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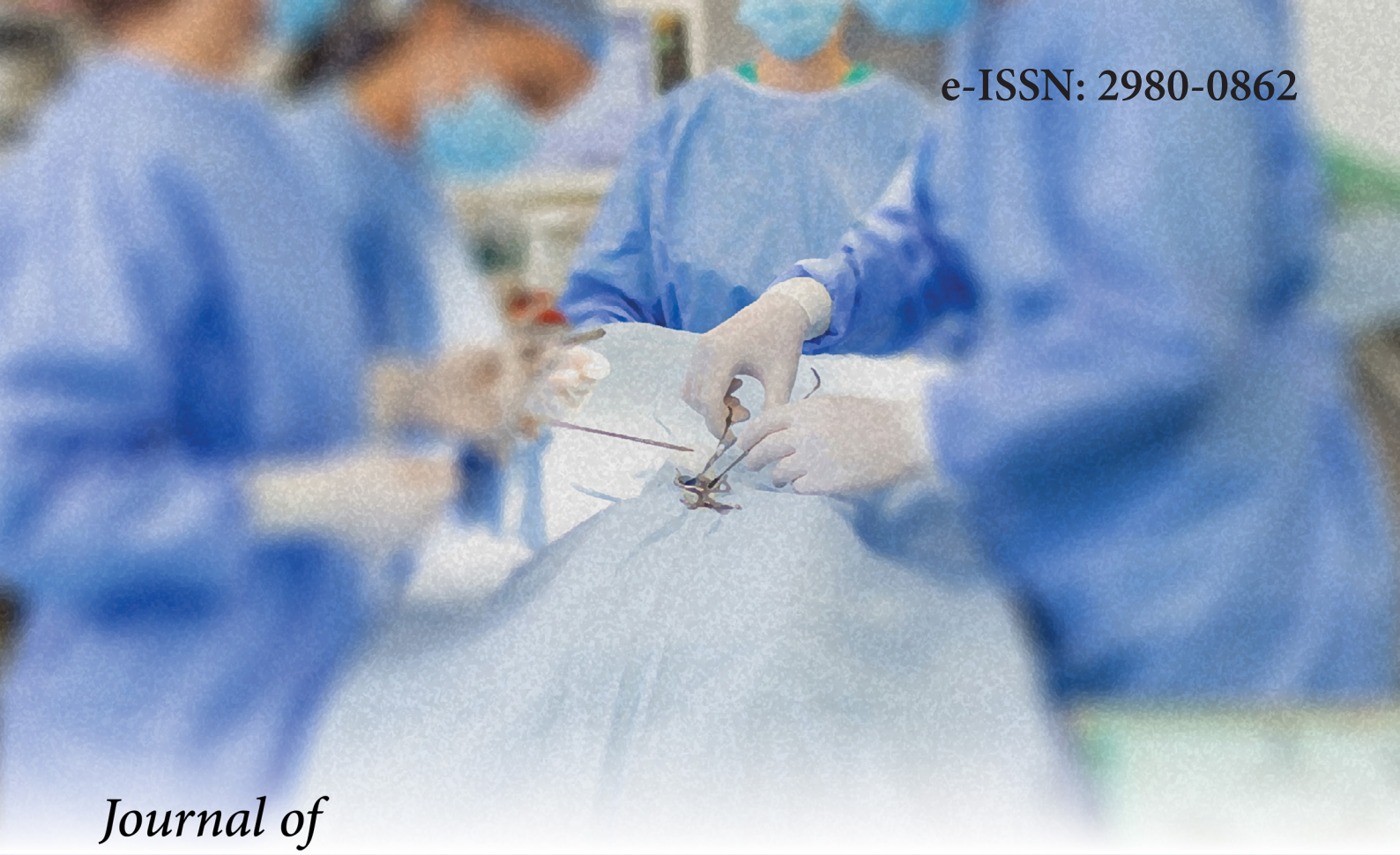

Journal of

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Volume: 1 Issue: 1 Year: 2023

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Journal of Comprehensive Surgery

EDITORIAL

Dear Colleagues,

Welcome to the inaugural issue of Journal of Comprehensive Surgery (JoCS), a new peer-reviewed academic journal of its kind. The publication of the journal has a far-reaching significance to both the professional purposes in all surgical fields and the researchers who need to conduct research for becoming an associate professor. The journal focuses on all aspects of surgical and related researchs, including original or experimental researchs, case reports, editorial letters and review articles. The editorial board of the journal consists of well-known experts at home. In cooperation with Medihealth Academy, we will provide efficient service to researchers that is full range, high quality, and high level. Our goal is to have JoCS included in nationally and internationally recognized scientific indexes.

As is well known, recently, one of the worst disasters of the century, a magnitude of 7.6 earthquake occurred in our country. It is a great challenge to publish a new journal during this difficult process, therefore I would like to express my deep gratitude to all the editorial board members, experts, editors and authors who make great efforts for the successful publication of the journal.

Finally, I sincerely hope that professionals both at home and abroad will warmly support the journal and work to make it a top-tier hub for scholarly communication. I wish JoCS a bright future!

Best Regards,

Mehmet Akif TÜRKOĞLU, MD
Editor in Chief

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CASE REPORT

Journal of Comprehensive Surgery

Original Article

DOI: 10.51271/JOCS-0001

Evaluation of the systemic inflammatory response in patients with a spinal synovial cyst or lumbar disc herniation

Mustafa Öğden[1], Mustafa İlker Karagedik[1], İbrahim Umud Bulut[1], Mehmet Zengin[2], Ulaş Yüksel[1], Bülent Bakar[1]

[1]Department of Neurosurgery, Faculty of Medicine, Kırıkkale University, Kırıkkale, Turkey
[2]Department of Pathology, Faculty of Medicine, Kırıkkale University, Kırıkkale, Turkey

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Corresponding Author: Mustafa Öğden, mustafaogden38@gmail.com

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ABSTRACT

Aims: This study aimed to determine whether a systemic inflammatory response occurs in patients with spinal synovial cysts (SSC) and patients with lumbar disc herniation.

Methods: Patients who underwent surgery for SSC (SSC group) or lumbar disc herniation (LDH group) between 2016-2022 were included in the study. To compare the results of these patients, patients who applied to the outpatient clinic due to headaches but did not find any abnormal findings in the radiological examinations (Control group) were also included. Age, gender, duration of stay in the hospital, comorbidity, and histopathological evaluation results were recorded for all patients. The lumbar T2 weighted MR sagittal and axial images obtained on admission to the hospital were examined. The venous blood samples taken from the patients on first admission to the hospital were analyzed biochemically.

Results: There was no statistical difference among the SCC, LDH, and Control groups regarding age, gender, and blood biochemistry parameters. Furthermore, no statistical difference was found between SSC patients and patients with lumbar disc herniation in age, gender, cyst or herniated disc localization levels, comorbidity, and blood biochemistry analysis results. Correlation analysis for the findings of all patients revealed that no parameters were correlated with the study groups. ROC-Curve test and Logistic Regression tests showed that no parameter could be a predictive marker in differentiating SSC from disc herniation.

Conclusion: Study results demonstrated that neither SSC nor disc herniation caused a systemic inflammatory or allergic reaction in the patients. In addition, it was determined that no biochemical parameter could distinguish disc herniation from SSC.

Keywords: Spinal synovial cysts, lumbar disc herniation, systemic inflammation

INTRODUCTION

Spinal synovial cysts (SSC) include both synovial cysts and ganglion cysts arising from the facet joint or ligamentum flavum. These lesions are most commonly seen in the lumbar spine and at the L4-5 level (88-99%).[1] The etiology of SSC is unknown, but it can be seen together with degenerative disc disease, osteoarthritis, spondylolisthesis, spinal stenosis, and scoliosis accompanied by increased spinal instability, and the role of trauma is discussed.[2] Today, the first choice for the diagnosis of SSC is MR imaging. Especially on T2-weighted MR axial images, cysts are often located on the medial side of an abnormal facet joint and show osteoarthritic changes and irregular articular surfaces, as well as a hypertrophic or focally thickened ligamentum flavum.[3] The clinical and radiological differential diagnosis of SSC includes Tarlov perineural cyst, extradural arachnoid cyst, dermoid cyst, neuroma with cystic changes, herniated disc, and free fragments of intervertebral disc.[4-6]

SSC may remain silent in some patients or may resolve spontaneously over time. To explain the spontaneous resolution of the cyst, some authors hypothesize that the cyst ruptures spontaneously, while others argue that progressive degenerative changes in the facet joint cause successive fixation of previously hypermobile facets, thereby reducing intra-articular pressure and shrinking the cyst.[7] Sometimes the SSC may undergo degenerative changes, lose contact with the facet joint, and shrink due to nutrient deficiency.[8] It has been reported in the literature that NSAID therapy acts on both the synovium, subchondral bone, and cartilage of the spinal facet joints and therefore may affect several different physiological pathways involved in the pathogenesis of lumbar spine synovial cysts (such as inhibition of proteoglycan synthesis,

induction of chondrocyte apoptosis and decreased production of cytokines and other mediators of inflammation). Therefore, it has been claimed that inhibition of inflammation in treating SSC may play an essential role in the pathogenesis of regression of lumbar spine synovial cysts.[9]

This study was conducted to determine whether a systemic inflammatory response occurs in patients with SSC. In addition, in this study, biochemical examination results of patients with SSC were compared with those of patients with lumbar disc herniation, and possible differences were tried to be revealed.

METHODS

Patients

The study was carried out with the permission of Kırıkkale University Medical Faculty Noninvasive Clinical Researches Ethics Committee (Date: 09.11.2022, Decision No: 2022/14-2022.11.06). A signed informed consent form was obtained from the patients.

Patients who underwent surgery for SSC or lumbar disc herniation between 2016-2022 were included in the study. To compare the results of these patients, patients who applied to the outpatient clinic due to headaches but did not find any abnormal findings in the radiological examinations were also included.

The working group was first divided into three groups as follows:

- Control group (n=14)
- CYST group (patients with a lumbar synovial cyst, n=13)
- LDH group (patients with single-level lumbar disc herniation, n=14)

Then, patients in the Control group who had an infection in the other facet joints, rheumatoid arthritis, other rheumatic diseases, bone metastases, or preoperative steroid injection were excluded from the study.

