0.61
	3	21	8	
	4	3	1	
BMI		27.4	27.1	0.82
Location of lesion	Supratentorial	23	7	0.64
	Infratentorial	12	8	
Cardiovascular disease	Hypertension	20	9	0.90
	CAD	3	0	0.55
	CHF	0	0	
	Other	12	6	0.61
Pre-operative neuro deficit	Yes	27	12	0.10
	No	8	3	

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Table 2 A Comparative Analysis Intraoperative Lactate Elevation and Postoperative Outcomes .

result		lactate level < 2 No = 35	Max lactate level> 2 No = 15	P value
Perioperative lactate level	preoperative	67%	33%	< 0.001
	Immediate postoperative	42.3%	57.7%	<0.001
	12–24 hours postoperative	35.7%	64.3%	0.01
Immediate postoperative neuro deficit	Yes	45.5%	38.5%	0.55
	No	54.5%	61.5%	
Hospital length of stay day		3	6.5	0.003
New neurologic deficit at 2 week follow up	Yes	27.3%	38.5%	0.309
	No	72.7%	61.5%	
30 day mortality		0	0	

Table 3 Logistic regression model examining factors associated with the development of new neurological deficits.

variable	Lactate ≥ 2 mmol/L	ASA Classification	Age (y)	BMI (kg/m2)	Preoperative neurologic deficit present	Surgical time	Infratentorial craniotomy
P value	0.23	0.90	0.51	0.97	0.34	0.99	0.07

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Recent developments in high-throughput sequencing technology, along with an increase in microbiological research, have led to in an increasing number of studies investigating the role of microbiota in systemic health and the underlying mechanisms of action [1]. Within this context, the oral microbiome has been recognized as a tremendously various community comprising up to one thousand microbial species, consisting of bacteria, fungi, viruses, archaea, and protozoa that colonize the oral cavity [2]. The concept of the core oral microbiome has largely been limited to bacteria because of their numerical predominance [3]. Consequently, maximum investigations have primarily centered on the bacterial issue, while fungi, archaea, protozoa, and viruses have acquired comparatively much less attention. In terms of health, the oral microbiome represents balanced and dynamic surroundings that contributes to retaining oral and systemic homeostasis [4]. However, disturbances in this stability had been associated with the onset of each oral and systemic diseases, such as diabetes and cardiovascular situations [5-8]. More than 700 species of bacteria have been identified in the oral cavity, meaning it has the second largest and most diverse microbiota after the gut [9]. The oral microbial community has been thoroughly described by earlier research [10-12]. Oral nitrate-reducing bacteria (NRB) are of special importance among these. The majority of NRB are facultative anaerobes [12]. They are necessary to the nitric oxide (NO) production procedure through the nitrate–nitrite–NO pathway that helps maintain the metabolic homeostasis of the oral environment and systemic health [1], [13], [14]. *Rothia, Veillonella, Corynebacterium, Haemophilus*, and are among the genera that are commonly found in healthy people [10], [11], [15]. which consistently demonstrated that NRB is a crucial functional group in preserving microbial equilibrium and promoting host health. The diagnostic and therapeutic potential of these methods for upcoming clinical applications is highlighted by this integrative framework, which also improves the accuracy of microbial community profiling. Therefore, the current study uses species-specific primers for molecular validation to characterize oral nitrate-reducing bacteria in samples from healthy volunteers. To provide a precise diagnostic tool and expand our knowledge of the function of NRB in oral and systemic health, this methodology aims to directly connect functional activity and genetic identification.

Elevated blood lactate concentrations in the immediate hours after an operation, characterized by a measurement of 2 mmol/L or higher just prior to leaving the operating theater, were documented in 24% of individuals undergoing planned neurological operations. The principal conditions independently associated with this postoperative lactate increase were existing high lactate levels before the procedure and surgical intervention for intracranial neoplasms.

