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Perioperative biochemical parameters in patients with vestibular schwannoma

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ABSTRACT

The correlation of cardiovascular and humoral responses to neurosurgical trauma near the cranial nerves is interesting for predicting of the postoperative period.

Objective. To study the correlation between cardiovascular and humoral responses to neurosurgical intervention for vestibular schwannoma (VS) by comparing changes in blood parameters during the intraoperative period.

Material and methods. The study included data of 92 neurosurgical patients who underwent surgery for vestibular schwannoma. The patients were divided into two groups: 46 patients without vegetative reactions during tumor removal; 43 patients who had type 1 vegetative reactions; 3 patients had type 2 vegetative reactions, but the last group for the small number of patients was excluded from statistical analysis. Biochemical indicators were studied, including glucose, lactate, urea, uric acid, total protein, albumin, total bilirubin, cholesterol, creatinine, creatine kinase, myoglobin, lactate dehydrogenase (LDH), alanine aminotransferase (ALT), aspartate aminotransferase (AST), amylase, iron, C-reactive protein (CRP), sodium, and potassium at various stages of the perioperative period.

Results. The level of uric acid in the plasma of patients in group 1 on the first postoperative day was higher than in patients in group 2. Creatine kinase levels increased in all groups. C-reactive protein exceeded the reference values 4—5 times on the first postoperative day.

Conclusion. Dynamics of changes in uric acid, CRP, glucose, and lactate levels are the result of the body's humoral response to multifactorial influences during neurosurgical intervention, most pronounced in combination with type 2 vegetative reactions, which are a consequence of hypercatecholaminemia.

Keywords: *vegetative reactions, vestibular schwannoma, uric acid, glucose, perioperative period.*

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Introduction

Vestibular schwannoma (VS) arises in internal auditory canal between myelin-producing oligodendrocytes and Schwann cells. VS accounts for over 30% of posterior cranial fossa tumors and up to 90% of cerebellopontine angle neoplasms. Annual incidence of VS is 1 case per 100.000 [1]. Surgery is the primary method for VS. These procedures are difficult due to adjacent brainstem structures, cranial nerves and vertebrobasilar vessels [1—4]. Cranial nerve dysfunction and cerebrospinal fluid (CSF) leakage are the most common postoperative complications. However, hemorrhagic and ischemic complications are the leading causes of postoperative mortality [1, 2, 5—8]. Physiological feasibility of surgical maneuvers during resection of VS relies on meticulous intraoperative hemodynamic and neurophysiological monitoring. Hemodynamic changes combined with persistent alterations in spontaneous and evoked bioelectrical activity (electroencephalography) and auditory brainstem evoked potentials diagnose functional deterioration of the brainstem and enable surgeon to respond to emerging critical situations. Intraoperative type 1 or type 2 centrogenic reactions warrant adjustment of neurosurgical strategy or, in some cases, termination of surgery [9—11]. Type 1 centrogenic reactions have a relatively distinct morphofunctional structure resembling a classic reflex arc. Most often, these are consequences of irritation of specific local centers or cranial nerve nuclei with a mixed motor-autonomic structure. These reactions usually include transient changes in blood pressure, heart rate and cardiac rhythm. Type 2 centrogenic reactions are based on hypoperfusion with irritation of midline brain structures and subsequent hypercatecholaminemia. These reactions are characterized by gradual elevation of blood pressure, heart rate and rhythm alterations, as well as suppression of cerebral

bioelectrical activity. The brain becomes tense, and bleedings become more common. In type 2 reactions, changes in cerebral bioelectrical activity may precede hemodynamic changes [9—11]. However, intraoperative monitoring is generally descriptive in nature and does not always early predicts brainstem damage resulting from neurosurgical procedure. Searching for additional markers (specifically, combinations of reflex and humoral bodily responses to surgical manipulations involving cranial nerves near the brainstem) seems interesting. This study is devoted to biochemical markers of functional impairment of the brainstem following VS resection.

The objective was to investigate the relationship between reflex and humoral responses to VS resection by correlating intraoperative changes in blood biochemical parameters with centrogenic reactions.