Materials

Age, gender, duration of stay in the hospital, and comorbidity (diabetes mellitus, hypertension, coronary artery disease, hypothyroidism, chronic obstructive pulmonary disease), were recorded for all patients. The lumbar T2 weighted MR sagittal and axial images obtained on admission to the hospital were examined in patients with SCC (**Figure 1**, **Figure 2**) and patients with lumbar disk herniation (**Figure 3**). Furthermore, Rosenstock classification type,[10] and NSURG classification[11] were evaluated in SCC patients. In addition, the histopathological analysis reports of all patients were recorded.

The venous blood samples taken from all individuals on first admission to the hospital were examined and the study results were obtained from these sample analyses. The blood hemoglobin level (reference range: 10-18 g/dL), leukocyte (reference range: 4400-11300 uL), neutrophil (reference range: 110-9600 uL), lymphocyte (reference range: 500-6000 uL), monocyte (reference range: 100-1400 uL), eosinophil (reference range: 0-1000 uL), basophil (reference range: 0-300 uL), and thrombocyte (reference range: 150,000-500,000 uL) count values were determined with an analysis device (Mindray BC-6800, Shenzhen, China). The erythrocyte sedimentation rate (ESR) value (reference interval <20 mm/hour) was measured by an automated system (ESR 40, Cystat Diagnostics). Furthermore, neutrophil-lymphocyte ratio (NLR), platelet-lymphocyte ratio results (PLR), and lymphocyte-monocyte ratio (LMR) were evaluated.

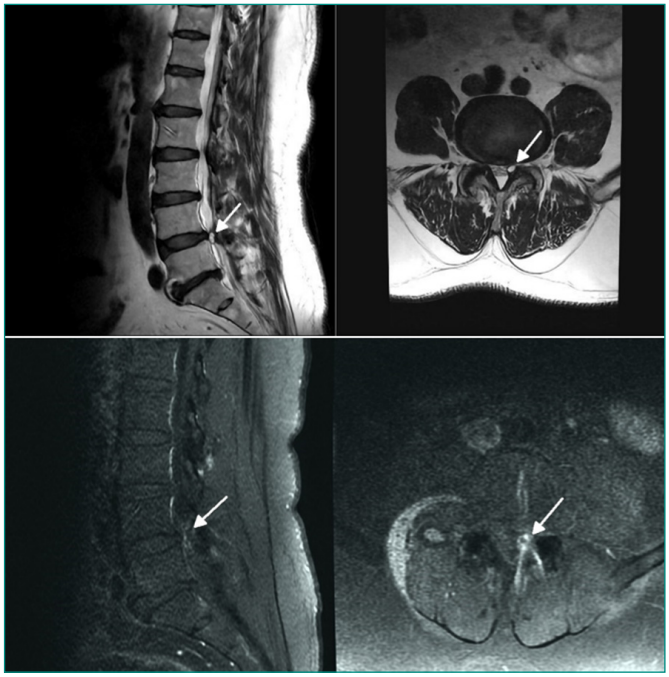

Figure 1. Microphotographs show T2-weighted MR axial images (1A) and T1-weighted MR axial images with gadolinium of a patient with a facet cyst.

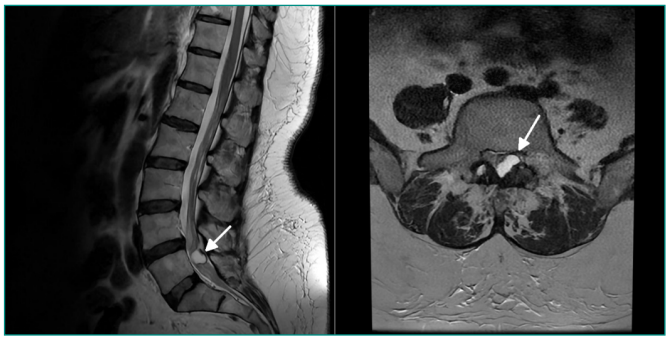

Figure 2. Microphotographs show T2-weighted MR axial images of a patient with a cyst of the ligamentum flavum.

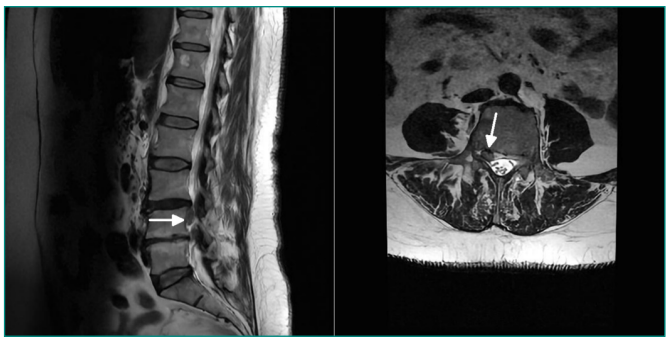

Figure 3. Microphotographs show T2-weighted MR axial images of a patient with right-weighted disc herniation in the L3-4 level.

Serum glucose (reference range: 74-109 mg/dL), blood urea nitrogen (BUN) (reference range: 17-43 mg/dL), creatinine (reference range: 0.84-1.24 mg/dL), alanine aminotransferase (ALT) (reference range: 5-41 U/L), aspartate aminotransferase (AST) (reference range: 5-40 U/L), C-reactive protein (CRP) (reference range: 0.15-5 mg/dL), total protein (reference range: 6.40-8.30 g/dL), albumin (reference range: 3.50-5.20 g/dL), sodium (reference range: 136-146 mmol/L) and potassium (reference range: 3.5-5.1 mmol/L) levels were obtained using original kits (Roche) on an automatic device (Roche Diagnostic COBAS c501).

Surgery

While the patient was in the prone position under general anesthesia, the spinal level at which the surgical intervention would be performed was determined by fluoroscopy. A midline vertical skin incision was then made. After unilateral subperiosteal stripping of the paravertebral muscles at the relevant level, partial hemilaminectomy and flavectomy were performed. Flavum cyst, facet cyst (**Figure 4**, **Figure 5**), or herniated disc fragment (**Figure 6**) exerting pressure on nerve tissues were resected and placed in neutral formalin solution for future pathological examination. The surgical layers were closed by the anatomy and the operation was terminated.

Statistical Analysis

The categorical variables were analyzed using Pearson's chi-square test ($p<0.05$).

Parametric data were analyzed with One Way-Analysis of Variance (ANOVA) to evaluate the differences between groups ($p<0.05$). Non-parametric data were analyzed using the Kruskal-Wallis test ($p<0.05$). The Mann-Whitney U test was applied in the binary comparisons of the groups ($p<0.05$).

In addition, Spearman's rho Correlation test was used to determine the presence of correlation between parameters belonging to patients ($p<0.05$).

The ROC-Curve test was used to determine which study parameters predict the hematoma expansion and the mortality risk, and the sensitivity and specificity rates of the parameters were determined by obtaining "cut-off" values. In addition, the Logistic Regression test was used to determine the "best predictive parameter" ($p<0.05$).

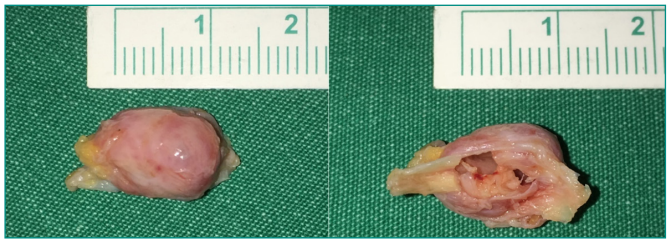

Figure 4. Macroscopic view of lumbar facet cyst.

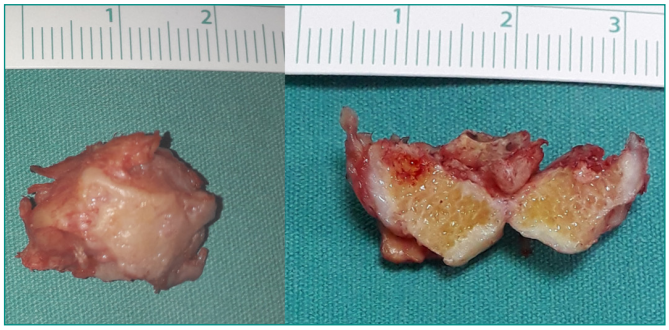

Figure 5. Macroscopic view of a lumbar facet cyst with dystrophic calcification.

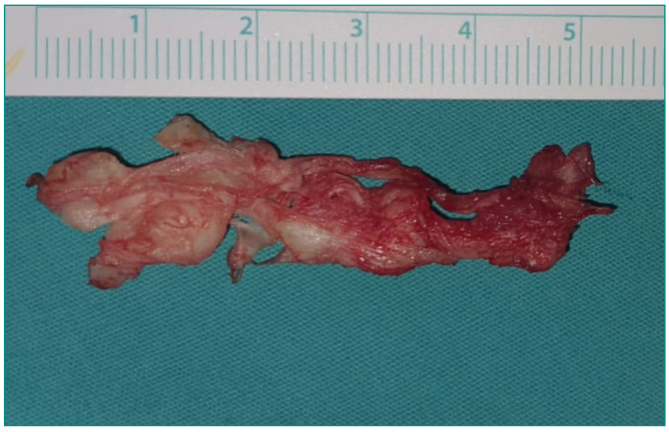

Figure 6. Macroscopic view of herniated lumbar intervertebral disc.

RESULTS

A total of 13 SSC patients (**Table 1**), 14 lumbar disc herniation patients, and 14 individuals without any spinal disease were included in the study. In the SCC group, two patients had synovial cysts with dystrophic calcification (**Figure 5**), two had synovial cysts with hyalinization, one had a ganglion cyst, and one had a ligamentum flavum cyst. The remaining seven patients had simple SSC (**Figure 4**, **Figure 7**).