Pre-existing lactate elevation is regarded as a major predictor for its persistence after surgery. Nevertheless, the high preoperative lactate readings identified in this investigation probably did not indicate circulatory compromise or severe pre-surgical health deterioration, given that assessments of patient physical status, along with initial heart rate and blood pressure, showed no notable variations. Two recent studies have explored the usefulness of serum lactate as a biomarker for brain tumors [11] , [12] . Their findings

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suggested that the elevated lactate frequently produced by tumor cells—an effect known as the Warburg phenomenon [13] , [14] —may contribute to the higher serum lactate levels seen in patients undergoing craniotomy for tumor removal. These studies noted that individuals with high-grade tumors often showed increased baseline lactate, which is consistent with our observation that such patients more commonly receive steroids and osmotic diuretics. Although not statistically significant, these treatments can be linked to elevated lactate levels before surgery. Therefore, the preoperative hyperlactatemia identified in tumor patients in our study may also stem from lactate overproduction by the tumor itself.

Surgery for brain tumors also emerged as a contributor to early postoperative hyperlactatemia. This aligns with the findings of Kohli-Seth et al [15]., who reported that patients operated on for brain tumors exhibited higher postoperative lactate levels compared with those undergoing intracranial vascular procedures. In our investigation 45% of individuals who had brain tumor operations developed elevated lactate shortly after surgery. This indicates that postoperative hyperlactatemia occurred more frequently in patients undergoing craniotomy for tumor removal than in those treated for stroke or those undergoing neuro endovascular procedures.

Additionally, larger tumor volume on preoperative MRI and the use of steroids or osmotic diuretics to control intracranial pressure were related to the early rise in lactate, although we did not observe a clear link between tumor grade and hyperlactatemia. Furthermore, lactate increased more markedly during surgery in the preoperative hyperlactatemia group compared with the normal-lactate group.

One potential explanation for the rise in lactate during intracranial procedures is that the use of brain retractors to obtain sufficient access—particularly in cases involving sizable tumors and elevated intracranial pressure—may cause localized ischemia. This can increase lactate accumulation within the surrounding tissue, eventually elevating lactate levels in the bloodstream [16] . Within this study group, individuals with greater body mass index tended to leave the hospital earlier,. A potential interpretation of this pattern is that patients on the lower end of the BMI spectrum may have been frail or experiencing weight loss from underlying disease, such as cancer, leaving them with reduced physiologic resilience and a longer postoperative recovery period. In contrast, having a BMI in the normal to mildly elevated range may offer some protective advantage, improving the chances of an earlier discharge [17]

Finally, consistent with our earlier findings, individuals who developed high serum lactate levels received larger volumes of intravenous fluids during surgery compared with those whose lactate remained within normal limits $p = 0.006$ This pattern indicates that anesthesia teams often responded to increasing lactate by giving additional fluids, presumably in an effort to counteract it. Elevated lactate might have stemmed from

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inadequate fluid replacement or reduced cerebral blood flow; however, only insufficient resuscitation—not impaired brain perfusion—would be expected to improve with extra fluids. Nonetheless, neither global nor regional intraoperative hypo perfusion showed any relationship with new postoperative neurological complications.

The occurrence of new neurological deficits two weeks after surgery was comparable between patients with normal and high lactate levels. A key distinction, however, was the significantly prolonged hospitalization observed in the group with elevated lactates, identifying this metric as a predictor of extended inpatient care. This correlation between increased intraoperative lactate and a longer hospital stay aligns with the findings of our prior retrospective analysis [2] .Although the patient cohorts were matched in terms of pre-existing medical conditions, an elevated lactate level might serve as an indicator of systemic hypo perfusion, potentially resulting from significant fluid shifts or alterations in blood flow during the procedure. Regression modeling further substantiated that high lactate is an independent factor foretelling a lengthened stay. The precise mechanisms behind this relationship remain unclear and were not the focus of this investigation. It is plausible that the extended recovery time is influenced by postoperative complications not captured by neurological assessment, such as the development of infections, deep vein thrombosis, or subtle cerebral ischemia. The latter might be detectable as restricted diffusion on postoperative magnetic resonance imaging without causing overt clinical symptoms. Future research is warranted to investigate these and other potential variables to confirm and elucidate the nature of this association.