Material and methods

A retrospective analysis enrolled 89 patients who underwent resection of VS between 2011 and 2022.

Inclusion criteria:
— resection of VS;
— age > 18 years;
— preoperative ASA class II / III.

Exclusion criteria:
— infectious-inflammatory diseases within a month prior to surgery;
— intraoperative transfusion.

Patients were divided into two groups: no centrogenic reactions during resection (46 patients, group 1) and patients with type 1 reactions (43 patients, group 2).

All surgeries were elective. Patients underwent surgeries in lateral decubitus position. There was no aggravation of chronic comorbidities. We analyzed serum glucose, lactate, urea, uric acid, total protein, albumin, total bilirubin, cholesterol, creatinine, creatine kinase, myoglobin, lactate dehydrogenase, alanine aminotransferase, aspartate aminotransferase, amylase, iron, C-reactive protein, sodium and potassium. There was blood sampling at five stages: I — one day before surgery; II — on the day of surgery prior to induction of anesthesia; III — after induction of anesthesia; IV — intraoperatively after resection; V — on the first postoperative day. Blood samples were harvested into vacuum tubes containing a clot activator to obtain serum. Analysis was performed using an Integra 400 plus biochemical analyzer (Roche Diagnostics GmbH, Germany).

Statistical analysis implied the Kruskal—Wallis test of differences between the medians in independent groups and Dunn's test as a post-hoc analysis for significant Kruskal—Wallis test. There were multiple comparisons, and we adjusted p-values using the Benjamini—Hochberg method at α=0.05. Conover and Tukey—Kramer tests were applied to verify the results with appropriate plots for each parameter.

Results

Both groups were comparable in patient number, sex and age **(Table 1)**. The study also included 3 patients with intraoperative type 2 autonomic reactions. Although these reactions are extremely rare, they involve hypercatecholaminemia. This makes them interesting for data analysis within this small subgroup. A small number of patients with type 2 centrogenic reactions precluded their assignment to another group for statistical analysis.

All surgeries were performed under total intravenous anesthesia. Mean drug dosages did not differ between groups: fentanyl (induction / maintenance): 4.7 μg/kg / 1.45 μg/kg/h; clonidine (induction / maintenance): 1.69 μg/kg / 0.59 μg/kg/h or dexmedetomidine (induction / maintenance): 1.3 μg/kg / 0.39 μg/kg/h; propofol (induction / maintenance): 2.8 mg/kg / 3.65 mg/kg/h; rocuronium bromide (0.6 mg/kg) or pipecuronium bromide (0.1 mg/kg). Mean surgery time was 204 ± 45 min.

Longitudinal analysis of biochemical parameters revealed mild elevation of serum glucose during resection of tumor in all groups. By the first postoperative day, glucose returned to baseline in both groups. The cause of this elevation is catecholaminemia as a natural protective response to surgical manipulation. In patients with type 2 centrogenic reactions, serum glucose did not return to baseline by the first postoperative day. This may indicate a prolonged stress response to surgical trauma.

Normal serum lactate indicated no perioperative impairment of tissue perfusion. Urea, uric acid, total protein and albumin were normal and slightly decreased at the final stage of the study. This could be a consequence of some perioperative hemodilution **(Table 2)**.

Total bilirubin, cholesterol, creatinine, ALT, AST, LDH and amylase were normal in all groups at all perioperative stages **(Table 3)**.

Creatine kinase in all groups exceeded the upper limit on the first postoperative day. This may be due to prolonged recumbent position. This explanation is supported by concurrent rise in myoglobin. There are literature data on significant elevations in serum creatine kinase and myoglobin after neurosurgical or laparoscopic procedures even those not involving massive muscle damage. The most significant correlation was noted with surgery time [13—15].