Table 1. Descriptive table of patients with spinal synovial cyst

Patient	Age	Sex	Hospitalization	Segment	Diabetes mellitus	Hypertension	Coronary artery disease	Hypothyroidism	Chronic lung disease	Rosenstock classification	NSURG classification			Histopathological diagnosis	Cyst diameters			
											Canal obstruction	Spondylolisthesis	Result		Length	Height	Width	Volume
1	74	F	4	L4-L5	-	+	-	-	-	2	0	0	1	Spinal ganglion cyst	10.5	5.61	4.9	144.3
2	74	F	2	L4-L5	+	-	-	-	-	2	0	0	1	Synovial cyst + dystrophic calcification	10.7	6.77	7.96	288.3
3	69	K	2	L3-L4	+	+	+	+	-	2	0	0	1	Degenerative synovial cyst + hyalinization	5.8	3.63	7.03	74.0
4	67	M	2	L4-L5	-	-	-	-	-	2	0	0	1	Synovial cyst	9.03	3.83	5.58	96.5
5	67	M	4	L5-S1	+	+	+	-	-	1	0	0	1	Synovial cyst	6.78	5.69	6.2	119.6
6	67	M	2	L2-L3	-	+	+	-	-	1	0	0	1	Ligamentum flavum cyst	13.8	6.86	9.42	445.9
7	67	M	12	L4-L5	-	-	-	-	-	1	0	0	1	Synovial cyst	25.2	15.1	13.3	2530.5
8	63	F	1	L4-L5	-	+	-	-	-	2	1	0	2	Degenerative synovial cyst + hyalinization	16.3	12.3	13.5	1353.3
9	53	F	2	L4-L5	+	+	-	-	+	3	0	0	1	Synovial cyst + dystrophic calcification	9.02	6.76	11.6	353.7
10	51	F	3	L4-L5	-	+	-	-	-	2	1	0	2	Synovial cyst	12.8	10.5	11.5	772.8
11	68	M	3	L4-L5	-	-	-	-	+	3	1	0	2	Synovial cyst	16.4	14.1	10.5	1214.0
12	60	F	3	L3-L4	+	+	-	-	-	2	0	1	4	Hemorrhagic synovial cyst	9.76	5.47	8.51	227.2
13	52	F	3	L3-L4	-	+	-	-	-	2	0	0	1	Synovial cyst	9.87	10	5.94	293.1

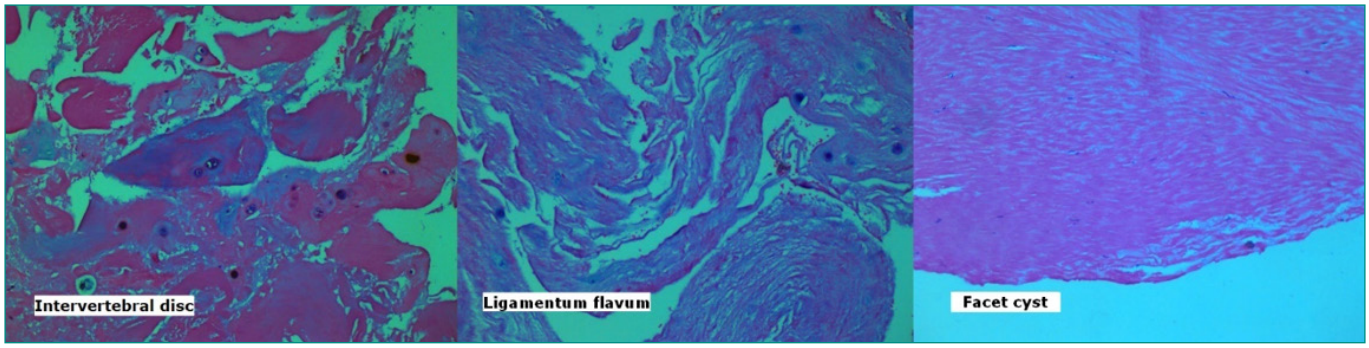

Figure 7. Histopathological examination images of a herniated intervertebral disc, a ligamentum flavum cyst, and a facet cyst.

It was found that the patients in the Control group were around 49 (34-69) years old, while the patients with SSC were around 67 (51-74) years old, whereas the patients in the LDH group were around 67 (36-89) years old. There was no statistical difference between the three groups in terms of age, gender, and blood biochemistry parameters (**Table 2**).

Table 2. Comparisons of the data of patients with spinal synovial cysts, patients with lumbar disc herniation, and the Control group patients.

Variable	Control	Cyst	LDH	F/ X2	p
	Mean ± SD Median (min-max)/ N (%)	Mean ± SD Median (min-max)/ N (%)	Mean ± SD Median (min-max)/ N (%)		
Age	49 (34-69)	67 (51-74)	67 (36-89)	-	-
Sex				0.659‡	0.719
Female	7 (29.2%)	8 (33.3%)	9 (37.5%)		
Male	7 (41.2%)	5 (29.4%)	5 (29.4%)		
Spinal level				1.639‡	0.651
L5-S1	-	1 (100.0%)	0 (0.0%)		
L4-5	-	8 (42.1%)	11 (57.9%)		
L3-4	-	3 (60.0%)	2 (40.0%)		
L2-3	-	1 (50.0%)	1 (50.0%)		
Hospitalization	-	3 (1-12)	2 (0-3)	-	-
Glucose	99.14±43.00	116.30±31.51	134.62±68.24	1.736†	0.190
BUN	29.14±7.35	36.63±7.64	37.18±12.56	3.072*	0.058
Creatinine	0.81±0.17	0.88±0.14	0.81±0.18	0.710*	0.498
ALT	24.50±10.65	24.23±16.47	26.61±14.70	0.118*	0.889
AST	25.50±12.24	19.66±7.15	22.12±8.06	1.301*	0.284
Protein	7.11±0.53	7.19±0.46	7.41±0.49	1.384*	0.263
Albumin	4.55±0.31	4.46±0.25	4.56±0.23	0.561*	0.575
CRP	3.06±3.98	3.30±3.19	4.77±5.68	0.607*	0.550
Sodium	141.14±2.14	140.82±2.66	140.04±2.41	0.776*	0.467
Potassium	4.75±0.41	4.79±0.29	4.70±0.39	0.229*	0.796
ESR	18.14±9.36	19.62±11.38	22.29±18.84	0.321*	0.728
Hemoglobin	14.51±1.36	13.79±2.05	14.21±1.35	0.668*	0.519
Leukocyte	6.40±1.71	5.18±1.92	5.90±3.17	0.901*	0.415
Platelet^10³	305±123334.26	279±76771.60	257±70651.91	0.939*	0.400
Neutrophile	2.15±0.85	2.13±0.79	2.69±1.49	1.167*	0.322
Basophil	32.14±18.88	30.00±15.28	36.43±19.85	0.441*	0.646
Eosinophil	162.14±95.69	214.62±219.11	183.57±113.38	0.412*	0.665
Monocyte	422.86±132.28	457.69±104.34	462.14±121.10	0.445*	0.644
NLR	2.15±0.85	2.13±0.79	2.69±1.49	1.167*	0.322
PLR	120.90±35.71	131.11±49.67	112.86±36.20	0.676*	0.514
LMR	6.40±1.71	5.18±1.92	5.90±3.17	0.901*	0.415

(*) F value, One Way-Analysis of Variance test; (†) X2 value, Kruskal Wallis test; (‡) X2 value, Pearson' s Chi-square test; p <0.05

At the end of the statistical analyzes performed by excluding the Control group, no statistical difference was found between SSC patients and patients with lumbar disc herniation in terms of age, gender, cyst or herniated disc localization levels, and comorbidity. In addition, no difference was found between these two patient groups in terms of blood biochemistry analysis results. However, a statistical difference was found between the two groups in terms of length of stay in the hospital (X2=-2.409, p=0.016) (**Table 3**).

Table 3. Comparisons of the data of patients with spinal synovial cysts and patients with lumbar disc herniation.

Variable	Cyst	LDH	F/ X2	p
	Mean ± SD Median (min-max)/ N (%)	Mean ± SD Median (min-max)/ N (%)		
Age	67 (51-74)	67 (36-89)	-0.271†	0.786
Sex				
Female	8 (47.1%)	9 (52.9%)	0.022‡	0.883
Male	5 (50.0%)	5 (50.0%)		
Level				
L5-S1	1 (100.0%)	0 (0.0%)	1.639‡	0.651
L4-5	8 (42.1%)	11 (57.9%)		
L3-4	3 (60.0%)	2 (40.0%)		
L2-3	1 (50.0%)	1 (50.0%)		
Hospitalization	3 (1-12)	2 (0-3)	-2.409†	0.016
Glucose	116.30±31.51	134.62±68.24	-0.884*	0.385
BUN	36.63±7.64	37.18±12.56	-0.137*	0.892
Creatinine	0.88±0.14	0.81±0.18	1.119*	0.274
ALT	24.23±16.47	26.61±14.70	-0.398*	0.694
AST	19.66±7.15	22.12±8.06	-0.838*	0.410
Protein	7.19±0.46	7.41±0.49	-1.220*	0.234
Albumin	4.46±0.25	4.56±0.23	-1.071*	0.294
CRP	3.30±3.19	4.77±5.68	-0.823*	0.418
Sodium	140.82±2.66	140.04±2.41	0.802*	0.430
Potassium	4.79±0.29	4.70±0.39	0.708*	0.485
ESR	19.62±11.38	22.29±18.84	-0.441*	0.663
Hemoglobin	13.79±2.05	14.21±1.35	-0.625*	0.538
Leukocyte	5.18±1.92	5.90±3.17	-0.708*	0.485
Platelet^10³	279±76771.60	257±70651.91	0.760*	0.454
Neutrophile	2.13±0.79	2.69±1.49	-1.212*	0.237
Basophil	30.00±15.28	36.43±19.85	-0.938*	0.357
Eosinophil	214.62±219.11	183.57±113.38	0.467*	0.644
Monocyte	457.69±104.34	462.14±121.10	-0.102*	0.920
NLR	2.13±0.79	2.69±1.49	-1.212*	0.237
PLR	131.11±49.67	112.86±36.20	1.097*	0.283
LMR	5.18±1.92	5.90±3.17	-0.708*	0.485

(*) t value, Independent Samples t test; (†) Z value, Mann Whitney U test; (‡) X2 value, Pearson' s Chi-square test; p <0.05

At the end of the correlation analysis for the findings of all patients, it was determined that no parameters were correlated with the study groups. As a result of the ROC-Curve test and Logistic Regression tests, it was concluded that no parameter could be a predictive marker in differentiating facet cysts from disc herniation.

DISCUSSION

SSC is symptomatic in many patients (90%) and may cause low back and leg pain.[12] It has been suggested that conservative treatment options such as physiotherapy, anti-inflammatory painkillers, and local steroid applications should be used primarily in treating symptomatic SSC, and surgical intervention should be applied when these treatments are insufficient. Percutaneous procedures

have been shown to have a statistically significantly lower symptom resolution rate than decompressive procedures.[13] There are three main treatment methods in the surgical treatment of SCC: percutaneous cyst aspiration, decompression surgery (hemilaminectomy, partial laminectomy, total laminectomy, etc.), and decompression surgery with fusion. While decompression surgery is sufficient in most patients, decompression with fusion is recommended for patients with spinal instability.[12] In our study, patients were treated with anti-inflammatory painkillers and physiotherapy, but local steroid therapy and/ or percutaneous cyst aspiration were not applied. Since none of the patients benefited from conservative treatments, hemilaminectomy, flavectomy, and cyst excision were performed by the microsurgical method, but fusion was not attempted. It was determined that the symptoms of all SSC patients improved after surgery.