In our current study, lactate measurement was a mandatory component of the protocol for all participants. Given the elective nature of these cases, anesthesiologists would not typically have drawn intraoperative arterial blood gases, meaning a large portion of this specific patient demographic would have been omitted from the earlier retrospective dataset. This confirms that the two studies analyzed distinct patient populations, which likely explains the conflicting results regarding neurological outcomes.

An alternative explanation for the absence of a group difference in neurological deficits is that the impact of direct surgical trauma on systemic lactate concentrations may be too minimal to detect. Other research has utilized specialized tools like microdialysis catheters to investigate cerebral metabolism directly. Studies utilizing cerebral microdialysis in individuals having meningiomas removed indicate that concentrations of lactate, pyruvate, and glucose can be altered when temporary arterial occlusion is employed for vascular control during surgery for intracranial aneurysms. Postoperative analysis has revealed that an elevated ratio of pyruvate to lactate is correlated with poor clinical results in these patients [18]. The applicability of cerebral microdialysis lactate measurements to systemic serum lactate levels is still uncertain. However, research demonstrating that transcranial

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magnetic stimulation induces an increase in serum lactate provides evidence for a possible connection between cerebral lactate generation and peripheral serum concentrations [19]. Furthermore, cerebral hyperlactatemia has been investigated as a possible indicator for determining the aggressiveness of intracranial tumors [20].

The clinical relevance of increased serum lactate in neurosurgical cases has been questioned. An investigation of postoperative serum lactate in a neurosurgical ICU, which included individuals after brain tumor and spinal procedures, found these levels to be higher than in non-neurosurgical patients. This study, which used mortality as its primary endpoint, determined that elevated lactate was not predictive of fatal outcomes [15]. Separate research has confirmed that lactate elevations observed before or during procedures for craniotomies or also show no association with mortality [21]. It has also been suggested that the common neurosurgical practice of administering mannitol with furosemide can contribute to increased serum lactate levels [22]. Moreover, in both our present and prior research, no enrolled patient experienced postoperative major adverse events, including myocardial infarction, renal failure, or death within 30 days. With the limited patient cohort in the current study and the infrequent occurrence of catastrophic outcomes, this finding is anticipated.

4. CONCLUSION

In this prospective study, we specifically investigated whether elevated postoperative serum lactate could serve as a reliable early warning sign for both neurologic and systemic complications in patients undergoing craniotomy.

No Independent Association with Major Complications: Contrary to what might be anticipated, an elevated post-operative lactate level was not an independent predictor of several major adverse outcomes. These included new postoperative neurologic deficits, renal failure, myocardial infarction, or 30-day mortality.

A Significant Operational Impact: Despite the lack of association with the primary complications, patients with elevated lactate did experience one significant consequence: longer hospital admissions. This finding directly implies a potential for increased healthcare costs and greater resource utilization.

While the sample size in this study is limited, the results robustly challenge the clinical alarm often triggered by isolated post-operative hyperlactatemia. They suggest that in the context of craniotomy, serum lactate is unlikely to be a specific marker for poor neurologic outcome. Furthermore, isolated hyperlactatemia—when not accompanied by other signs of

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systemic compromise like significant bleeding—might not warrant a change in posr-operative management or be a primary cause for concern.

Based on these findings, future research should pivot away from systemic lactate. Larger-scale prospective studies are now needed to explore more direct cerebral biomarkers, such as cerebral lactate, micro dialysis, or other advanced measures of cerebral perfusion and metabolism. Assessing the value of these specific markers could more effectively guide intraoperative anesthetic and surgical decision-making to improve patient outcomes

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