Myoglobin significantly elevated in patients with type 2 reactions. Mean value increased from baseline 41.9 μg/L to 190.6 μg/L on the first postoperative

Table 1. **Patient data**

Variable	Group 1	Group 2
Number of patients, *n*	46	43
Men, %	39.13	46.51
Mean Karnofsky score	75.87±7.48	75.70±10.09
Mean age, years	54.41±11.21	50.44±14.56

Table 2. **Serum glucose, lactate, urea, uric acid, total protein and albumin**

Variable	Stage	Group		*p*-value for the Kruskal—Wallis test
		1	2	
Glucose, mmol/l	1	6.48	6.29	0.5678
	2	5.62	5.60	0.783
	3	6.28	6.55	0.3453
	4	7.20	7.03	0.7708
	5	6.52	6.65	0.2789
Lactate, mmol/l	1	2.30	2.46	0.6941
	2	2.08	2.33	0.3996
	3	1.30	1.36	0.4353
	4	1.40	1.22	0.4374
	5	1.59	1.73	0.1572
Urea, mmol/l	1	4.47	4.63	0.4434
	2	4.35	4.46	0.8257
	3	4.17	4.33	0.7553
	4	4.06	4.10	0.743
	5	3.67	3.96	0.519
Uric acid, µmol/l	1	282	259	0.3759
	2	281	273	0.8706
	3	257	247	0.7961
	4	222	188	0.06281
	5	210	169	0.02791
Total protein, g/l	1	71.86	73.94	0.02791
	2	68.28	71.55	0.04491
	3	58.21	61.17	0.01602
	4	57.29	57.18	0.01422
	5	64.04	63.81	0.1712
Albumin, g/l	1	44.99	44.9	0.8461
	2	42.73	42.90	0.8638
	3	37.04	37.31	0.8283
	4	35.91	34.78	0.2216
	5	39.08	37.54	0.1375

Table 3. **Total bilirubin. cholesterol. creatinine. LDH. AST. ALT and amylase**

Variable	Stage	Group		*p*-value for the Kruskal—Wallis test
		1	2	
Total bilirubin. µmol/l	1	8.14	8.19	0.9163
	2	10.13	10.00	0.9903
	3	8.50	8.08	0.8796
	4	8.74	8.33	0.7911
	5	10.06	10.50	0.9642
Cholesterol. mmol/l	1	5.10	5.44	0.2267
	2	4.95	5.3	0.2202
	3	4.26	4.54	0.2972
	4	4.15	4.27	0.6003
	5	4.63	4.87	0.4333
Creatinine. µmol/l	1	65.5	66.1	0.5885
	2	61.6	64.7	0.5412
	3	53.6	55.9	0.5006
	4	53.4	55.5	0.6894
	5	55.7	60.6	0.3531
LDH. U/l	1	168	176	0.2152
	2	168	208	0.113
	3	145	157	0.4162
	4	144	150	0.511
	5	169	172	0.3465
ALT. U/l	1	17.4	23.3	0.3454
	2	18	27	0.3082
	3	15.6	22	0.3874
	4	16.4	21.1	0.2655
	5	18.4	21.9	0.8357
AST. U/l	1	16.3	18.3	0.3179
	2	17	20.9	0.02962
	3	14.6	16.6	0.1303
	4	14.7	17.4	0.2526
	5	18	20.5	0.211
Amylase. U/l	1	57.9	58.8	0.4728
	2	52.3	53.9	0.5195
	3	46.6	45.1	0.5641
	4	44.1	42.1	0.8687
	5	55.2	55.8	0.9673

day. Notably, no increment in urea or creatinine ruled out renal injury. Sodium and potassium were normal. Serum iron decreased by the end of the study likely due to intraoperative blood loss. CRP was 4—5 times higher than upper limit in both groups. This finding was interpreted as a response to aseptic inflammation after surgical trauma **(Table 4)**.

The Kruskal—Wallis test revealed significant between-group differences (α=0.05) in uric acid, total protein and AST. At the fifth stage of the study, mean uric acid differed insignificantly between groups (Dunn's test, p=0.05834; Tukey—Kramer test, p=0.051). However, the Conover test (p=0.047) revealed a significant difference **(Fig. 1)**.

Significant negative correlation coefficients support higher serum uric acid at the fifth stage of the study in the 1st group. The Spearman correlation coefficient (ρ) was -0.2856 (p=0.0045), the Kendall correlation coefficient (τ) — -0.2324 (p=0.0100).