It has been argued that ganglion cysts develop from mucinous degeneration of connective tissue in the mobile spine and have no direct connection with the facet joint. Besides, it has been claimed that synovial cysts are caused by laxity of the synovium of the facet joints, and therefore, they are highly correlated with segmental instability.[6] However, in our study, grade 1 spondylolisthesis was detected in only one patient. Since instability was not detected in this patient's preoperative standing lateral flexion and extension radiographs, decompression was performed, but fusion was not performed. Therefore, it was thought that SSC in these patients could not be related to instability.

On the other hand, it has been shown that the incidence of SSC is higher in patients with spondyloarthropathy, and it has been claimed that facet joint inflammation has an important role in the formation and development of SSC.[14] In addition, some studies have shown that angiogenic factors are released during the formation of synovial cysts, suggesting that these new vessels may increase the proliferation of synovial structures and that these factors may be associated with chronic inflammatory processes accompanying the progression of synovial cysts.[15] Many of these cysts have been shown to contain peculiar fine granular basophilic calcification, usually associated with a foreign body giant cell reaction and surrounding vascular and fibroblastic/ myofibroblastic proliferation.[16,17] For these reasons, it has been claimed that inhibition of inflammation in the treatment of SSC may play an important role in the pathogenesis of regression of lumbar spine synovial cysts.[9]

However, in a study, it was reported that after local steroid injections were applied to patients with SSC-related radicular pain, an excellent or good clinical result was obtained in 36% of patients in long-term follow-up, but additional steroid injections were made in one-third of these patients. It has been reported that in patients with moderate or poor results and patients with recurrent pain, repeated steroid injections were ineffective and eventually required surgery in these patients.[18] In another study, it was reported that in SSC patients, percutaneous cyst rupture or local intra-articular steroid injections were not superior to each other, both methods had an excellent short-term pain relief effect, but the effect decreased over time. Therefore, it has been said that percutaneous cyst rupture or local steroid injections can reduce the need for surgery in a significant number of patients and therefore may be recommended as first-line therapy, especially for those at high surgical risk.[19]

In our study, two SSC patients had dystrophic calcification and two had hyalinization. The remaining seven patients had simple SSC, one had a ganglion cyst, and one had a ligamentum flavum cyst. On the other hand, in the histopathological examinations, no chronic inflammation or foreign body giant cell reaction was detected in the specimens of the other patients, except for one patient. In addition, when the results of the biochemical analysis were examined, it was observed that there was no increase in inflammatory cytokine (CRP) or increase in inflammatory cells. With these findings, it was thought that these cysts did not induce systemic inflammatory reactions. In addition, it was observed that both groups' biochemical results were similar to those of the Control group individuals. In particular, CRP, leukocyte, neutrophil, lymphocyte, monocyte, eosinophil, and basophil values were observed to be similar in all three groups. In addition, ESR, NLR, PLR, and LMR values were found to be similar. These findings showed that neither SSC nor herniated discs caused a systemic inflammatory or allergic response. With these findings, it was considered that the cause of axial and or radicular pain in these patients was not due to systemic inflammatory reactions. Therefore, it was thought that the NSAIDs used in these patients were effective in suppressing or regressing the local inflammatory response and edema secondary to the lack of nutrition and hypoxia in these neural tissues during the pressure exerted by the SSC or disc fragment on the neural tissues, and thus they could reduce the pain and symptoms in the patients. However, this study could not support this hypothesis, since local inflammatory cytokines at the distance of SSC and herniated disc could not be measured in these patients.

On the other hand, it was determined that none of the biochemical parameters used in the study could distinguish disc herniation from SSC. This was thought to be due to the similar biochemical results in both groups.

CONCLUSION

As a result, it was determined that neither SSC nor disc herniation caused a systemic inflammatory or allergic reaction in the patients. In addition, it was determined that no biochemical parameter could distinguish disc herniation from SSC.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Kırıkkale University Medical Faculty Noninvasive Clinical Researches Ethics Committee (Date: 09.11.2022, Decision No: 2022/14-2022.11.06).

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Mustafa Öğden

I graduated from Karadeniz Technical University Medical Faculty in 1999. Between 2000 and 2006, I completed my neurosurgery specialization at Erciyes University. After working in various public and private hospitals for 9 years, I started Kırıkkale University as an Assistant Professor in 2015. I became an associate professor in 2020, I am still actively working at Kırıkkale University Faculty of Medicine.

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Review

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Crush syndrome: a review of current knowledge and treatment strategies

Alparslan Koç

Department of Anesthesiology and Reanimation, Mengücek Gazi Training and Research Hospital, Erzincan Binali Yıldırım University, Erzincan, Turkey

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Corresponding Author: Alparslan Koç, dralparslankoc@gmail.com

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ABSTRACT

Crush syndrome is a severe systemic manifestation resulting from the breakdown of muscle cells leading to the release of toxic substances into the bloodstream. This condition can occur when a part of the body experiences a significant amount of pressure for an extended period. Crush syndrome presents with severe metabolic disruptions such as acute kidney injury, electrolyte disturbances, and cardiovascular collapse. It is essential to understand the pathophysiology and clinical features of crush syndrome for effective management and prevention of potentially devastating outcomes. The act of crushing and rupturing muscular cells generates a mechanical force that triggers the discharge of myoglobin. Subsequently, myoglobin undergoes a conversion into metmyoglobin and acid hematin, which are subsequently released into the systemic circulation. The muscular tissue harbors a variety of electrolytes and enzymes, which may attain toxic levels upon entry into the circulation in excessive quantities. The release of sodium, calcium, and fluids due to regional ischemia leads to increased muscle volume and tension, depletion of creatine kinase (CK) and ATP, and muscle vasodilation, further exacerbating hypotension. Crush syndrome can also lead to cardiovascular instability and renal failure due to vasomotor and nephrotoxic factors. Elevations in serum CK levels exceeding 1000 IU/l, along with accompanying clinical features, are widely recognized as indicative of crush syndrome. Diagnostic investigations commonly involve assessing serum levels of aldolase, myoglobin, and myoglobin degradation products. Progressive increases in serum levels of lactic acid, aspartate aminotransferase (AST), alanine aminotransferase (ALT), and lactate dehydrogenase (LDH) are observed, while levels of serum urea and creatinine exhibit a steep rise, particularly following prolonged compression, and serve as valuable predictors of renal failure. The treatment of crush syndrome requires a multidisciplinary approach that addresses the metabolic, cardiovascular, and renal complications associated with this condition. The mainstay of treatment includes early release of the affected limb or compartment, fluid resuscitation, correction of electrolyte abnormalities, and alkalinization of urine. Additionally, renal replacement therapy and hyperbaric oxygen therapy may be beneficial in managing acute kidney injury and tissue hypoxia, respectively. Crush syndrome, albeit infrequent, represents a potentially fatal medical condition that demands a thorough comprehension of its underlying pathophysiology, clinical manifestations, and treatment modalities. Early recognition and appropriate management of this condition can significantly reduce morbidity and mortality associated with crush syndrome.

Keywords: Crush syndrome, rhabdomyolysis, myoglobin, renal failure

INTRODUCTION

Crush syndrome is a rare but potentially life-threatening condition that occurs when a significant amount of pressure is applied to a part of the body for an extended period, leading to muscle injury and the release of toxic metabolites into the bloodstream. Crush injury, resulting from compressive forces, can be a severe and potentially life-threatening condition. Although it is primarily a localized injury to the affected body part, it can have systemic effects as well. Crush syndrome, also known as traumatic rhabdomyolysis, is a rare but severe systemic manifestation that occurs as a result of the breakdown of muscle cells, leading to the release of their contents into the bloodstream. This release of toxic substances, including myoglobin, can lead to severe complications such as acute kidney injury, electrolyte disturbances, and cardiovascular collapse. Understanding the pathophysiology and clinical features of crush syndrome is crucial for effective management and prevention of potentially devastating outcomes.[1,2] Despite being acknowledged by German physicians during the First World War and following the Messina earthquake of 1909, it was not until 1941 that the first documented account of crush syndrome in English language literature was reported by Bywaters and Beall.[3] Crush injuries are frequently encountered during natural disasters, such as earthquakes. Nevertheless, emergency physicians more commonly encounter crush syndrome in individuals who have been involved in motor vehicle collisions, particularly

those with prolonged extrication times, and those who are victims of physical assaults.[4] The terminologies related to crush injury encompass a spectrum of conditions. "Crush injury; refers to the direct physical damage caused to the muscles as a result of external pressure. Crush syndrome; also known as rhabdomyolysis, is a severe manifestation of muscle injury caused by disruption of cellular integrity and release of muscle contents into the bloodstream. Compression syndrome; on the other hand, occurs when there is an indirect injury to the muscle caused by a slow compression of a muscle group leading to ischaemic damage and subsequent release of crush substances into the circulation. Finally, compartment syndrome is a localized and rapid rise of tension within a muscle compartment leading to metabolic disturbances similar to those seen in rhabdomyolysis."

PATHOPHYSIOLOGY

Crush injuries may lead to fatal consequences, with a high percentage of patients succumbing to head injuries or asphyxiation. Only a fraction of patients that make it to the hospital experience a successful recovery, with the remaining percentage developing crush syndrome, which presents with severe and extensive metabolic disruptions. It is therefore critical for medical professionals to prioritize and implement effective treatment approaches to address the complex and life-threatening complications associated with crush syndrome.[5,6] The mechanical trauma resulting from crush and rupture of muscular cells provokes the extrusion of myoglobin, which undergoes subsequent conversion into metmyoglobin and subsequently acid hematin before entering the systemic circulation. Muscles contain various electrolytes, such as potassium, magnesium, phosphate, acids, and enzymes including CK and lactate dehydrogenase (LDH). Although these are necessary for cell function, they become toxic in the circulation when released in excessive amounts. Regional ischemia occurs in crush syndrome due to the obstruction of micro and macrocirculation in the muscles, prompting the release of sodium, calcium, and fluids, resulting in increased muscle volume and tension. This process leads to the depletion of CK and ATP. Moreover, the activation of the nitric oxide system leads to muscle vasodilation and further exacerbates hypotension.[7] Crush syndrome can lead to cardiovascular instability which may have several underlying causes. The translocation of fluids from the extracellular compartment towards the injured muscular cells may induce depletion of intravascular volume, culminating in the development of hypovolemic shock. Additionally, cardiovascular compromise can arise from blood loss associated with the injury, as well as myocardial toxicity resulting from electrolyte disturbance. Moreover, experimental models have demonstrated that substances released by the muscular cells are capable of inducing direct depression of the cardiovascular system.[8] The most severe complication of crush syndrome is renal failure.[9] The pathogenesis of renal failure in crush syndrome is intricate and entails the interplay of vasomotor and nephrotoxic factors.[10]

INVESTIGATIONS

Studies have demonstrated a strong association between the elevation of CK and the development of renal failure as well as mortality in patients with crush syndrome.[11] An elevation in serum CK levels exceeding 1000 IU/L, in conjunction with relevant clinical features, is frequently considered diagnostic of crush syndrome. The normal range for CK levels is 25-175 U/L, which typically starts to rise within 2 to 12 hours following a crush injury, peaks between 1 to 3 days, and then gradually declines after 3 to 5 days. Additional investigations that may assist in diagnosing crush syndrome include measurement of serum aldolase levels. Serum myoglobin and its degradation products are highly sensitive laboratory tests for detecting the release of muscle proteins into the systemic circulation. Additional biomarkers that exhibit a progressive elevation comprise serum AST, ALT, lactic acid, and LDH. Moderate increments in serum uric acid may also be detectable. Serum urea and creatinine concentrations show a sharp surge, particularly following an extended period of crush, and serve as valuable predictors of renal failure. Additionally, an early increase in serum potassium levels can serve as a predictor for dialysis.[12] In crush syndrome, there may be a concomitant occurrence of hypocalcemia and stress-related hyperglycemia. Presence of myoglobin products in urine can be detected by urine RE. In addition, blood gas analysis, complete blood cell count, and electrocardiography are indispensable diagnostic modalities. Intracompartmental pressure monitoring is crucial, as readings surpassing 30 mm Hg warrant the performance of fasciotomy. Doppler ultrasonography is conducted to detect limb ischemia, and the body weight of the patient is documented.[13]

TREATMENT STRATEGIES

The immediate treatment of individuals affected by crush injuries is crucial to minimize morbidity and mortality. The possibility of concurrent injuries such as fractures, spinal or solid organ damage should be considered and managed accordingly after the assessment of airway, breathing, and circulation. Prompt oxygen administration and control of any visible bleeding should be prioritized. The provision of fluid therapy, either parenteral or enteral, contingent on resource availability and the extent of the casualties, is essential to sustain intravascular volume. Nevertheless, parenteral therapy is usually the preferred mode of administration. Swift evacuation to a facility with definitive care is imperative. Upon admission, patients must undergo hourly urine output measurements, electrolyte surveillance, arterial blood gas analysis, and monitoring of muscle enzymes. Central venous pressure and invasive arterial monitoring should be taken into consideration. Prevention of acute kidney injury is paramount, as its occurrence is linked to reduced survival rates.[9,14] Resuscitation for crush syndrome ideally starts at the site of injury, as casualties are often in shock and may lose significant amounts of extracellular fluid into the injured extremity. In combat scenarios, obtaining a comprehensive patient history may not always be feasible, and the syndrome can progress surreptitiously in patients who seem initially stable. Hence, personnel must receive adequate training to identify the condition promptly and manage it proactively with fluid therapy. The recognition and management of crush syndrome mandate close collaboration among anesthesiologists, trauma surgeons, biochemists, physicians, and radiologists.

The choice of fluid administered in prehospital settings is determined by local resources and protocols for fluid replacement., prehospital vehicles typically carry saline as the preferred fluid, which was recommended for use in

crush injuries during a recent consensus meeting.[15] The administration of solutions containing potassium carries a theoretical disadvantage, as it may exacerbate hyperkalemia. The amount of fluids administered varies greatly, with reports of infusion of more than 25 liters of saline in a single day. While there is agreement on the use of saline as the preferred fluid, in order to address metabolic acidosis, supplementation with bicarbonate, lactate or even oral citrate is necessary. Alkalinization treatment, also known as bicarbonate therapy, has been proposed as a potential treatment for crush syndrome. Gunal et al recommend the administration of 50 mmol of bicarbonate per liter of isotonic saline.[16] It is widely recognized that substantial quantities of fluid may accumulate within the traumatized muscle, with as much as 12 liters in the initial 48 hours for an adult weighing 75 kilograms. To reduce the salt burden, Better advises an initial infusion rate of 1 to 1.5 liters of saline per hour, followed by 5% glucose.[17] The patient's central venous pressure (CVP), blood pressure (BP), pulmonary status, and urinary output are closely monitored. An insulin glucose drip may be administered to mitigate a sharp increase in serum potassium concentration. In cases of crush syndrome, patients often require multiple blood product transfusions, and it is important to properly address the logistical challenges associated with collection, storage, and transportation.[18]

Diuresis

It is important to emphasize that preserving optimal kidney function is a critical component of managing crush injuries. In situations where crush syndrome has been confirmed, urinary output must be upheld at a minimum rate of 300 ml/hr, corresponding to a minimum fluid intake of 12 liters daily, given that fluid accumulation within the injured muscles may amount to as much as 4 liters.[18] There is a divergence of opinions regarding the necessity of inducing diuresis versus maintaining sufficient hydration to prevent renal failure. While some experts advocate that administering mannitol is unnecessary and that adequate fluid replacement and alkalinization suffice, others contend that mannitol may offer additional benefits beyond its diuretic properties.[16] Holt et al. suggest that while mannitol is indicated for use in cases of compartment syndrome, it is not superior to intravenous fluids alone.[19]

Dialysis

The diagnosis of acute kidney injury (AKI) was established using the Kidney Disease Improving Global Outcomes (KDIGO) composite staging criteria. This involves an increase in serum creatinine of 0.3 mg/dl or more within 48 hours or a reduction in urine output below 0.5 ml/kg/h for six hours, in the absence of pre-existing renal disease. Significant predictive factors for requiring dialysis comprise anuria, fluid overload, levels of serum creatinine, blood urea nitrogen (BUN), and bicarbonate levels.[16] Additionally, elevated potassium levels above 7 meq/1 are an important independent predictor for dialysis. In some cases, dialysis may need to be performed two to three times daily for up to 15 days. Patients at high risk for hyperkalemia may require prophylactic dialysis.[20]

Renal Replacement Therapy

Renal replacement therapy (RRT) is a crucial component of the management of AKI in crush syndrome patients. Crush syndrome-induced AKI results from the release of myoglobin into the circulation, leading to acute tubular necrosis and renal failure. RRT aims to remove the toxic substances, maintain fluid balance, and correct electrolyte imbalances in patients with AKI.[21] A study conducted by Ishii et al. in 2020 found that early initiation of continuous RRT in patients with crush syndrome-associated AKI significantly improved renal recovery and overall survival.[22] A study published in the Journal of Trauma and Acute Care Surgery found that early initiation of renal replacement therapy (RRT) in patients with crush syndrome and AKI can improve overall outcomes, reduce complications, and decrease mortality rates.[23]

Moreover, some researchers have suggested that extracorporeal membrane oxygenation (ECMO) may have a role in the management of crush syndrome-induced AKI. A recent study reported successful ECMO support in a patient with severe crush syndrome complicated by AKI and respiratory failure.[24] However, further studies are required to determine the optimal timing and duration of ECMO in this setting.

In conclusion, RRT plays a crucial role in the management of AKI in crush syndrome patients. Early initiation of continuous RRT may improve renal recovery and overall survival. High-volume hemofiltration has also been shown to be effective in removing circulating myoglobin. While ECMO may have a role in managing AKI in these patients, further research is needed to determine its optimal use.

Hyperbaric Oxygen

Exposure to high pressures has been shown to increase the physically dissolved levels of oxygen in the plasma, resulting in improved tissue viability, mild vasoconstriction, and reduced fluid outflow from the vascular compartments, thereby decreasing tissue edema. This process directly benefits wound healing by promoting fibroblast proliferation. Moreover, exposure to high pressure has the potential to decrease anaerobic bacterial growth in necrotic muscle.[25] Typically, the standard treatment involves exposure to 2.5 atmospheres of pressure for approximately one and a half hours, administered twice a day for a duration of one week.[26]

CONCLUSION

In conclusion, crush syndrome is a serious medical condition that can result in significant morbidity and mortality if not managed appropriately. The initial assessment of patients with suspected crush injury should focus on timely extrication and provision of effective fluid resuscitation. The maintenance of effective kidney function is critical in the management of this condition, with measures such as prophylactic dialysis and the administration of mannitol potentially playing a key role. The importance of close monitoring and management of electrolyte imbalances, particularly hyperkalemia, cannot be overstated. The use of hyperbaric oxygen therapy and other advanced wound care techniques can aid in tissue healing and may prevent the need for amputations. Overall, early recognition and aggressive management of crush syndrome are essential to improve outcomes and minimize complications in affected patients.

ETHICAL DECLARATIONS

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Alparslan Koç

He was born in Erzincan. He graduated from Ankara Medical Faculty in 2003. He received his specialization from Haseki Training and Research Hospital 1st Anesthesiology and Reanimation Clinic. He did his military service as a reserve officer at Tatvan Military Hospital, and then worked at Kütahya Evliya Çelebi Training and Research Hospital for 7 years. He is still working at Erzincan Binali Yıldırım University Mengücek Gazi Training and Research Hospital. He is married and has three children.

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Case Report

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Rectal perforation due to malignancy as a rare cause of acute abdomen in a third trimester pregnant patient: a case report

Abidin Göktaş, Tezcan Akın, Şükrü Melih Bayazıtlı, Erdinç Çetinkaya, Özgür Akgül, Hüseyin Berkem, Ahmet Keşşaf Aşlar, Bülent Cavit Yüksel, Sadettin Er

Department of General Surgery, Ankara Bilkent City Hospital, Ankara, Turkey

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Corresponding Author: Sadettin Er, ersadettin74@gmail.com

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ABSTRACT

Anatomical and physiological changes during pregnancy may complicate diagnosis and treatment. While the cause of non-obstetric acute abdomen is encountered in one in 500 pregnancies, the most common one may be acute appendicitis. The incidence of colorectal cancer (CRC) during pregnancy is 0.002%. Common symptoms, such as abdominal pain, nausea, vomiting, and changes in bowel movements, are often observed in a regular pregnancy. Thus, most colorectal cases are overlooked and diagnosed in advanced stages associated with a poor prognosis. This case report presents a 31-year-old female patient at 37 weeks of gestation who applied with an acute abdomen, underwent emergency explorative laparotomy, had a 2 cm perforation detected in the rectum, and was referred to neoadjuvant chemo-radiotherapy due to perforation due to malignancy detected in the examinations performed in the postoperative follow-up. The relevant literature notes that colorectal cancer is rarely seen among expectant mothers. It should be suspected in the diagnosis among patients with gastrointestinal complaints for whom conservative treatment has failed. It should also be noted that bowel perforation, a rare complication of colorectal cancer, may also be present in emergency department applications with an acute abdomen.