Total protein differed significantly between groups at the third stage of surgery (Conover test, p=0.042; Tukey—Kramer test, p=0.048). The third stage took place after induction of anesthesia and prior to multifactorial

Table 4. **Creatine kinase. iron. CRP. sodium. potassium and myoglobin in both groups**

Variable	Stage	Group		*p*-value for the Kruskal—Wallis test
		1	2	
Creatine kinase. U/l	1	65.8	61.9	0.3126
	2	80.5	79.2	0.9129
	3	81.3	74.5	0.5868
	4	93.5	87.8	0.4358
	5	234.9	189.6	0.464
Iron. µmol/l	1	17.14	17.48	0.9982
	2	19.11	20.10	0.6201
	3	16.37	16.78	0.8451
	4	16.60	14.8	0.4163
	5	6.58	7.48	0.2622
CRP. mg/l	1	1.01	0.77	0.8413
	2	1.01	0.85	0.8226
	3	0.80	0.67	0.6984
	4	0.77	0.62	0.766
	5	17.97	19.22	0.7772
Sodium. mmol/l	1	138	139	0.03228
	2	139	139	0.4551
	3	138	138	0.3978
	4	138	138	0.408
	5	136	136	0.5028
Potassium. mmol/l	1	4.1	4.1	0.8002
	2	3.8	3.8	0.628
	3	3.5	3.5	0.7302
	4	3.8	3.7	0.6417
	5	3.7	3.6	0.8172
Myoglobin. µg/l	1	27.1	26.5	0.1366
	2	46.5	59	0.1173
	3	53.7	55.9	0.6165
	4	60	64.8	0.7591
	5	64.3	89.6	0.06508

surgical impact on the brain. Therefore, slightly lower serum protein in the 1st group 1 at initial stages is not diagnostically significant.

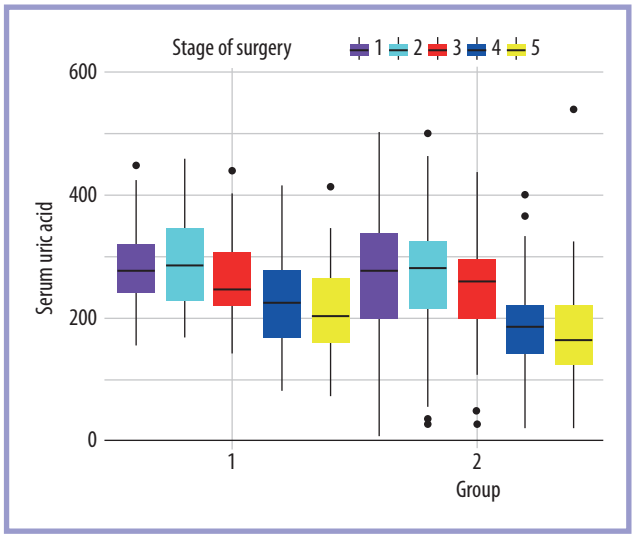
Discussion

After analysis of biochemical blood parameters and correlation between humoral changes, stages of neurosurgical procedure and centrogenic reactions, we can tentatively distinguish several types of biochemical changes:

— "rapid" reactions occur due to synthesis, redistribution or potentially deposition of specific substances such as iron, sodium, potassium, total protein, albumin, creatine phosphokinase, and myoglobin. Deposition or redistribution may result from relative hemodilution, blood loss or consumption following clotting;

— "slow" reactions minimally change during surgery without a great interest for monitoring purposes—cholesterol, urea, creatinine, bilirubin, amylase, alanine aminotransferase and aspartate aminotransferase;

— protective-compensatory reactions result from humoral response to multifactorial impact during neurosurgical procedure—uric acid, C-reactive protein, glucose, lactate and lactate dehydrogenase.



Uric acid in both groups at different stages of surgery.