Keywords: Pregnancy, colorectal cancer, perforation

INTRODUCTION

Acute abdomen continues to pose a challenge regarding diagnosis and treatment in pregnancy. Thus, physicians had better be aware of the non-obstetric causes of abdominal pain among pregnant patients and the atypical presentations of surgical emergencies. Colorectal cancer (CRC) is a rare but potentially fatal condition complicating pregnancy. The incidence peaks among patients aged 50 years, but contemporary research reports that it is becoming more frequent among those younger than 40 years.[1] It can also occur during pregnancy, as its incidence increases among younger women. Perforations in CRC are rare but severe complications with a mortality rate of 30-40%.[2] The present study aims to present both a rare case of rectal cancer and a rare case of rectal perforation due to malignancy as a cause of acute abdomen in third-trimester pregnancy.

CASE

A 31-year-old female patient at 37 weeks of gestation applied to the emergency department of the obstetrics clinic with complaints of sudden onset of abdominal pain and vaginal bleeding. General surgery was also consulted on concomitant severe abdominal pain.

In her physical examination, her blood pressure was 110/70 mmHg, her pulse was 90/min, and the Glasgow Coma Scale score was 15. She was conscious, oriented, and cooperative. Her abdominal examination yielded tenderness, defense, and rebound in the bilateral lower quadrants and normal stool contamination on the rectal touch.

The laboratory findings were as follows: While blood cell (WBC) = 22.2x10 (20.8 Neu), Hemoglobin (HBG) = 9.1 mg/dL, C-reactive protein (CRP) = 60 mg/l, Creatinine (CRE) = 0.6, Urea = 20, Urinalysis Test (UAT) = Leukocyte esterase 1+ (6 leukocytes), Nitrite = negative, international normalized ratio (INR) = 1.2.

The abdominal USG performed under emergency conditions yielded nothing in the uterine cavity except FHB+ live fetus. The emergency abdominal MRI showed minimal smearing-style free liquid in the right and left paracolic areas in the liver and in the pelvis, irregular wall thickening reaching 14 mm in the widest part of the rectosigmoid section, and diffusion restriction in this area.

Accordingly, an emergency operation was planned for the patient with an acute abdomen and whose pregnancy reached terms. Purulent content mixed with stool in all quadrants, particularly in Douglas, was discovered in the patient taken

to an emergency cesarean section in the obstetrics clinic. After washing, a rectal perforation of approximately 2 cm was observed at the level of the peritoneal reflection, and Hartmann's procedure was performed on the patient due to the intense intrabdominal contamination.

Etiological investigations continued in the postoperative follow-up. In abdominal tomography, there were asymmetrical wall thickening up to 28 mm on the left lateral wall in the thickest part of the rectum along the long segment and multiple lymph nodes around the rectum, the largest of which reaches 14×9 mm in size. Pelvis MRI resulted in tumor wall thickening surrounding the rectum wall in the approximately 12 cm segment in the rectosigmoid region. It was measured at 32 mm where it was most emergent (**Figure 1**). Yet, the relationship of tumor tissue with mesolectal fascia and fatty tissue could not be evaluated with this examination. No pathologically sized lymph node was detected. In the colonoscopy, a broad-based ulcerovegetan mass protruding into the lumen and surrounding 50% of the lumen at 10 cm from the anal verge was observed, and biopsies were taken. The patient, whose drain was terminated during the follow-ups and who had normal findings in further physical examinations, tolerated the regimen, and had discharge from the ostomy, was referred to the oncology clinic for neoadjuvant chemo-radiotherapy.

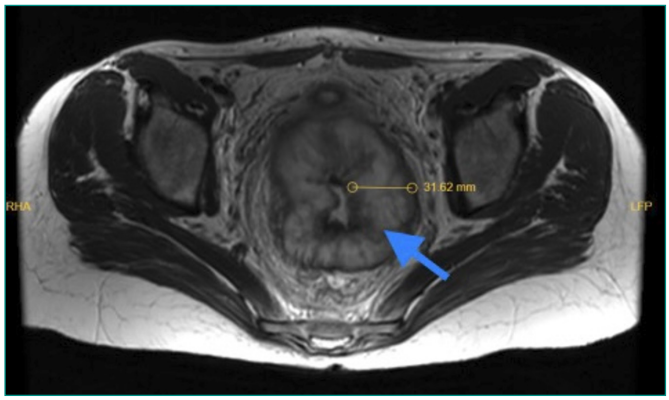

Figure 1. Irregular wall thickening in the rectum in pelvic MRI (blue arrow)

DISCUSSION

Physiological and anatomical changes during pregnancy may lead to diagnostic difficulties among patients presenting with acute abdomen due to the unpredictability of obstetric complications and changes in physical examination findings, laboratory findings, and imaging methods.[3] The incidence of an acute abdomen during pregnancy is often reported to be one in 500.[4] The causes of abdominal pain can be divided into obstetric and non-obstetric causes.[4] The most common non-obstetric cause is acute appendicitis, corresponding to 25-30% of expectant mothers undergoing surgery.[4] Other causes include intestinal obstructions, acute cholecystitis, inflammatory bowel diseases, peptic ulcer, and acute pancreatitis.[4] Of these, intestinal perforation is a life-threatening condition despite being extremely rare.[5] It can lead to adverse maternal and fetal outcomes such as miscarriage, premature birth, and intrauterine death.[5]

USG is a safe, first-choice, and easily accessible imagining method among those to be utilized during pregnancy. However, intra-abdominal organs may be displaced with the enlargement of the uterus, making USG imaging difficult and reducing sensitivity.[6] However, high accuracy rates are reported for MRI when compared with medical follow-ups and post-surgical diagnoses.[7] In the present case, an acute abdomen was considered as a result of physical examination and laboratory findings, and MRI was planned as diagnostic results could not be achieved with USG. As a result of the examinations, the patient was taken into operation since being in the third trimester, and the fetus had reached the required maturity. The investigation of the cause of the postoperative perforation pointed out rectal cancer.

The literature shows the most common causes of colon perforations as colon diverticulum with a rate of 58-63% and CRC with a rate of 14-21%.[8] The incidence of cancer in pregnancy varies between 0.02% and 0.1%, and CRC is one of the eight most common malignant neoplasms in pregnancy.[9] Despite being rare among the causes of acute abdomen, perforated CRCs are characterized by high mortality rates.[2] CRC is not considered in the initial diagnosis among young patients without family history and predisposing factors. Yet, the present case may be regarded as a robust example that CRC can be encountered in young patients without known risk factors.

Now, about one out of every ten new CRC diagnoses corresponds to individuals aged 50 years and younger.[10] The relevant research also documents that the incidence of CRC at young ages may increase in the coming years.

CONCLUSION

Given the relevant research and its increasing incidence, it should be noted that CRC may be encountered in young cases and that CRC perforation had better be included among the preliminary diagnoses in gastrointestinal system perforations in rare cases. Surgeons and obstetricians may need to act in harmony and intervene early with a multidisciplinary approach to pregnant women demonstrating the picture of an acute abdomen due to the physiological and anatomical changes during pregnancy and difficulties in the diagnosis and treatment processes.

ETHICAL DECLARATIONS

Informed Consent: All patients signed the free and informed consent form.

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Abidin Göktaş
I was born in Konya in 1993. I studied primary and high school in Konya. I graduated from Selcuk University Faculty of Medicine in 2017. I worked as a general practitioner at Cumra State Hospital between 2017-2019. I have been working as a research assistant at Ankara Bilkent City Hospital General Surgery Clinic since 2019.

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Case Report

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Anesthesia management in a pediatric patient with craniopharyngioma

Nurhan Eren, Alparslan Koç

Department of Anesthesiology and Reanimation, Mengücek Gazi Training and Research Hospital, Erzincan Binali Yıldırım University, Erzincan, Turkey

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Corresponding Author: Alparslan Koç, dralparslankoc@gmail.com

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ABSTRACT

Craniopharyngiomas are the most common sellar or suprasellar tumors in childhood. The most critical issue in neuroanesthesia is ensuring adequate cerebral perfusion pressure and appropriate surgical conditions without altering autoregulation of cerebral circulation. The aim of this article is to provide an overview of anesthetic management for successful craniopharyngioma surgery in pediatric patients. An 11-year-old girl who presented to the pediatric outpatient clinic with complaints of headache and visual disturbances was diagnosed with craniopharyngioma, and transsphenoidal pituitary surgery was planned. Noninvasive monitoring (pulse oximetry, ECG, blood pressure, and temperature) was performed, and a precordial USG was prepared to detect air embolism. Mild to moderate hypothermia of 34-36°C was achieved. The patient had no intraoperative complications, was extubated postoperatively, and transferred to the intensive care unit for close monitoring. The goals of anesthesia for transsphenoidal pituitary surgery include optimizing cerebral oxygenation, maintaining hemodynamic stability, managing intraoperative complications, and facilitating rapid and smooth recovery.

Keywords: Craniopharyngioma, pituitary surgery, anesthetic management

This article was presented orally at the 6th Palandoken anesthesia symposium.

INTRODUCTION

Craniopharyngiomas are the most common sellar or suprasellar tumors in childhood. Because they grow slowly, they can become a huge mass if they are asymptomatic. Treatment should be personalized with a multidisciplinary approach. The goal of surgery should be total resection without mortality or acceptable morbidity.[1] The most critical issue in neuroanesthesia is ensuring adequate cerebral perfusion pressure and appropriate surgical conditions without altering autoregulation of the cerebral circulation. Successful surgical management of patients with pituitary tumors requires a multidisciplinary approach and depends critically on the quality of perioperative care. A thorough preoperative examination and screening are essential in all patients with pituitary tumors. Knowledge of potential complications, their management, and prevention strategies are essential for successful perioperative patient care. Ensuring the safety of premedication and preoperative sedation is of utmost importance in neurosurgical cases, particularly in pediatric patients who may experience separation anxiety from parents and fear of the operating room, which can complicate hemodynamic management. Hence, premedication should be administered with great care under the supervision of a physician, and the patient should be transported to the operating room expeditiously.[2] Effective surgical management of pituitary tumor patients is dependent on a multidisciplinary approach and the provision of high-quality perioperative care. This case report illuminates the anesthesia management of a successful craniopharyngioma case in the context of current literature.

CASE

An 11-year-old girl who presented to the pediatric outpatient clinic with complaints of headache and visual disturbances was diagnosed with craniopharyngioma and referred to us for preoperative anesthesia examination. Physical examination of the patient, which was performed preoperatively, revealed growth retardation and bitemporal hemianopsia. The patient's blood count, biochemistry and coagulation tests were within normal limits, and the level of thyroid hormones was low. The patient, with a GCS of 15, had no defect on cardiovascular examination. No respiratory pathology was detected in the patient. Thyroid hormone replacement and transsphenoidal pituitary surgery were planned for the patient, who had no surgical history and normal cortisol levels. Midazolam (0.04 mg/kg IM) was administered in the premedication room. The patient was transferred to the operating table. Noninvasive monitoring (pulse oximetry, ECG, blood

pressure, and temperature monitoring) was performed. An urinary catheter was placed. A precordial USG was prepared to detect air embolism. The induction of mild to moderate hypothermia to a range of 34-36°C was achieved by employing cooling pads (Variotherm 680-Austria) and cooling the surrounding air. Rectal temperature measurements were utilized to monitor the patient's body temperature. Propofol (3 mg/kg IV) and fentanyl (1 mcg/kg) were used to induce anesthesia. After rocuronium (0.6 mg/kg IV), the patient was intubated and care was given with total intravenous anesthesia (TIVA) (remifentanyl 0.5 mcg/kg/min and propofol 75 mcg/kg/min). The patient was ventilated with maintenance 50% O_2-air inhalation. Subsequent to endotracheal intubation, an intra-arterial cannula was inserted to facilitate invasive blood pressure monitoring. Intermittent blood gas monitoring was performed. Mechanical ventilation was adjusted to maintain the end-tidal carbon dioxide level within a range of 35±1, which was confirmed via blood gas analysis. The isotonic crystalloid was preferred to the maintenance fluid. A 300 mg intravenous infusion of paracetamol (Partemol 1g/100 ml, VEM Drugs) was administered to the patient in the absence of any intraoperative complications to facilitate postoperative pain management. The cooling pads were removed once the patient's rectal body temperature had risen to 37.2°C, which was achieved by increasing the temperature of the pads towards the end of the procedure. The propofol infusion was ceased 10 minutes prior to the conclusion of the operation, while the remifentanil infusion was discontinued with the last skin suture. In addition, 100% oxygen was initiated. The patient was extubated after being decurarized with neostigmine (0.05 mg/kg) and atropine (0.02 mg/kg) postoperatively. The patient's hematocrit levels did not exhibit any alterations that necessitated intervention. The patient's postoperative pain level was assessed using a 10-point visual analog scale (VAS), while sedation was evaluated using the Aldrete scoring system at 0, 10, and 30 minutes. The patient, who had a VAS score of 3 and an Aldrete score of 8, received advanced care and advanced treatment for 1.5 hours after the operation before being transferred to the intensive care unit.

DISCUSSION

The local mass effect of the expanding intrasellar mass on adjacent structures can be observed in all types of pituitary tumors. Most commonly, visual loss occurs due to compression of the optic chiasm due to an adenoma. Intrasellar enlargement can cause anterior pituitary compression and dysfunction leading to hypopituitarism.[3] As with any expanding intracranial mass, patients may experience increased intracranial pressure (ICP). Although rare, patients with headaches accompanied by papilledema, nausea, and vomiting may have increased intracranial pressure. A pituitary tumor may increase ICP directly through the tumor itself or through third ventricle obstruction. If intracranial hypertension is suspected, it is important to avoid maneuvers that may further increase ICP. Preoperative administration of mannitol should be considered to decrease ICP. If the surgical plan includes placement of a lumbar intrathecal drain, the possibility of herniation should always be considered.[4]

Anesthesia for neurosurgical procedures in the pediatric age group is important for patients with a developing central nervous system (CNS). Age-related anatomical and physiological differences distinguish children from adults. Brain tumors in children are the most common solid malignancies. The location and histology of these tumors differ from those of adults.[5]

Compared with adults, there is a higher risk of morbidity and mortality, which may be related to perioperative cardiac and respiratory problems. For this reason, a good history and physical examination are essential before surgery.

Drug and food allergies in children must be thoroughly investigated. It should also be checked for the presence of diseases such as asthma and eczema and whether they have had previous surgery. In addition, findings due to existing cranial pathology should be known. The findings of increased intracranial pressure should be questioned.[2] Lethargy, seizures, cranial nerve involvement, and hormonal deficiencies of the pituitary axis may occur, especially in the presence of pituitary tumors (diabetes insipidus, syndrome of inappropiate secretion of antidiuretic hormone (SIADH), hypo- or hyperthyroidism, adrenal insufficiency, or hormone surges); nausea and vomiting should be questioned. It should be considered that nutritional problems may occur due to nausea and vomiting. Glasgow coma scale should be checked by neurologic examination in the preoperative period. The cardiovascular system should be thoroughly examined and it should be checked if there are shunts or defects such as atrial septal defect and ventricular septal defect. These pathologies alert us to the possibility of a fatal condition, such as air embolism. Because separation anxiety is high in children, premedication with oral or intravenous benzodiazepines may be considered.[6,7]

The goals of anesthesia for transsphenoidal pituitary surgery include optimizing cerebral oxygenation, maintaining hemodynamic stability, managing intraoperative complications, and facilitating rapid and smooth recovery. Invasive arterial monitoring can be necessary in patients with acromegaly or Cushing's disease and heart disease, as they may have significant hemodynamic changes. However, in acromegalic patients with carpal tunnel syndrome, ulnar artery compression can be a problem. These patients may have a "radial dominant" circulation, which means that the radial artery supplies more blood to the hand than the ulnar artery. This can increase the risk of complications from radial artery catheterization, such as ischemia or thrombosis, and should be considered when deciding on the most appropriate site for arterial cannulation.[8] The selection of anesthetic technique is contingent upon the patient's medical comorbidities and prior anesthetic experiences. Strategies that involve swiftly metabolized medications, such as propofol and remifentanil, or inhaled anesthetics with limited blood solubility, such as sevoflurane, may be appropriate to allow for a prompt neurological evaluation. The administration of inhaled anesthetic agents enhanced with remifentanil may confer increased hemodynamic stability and accelerate neurological examination.[9] The most critical situation during induction in these patients is to prevent an increase in intracranial pressure to avoid exacerbation of existing comorbidities. In the evaluation of the impact of inhaled anesthetics on the cerebral system,

the effects on intracranial pressure (ICP) and cerebral vascularity are typically assessed. Volatile anesthetics are recognized as potent cerebral vasodilators that increase cerebral blood flow. Furthermore, all volatile anesthetics diminish cerebral metabolic rate, with isoflurane exhibiting a more significant effect than halothane. Therefore, sevoflurane is the preferred inhaled anesthetic in patients undergoing neurosurgical procedures.[10] In this case, we preferred the combination of remifentanil and propofol for anesthesia maintenance. Studies evaluating the effects of remifentanil on ICP have shown that its administration leads to a reduction in ICP while causing minimal changes in cerebral perfusion pressure (CPP). Therefore, in the neurosurgery intensive care unit, the use of remifentanil is recommended to control ICP, treating high ICP cases that do not respond to propofol/mannitol treatment, and manage agitation that is unresponsive to standard treatments.[11] Transsphenoidal pituitary surgery is typically characterized by low intraoperative blood loss; nonetheless, there is a rare but potentially life-threatening risk of carotid artery injury during the procedure.[12] Maintaining muscle relaxation during the procedure is crucial to avoid any movement by the patient, which may result in CSF leakage, visual pathway injury, or vessel damage. In neuroanesthesia, the decision to administer blood transfusions should be carefully considered, taking into account the benefit/harm ratio of the blood and/or blood products to be administered to the patient. It is important to assess whether the patient actually requires a transfusion, which blood product to select, and the mode of administration. In neurosurgery cases, the target hematocrit level should typically be maintained between 30-33%.

According to a study by Ghahari et al., hypothermia has been recognized as an effective approach to mitigate brain injury resulting from various neurological insults.[13] The neuroprotective effects of mild hypothermia were well documented in experimental models.[14] In our case, mild hypothermia was employed. The use of moderate hypothermia, with body temperature lowered to 33-34°C, is generally acknowledged as neuroprotective against cerebral ischemic injuries while having few side effects. There exist studies that show that a one-hour duration to be enough to lessen neurological and functional deficits, as well as apoptosis in neurosurgical instances involving intraoperative hypothermia.[15]

Care should be taken during the intraoperative position to prevent air embolism, as this is a potential risk. Due to the peculiarities of the intraoperative situation, attention should be paid to the risk of air embolism.[16] Pituitary tumors may cause the syndrome of SIADH or cerebral salt wasting syndrome, and appropriate fluid maintenance and vasopressin dosages should be considered for treatment. Isotonic crystalloids are preferred as the ideal fluid, while hypertonic saline and mannitol can reduce intracranial pressure by drawing fluid from the interstitial and intracellular space into the intravascular space. However, its use in pediatric patients should be approached with caution.[17-19] Postoperative intensive care unit admission with close monitoring of all system parameters is recommended to manage complications such as regression of consciousness, airway obstruction, respiratory depression, cerebral edema, hydrocephalus, bleeding, seizures, tachycardia, hypotension, and hypertension. The most common hemodynamic problem observed is bradycardia.[20,21] Serum sodium osmolality and fluid electrolyte intake and output should be strictly monitored in patients with craniopharyngioma during the preoperative and postoperative periods. Good postoperative analgesia is essential, and opioids should be avoided due to their sedative effects on minute ventilation, which can lead to hypercapnia. Non-steroidal anti-inflammatory drugs (NSAIDs), steroids, and acetaminophen can be used for analgesia. Successful surgical management of patients with pituitary tumors depends on the quality of perioperative care. Pituitary tumors can cause diabetes insipidus, SIADH and cerebral salt wasting syndrome. They can be treated with an appropriate dose of fluid maintenance and vasopressin therapy. Isotonic fluids are preferred as the ideal fluid.

CONCLUSION

The goals of anesthesia for transsphenoidal pituitary surgery include optimizing cerebral oxygenation, maintaining hemodynamic stability, managing intraoperative complications, and facilitating rapid and smooth recovery.

ETHICAL DECLARATIONS

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

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Nurhan Eren
Dr. Nurhan Eren is an anesthesiology specialist at Erzincan Binali Yıldırım University Mengucek Gazi Training and Research Hospital. She is from Erzincan, is married, and has one child.