Elevated serum lactate may be observed in patients with certain intracranial tumors (typically glial neoplasms), when hyperlactatemia results from anaerobic glycolysis. This process is known as the Warburg effect [12, 16]. In our study, normal lactate in both groups is a marker of satisfactory tissue perfusion under anesthesia. Patients with type 2 centrogenic reactions had elevation of serum lactate on the first postoperative day. This could result from tissue hypoperfusion since lactate is a well-known marker of ischemia. Another cause is autonomic imbalance associated with type 2 centrogenic reactions. However, non-ischemic release of lactate is more likely. Following brain injury, lactate release reflects activation of cerebral glycolysis to meet higher energy demands. This mechanism is crucial for maintaining neuronal activity under stress and can be recognized as adaptive response to enhanced energy requirements [17].

Uric acid is the end product of purine metabolism and possesses numerous antioxidant properties, including neutralization of free radicals and inhibition of lipid peroxidation [18—21]. Uric acid primarily inhibits release of reactive oxygen species through autoregulation of endogenous antioxidants and exerts protective effect on neuronal cells by maintaining intracellular calcium balance and stabilizing mitochondrial transmembrane potential. Under ischemic brain damage, xanthine oxidase is converted from

xanthine dehydrogenase for enhanced release of uric acid. As a renewable antioxidant, uric acid facilitates free radical neutralization, reduces oxidative stress and protects penumbra cells from ischemic injury [19, 22, 23].

VS resection does not result in tumor lysis or active release of cellular breakdown products (purines). However, between-group differences in uric acid (higher concentration in the 1st group at the fifth stage) may indirectly indicate more aggressive resection without centrogenic reactions. In this scenario, surgical actions are not constrained by intraoperative reflex hemodynamic or neurophysiological changes.

Between-group differences regarding humoral disturbances relate primarily to compensatory-adaptive biochemical changes. Serum glucose, lactate, LDH and CRP more significantly increased at the fifth stage in patients with type 2 centrogenic reactions. Autonomic imbalance appears to be the cause of type 2 reactions and a self-perpetuating mechanism. Thus, the risk of humoral disturbances is higher in type 2 centrogenic reactions, whereas type 1 reactions are short-lived and do not lead to persistent sympathicotonia. The last one together with hypercatecholaminemia drives more significant changes in biochemical parameters.

Conclusion

Dynamics of biochemical parameters at various neurosurgical stages for vestibular schwannomas makes it possible to assess the quality of protection against surgical stress.

Compliance with ethical standards

The study was conducted in accordance with the Declaration of Helsinki (2013). The study protocol was approved by the local ethics committee (protocol No. 1 dated April 11, 2011).

Author contribution:
Concept and design of the study — Lesteva N.A., Kondratyev A.N.
Collection and analysis of data — Lesteva N.A., Dryagina N.V.
Statistical analysis — Lesteva N.A.
Writing the text — Lesteva N.A., Dryagina N.V.
Editing — Kondratyev A.N.

No conflict of interests to declare.

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Comment

Resections of cerebellopontine angle tumors are among the most technically challenging neurosurgical procedures due to their proximity to brainstem structures. Thus, resection is relatively often accompanied by systemic autonomic reactions with severe hemodynamic and other changes. The last ones can predict a complicated postoperative course or even adverse outcomes.

Colleagues from the Polenov Russian Neurosurgical Research Institute long ago identified several types of such reactions with different prognostic significances and based on the relationship between reflex and humoral mechanisms. The essence of this study is clear. The authors tried to identify the most informative biochemical markers for predicting early postoperative period. To this end, the authors retrospectively analyzed changes of various biochemical parameters and determined the most informative ones. Notably, they succeeded. Uric acid, creatine kinase, lactate, myoglobin and C-reactive protein proved to be precisely such markers, and it is a significant achievement. I am unaware of any similar national and foreign large-scale studies of neurosurgical patients with this specific pathology. The only debatable point was the authors' attempt to attribute appropriate patterns solely to stress resulting from hypercatecholaminemia. I believe the process is more complex with hypercatecholaminemia being merely one factor. It would be interesting to assess key hemostatic parameters, as this system known to be subject to complex neurogenic and humoral regulation. However, that is a suggestion for future research. I find this study novel and interesting.

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