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Case Report

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A rare case: negative-pressure pulmonary edema after rhinoplasty

Ziya Şencan[1], Kevser Peker[2]

[1]Department of Ear Nose Throat, Faculty of Medicine, Kırıkkale University, Kırıkkale, Turkey
[2]Department of Anesthesiology, Faculty of Medicine, Kırıkkale University, Kırıkkale, Turkey

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Corresponding Author: Ziya Şencan, drsencan@gmail.com

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ABSTRACT

Negative-pressure pulmonary edema (NPPE) is a very rare and dangeorus condition that generally occurs after general anesthesia in otorhinolaryngology practices. Post-obstructive pulmonary edema or NPPE caused by an acute pulmonary edema secondary to upper respiratory tract obstruction. Its sudden appearance in the early postoperative period can stress the surgeon and the anesthesiologist. Difficult breathing effort due to upper airway obstruction creates strong changes in intra-thoracic pressure, and high negative intrathoracic pressure increases the permeability of the alveolar capillary membrane, causing significant fluid changes in the pulmonary tissues. Treatment should be evaluated together with anesthesiologist or pulmonologist and the first goal should be to reduce the hypoxia.

Keywords: Rhinoplasty, airway, pulmonary edema, respiratory distress

INTRODUCTION

Negative-pressure pulmonary edema (NPPE) represents a rare and perilous complication that typically manifests subsequent to general anesthesia in otorhinolaryngologic procedures (1). NPPE, otherwise known as post-obstructive pulmonary edema, is a clinical entity characterized by the acute onset of pulmonary edema that arises as a consequence of an upper airway obstruction.Its sudden appearance in the early postoperative period can stress the surgeon and the anesthesiologist. NPPE encompasses two distinct subtypes. Type I NPPE presents acutely following a forceful inspiratory effort that arises subsequent to upper airway obstruction, as seen in conditions like laryngospasm, foreign body aspiration, and epiglottitis (2). Type 1 NPPE is common in rhinology interventions. Type 2 appears to be secondary to chronic upper airway obstruction, such as adenotonsillar hypertrophy, obesity, obstructive sleep apnea syndrome (2).

Respiratory distress caused by upper airway obstruction elicits marked alterations in intrathoracic pressure, with concomitant increases in negative intrathoracic pressure that promote increased alveolar capillary membrane permeability, ultimately leading to significant fluid shifts within the pulmonary parenchyma

CASE

A 23-year-old ASA I, 80 kg, 180 cm male patient underwent septorhinoplasty due to septum deviation by the otolaryngology clinic. (PA) X-ray (Fig. 1), electrocardiogram (ECG), laboratory tests were considered normal.Standard monitoring was applied (heart rate, non-invasive blood pressure measurement, peripheral oxygen saturation measurement).

In the induction of anesthesia, 2 mg / kg propofol, 1.5 microgram / kg fentanyl and 0.6 mg / kg rocuronium bromide were given and after waiting for the muscle relaxation period, he was intubated with an 8.0 mm size endotracheal tube without any problem.

Anesthesia was maintained with a flow rate of 4 l / min in a mixture of 2% volume sevoflurane, 50% N_2O and 50% O_2. 1000 ml crystalloid (isotonic) fluid was given during the 2-hours operation. There were no respiratory or hemodynamic problems during the surgery.

Following completion of the surgical procedure, the patient received a reversal agent comprising 3 mg neostigmine and 0.5 mg atropine. Subsequently, after ensuring adequate spontaneous ventilation, the patient was extubated without any immediate complications. [110/60 blood pressure (BP), respiratory rate: 15 saturation of peripheral oxygen (SPO_2): 98%]. He was taken to the postoperative care room for follow-up. After half an hour, respiratory distress, severe cough and agitation started. The patient's SPO_2 value started to decrease. An anesthesiologist was urgently consulted. CPAP with 100% O_2 was applied to the patient. However, when the SPO_2 decreased to 70%, positive ventilation was applied with 100% FIO_2. Intravenous 1 mg / kg methyl prednisolone was administered. There were crackles in all lung fields by auscultation. The patient started ,confusing, coughing and producing pink frothy pulmonary secretions.

With a face mask and 6 L / min O_2, the patient's SPO_2 value reached 90%, he was taken to intensive care and chest X-ray was performed (**Figure 2**). On the chest X-ray, signs of pulmonary edema were observed. PH: 7.4, PaO_2: 49.5, $PaCO_2$: 40.5, HCO_3: 25.1, SPO_2: 70% in the arterial blood gas taken in intensive care room. Despite O_2 support, noninvasive positive pressure ventilation (NPPV) was initiated for the patient since the SPO_2 was 70% [applied as: 6x1, Positive End-Expiratory Pressure (PEEP): 8, Fractional İnspiratory Oxygen Concentration (FiO_2): %70]. In conjunction with inhalational administration of bronchodilators and steroids, the patient was initiated on diuretic therapy, specifically furosemide at a dose of 20 mg. The patient's respiratory distress started to regress in the 8th hour postoperatively and the urine volume was 2000 ml. The patient's respiratory distress decreased and the arterial blood gas result came as; pH: 7.39, PaO_2: 59.5, $PaCO_2$: 41.4, HCO_3: 24.7, sPO_2: 85%. Non-invasive positive pressure ventilation was reduced and stopped at the postoperative 20th hour according to blood gas monitoring. The arterial blood gas from the patient was at pH: 7.44, PaO_2: 79.8, $PaCO_2$: 47.10, HCO_3: 29.2, sPO_2: 94%. The patient's general condition and respiratory distress improved. He was transferred to the service on the second day. After 24 hours in the ward, findings were SPO_2: 98% ; BP: 120/75 and respiratory rate:13/minute. He was discharged with full recovery on post-operative third day.

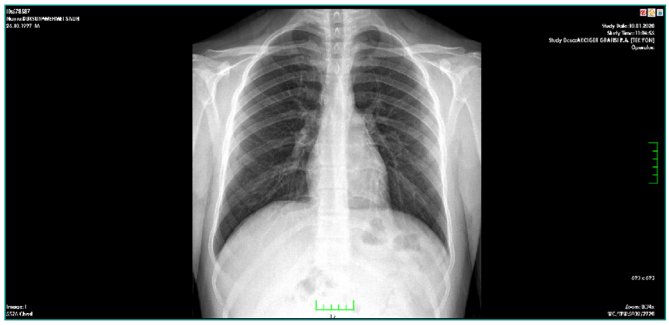

Figure 1. Preoperative PA chest X-ray.

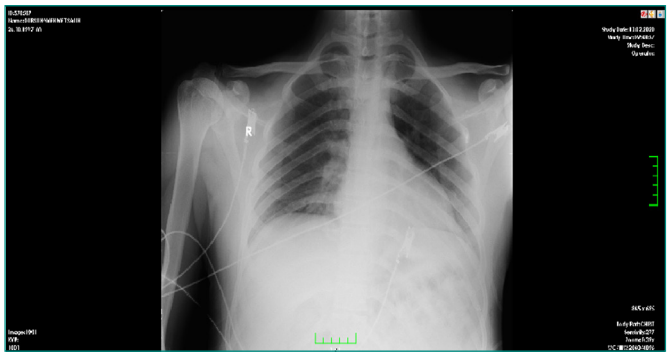

Figure 2. Postoperative half an hourPA chest X-ray.

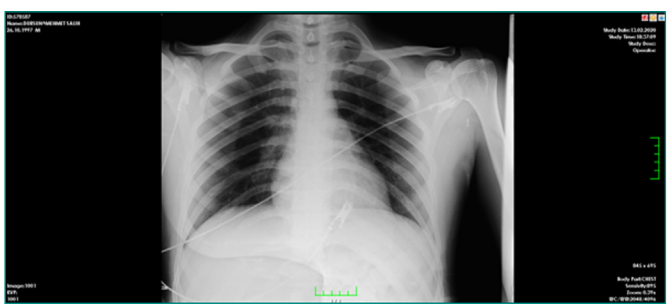

Figure 3. Postoperative fourth day PA chest X-ray.

DISCUSSION

Type I NPPE which develops immediately after the onset acute upper airway obstruction, such as laryngospasm, foreign body aspiration and epiglottitis. Type I is the common reason in otorhinolaryngology practices. In accordance with the literature, we focused on a late-developing type of laryngospasm suggestive of type 1 NPPE. When the literature was reviewed, it was also discussed that there may be a type of drug-induced NPPE, but we considered laryngospasm more prominently (4,5). The complaints of the patient started in the postoperative period (after half an hour), and it was evaluated as the third case with late NPPE in rhinological surgery in the literature (6,7). NPPE must be investigated in the presence of sudden dyspnea and pink frothy sputum in a post-operative patient. The sudden onset of agitation, tachypnea and bilateral common crackles by auscultation guide the examination. For the diagnosis of NPPE, X-ray graphs and arterial blood gas examinations are usually required (6).146 adult NPPE cases compiled in a review;seventy-four cases (50.6%) involved upper aerodigestive tract or deep neck surgery, and 12 (8.2%) involved only the nose (septoplasty and/or rhinoplasty) surgery (1). The patients were intubated for an average of 11.75 hours and three mortalities (2%) occurred (1). In a case report, it was stated that a patient who underwent septoplasty had acute myocardial infarction (AMI) after the delayed diagnosis of NPPE (8). Early diagnosis is very important in NPPE. Physician awareness will help to facilitate early recognition. The most important steps in treatment are to reduce hypoxia and decrease pulmonary edema. If the patient does not have fluid overload, diuretic therapy should be initiated by performing urine output monitoring to reduce pulmonary edema. Noninvasive ventilation should be used to reduce hypoxia. If hypoxia is severe, reintubation and invasive mechanical ventilation should be performed (1). We were successful in the treatment of our patient by using this treatment modality.

CONCLUSION

Negative-pressure pulmonary edema (NPPE) is a very rare and dangerous complication which can lead to mortality if diagnosed late. After otolaryngology surgeries, patients should be observed postoperatively and warning signs such as sudden onset of breathing difficulty and pink sputum should be kept in mind. Treatment should be evaluated together with anesthesiologist or pulmonologist and the first goal should be to reduce the hypoxia.

ETHICAL DECLARATIONS

Informed Consent: All patients signed the free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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Ziya Şencan

He was born in Ankara in 1984. After graduating from medical school, he received his ENT specialty at Ankara Oncology Education Research Hospital. He is still working as an associate professor in Kırıkkale University, Faculty of Medicine, ENT department.