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The Effect of Nasal *Staphylococcus Aureus* Colonization on Idiopathic Epistaxis

Nazal *Staphylococcus Aureus* Kolonizasyonunun İdiyopatik Epistaksis Üzerine Etkisi

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ABSTRACT Objective: Idiopathic epistaxis is a common condition in children. This study aims to investigate the relation between *Staphylococcus aureus* and 'idiopathic epistaxis' in paediatric patients with idiopathic epistaxis. **Material and Methods:** The causes of nosebleeds in patients admitted to the hospital were investigated. After the known causes and the causes revealed by examination were excluded, patients diagnosed with idiopathic epistaxis were included in the study. All blood parameters related to inflammation were evaluated. Nasal swap samples were collected from the nose. Mean platelet volume (MPV), platelet, hemoglobin, hematocrit, lymphocyte, leukocyte, neutrophil-lymphocyte ratio (NLR), platelet-lymphocyte ratio (PLR), and systemic immune inflammation index (SII) levels of these patients were compared with age- and sex-matched controls. **Results:** Eighty six patients were included in the study. Thirty six of 86 patients were female and 50 were male. The mean age was 11.4±5.9 years. The age and gender distribution of the patients did not differ significantly between the groups with and without *S. aureus*. *S. aureus* growth was observed in 28 (32.6%) patients. Platelet count, neutrophil count, lymphocyte count, MPV, N/LR, P/LR, and SII values did not differ significantly between groups with and without *S. aureus*. **Conclusion:** Our study affirms the colonization of *S. aureus* in a specific group of idiopathic epistaxis patients. However, no significant differences in systemic inflammatory markers, including NLR, PLR, and SII, were found between patients with and without *S. aureus* colonization. This indicates that bacterial colonization plays a role in idiopathic epistaxis through local inflammatory effects.

Keywords: Idiopathic epistaxis; *Staphylococcus aureus*; bacterial colonization; systemic immune inflammation index

ÖZET Amaç: İdiyopatik burun kanaması çocuklarda sık görülen bir durumdur. Bu çalışma, idiyopatik burun kanaması olan pediatrik hastalarda *Staphylococcus aureus* ile idiyopatik burun kanaması arasındaki ilişkiyi araştırmayı amaçlamaktadır. **Gereç ve Yöntemler:** Hastaneye başvuran hastalarda burun kanamasının nedenleri araştırıldı. Bilinen nedenler ve muayene ile ortaya çıkan nedenler dışlandıktan sonra idiyopatik burun kanaması tanısı alan hastalar çalışmaya dâhil edildi. İnflamasyonla ilgili tüm kan parametreleri değerlendirildi. Burundan nazal sürüntü örnekleri toplandı. Bu hastaların ortalama trombosit hacmi [mean platelet volume (MPV)], trombosit, hemoglobin, hematokrit, lenfosit, lökosit, nötrofil-lenfosit oranı [neutrophil-lymphocyte ratio (NLR)], trombosit-lenfosit oranı [platelet-lymphocyte ratio (PLR)] ve sistemik immün inflamasyon indeksi (Sİİ) düzeyleri yaş ve cinsiyet uyumlu kontroller yaş ile karşılaştırıldı. **Bulgular:** Çalışmaya 86 hasta dâhil edildi. Seksen altı hastanın 36'sı kadın, 50'si erkekti. Ortalama yaş 11,4±5,9 yıldı. Hastaların yaş ve cinsiyet dağılımı *S. aureus* olan ve olmayan gruplar arasında anlamlı farklılık göstermedi. Hastaların 28'inde (%32,6) S. *aureus* üremesi görüldü. Trombosit sayısı, nötrofil sayısı, lenfosit sayısı, MPV, N/LR, P/LR ve Sİİ değerleri *S. aureus* olan ve olmayan gruplar arasında anlamlı farklılık göstermedi. **Sonuç:** Çalışmamız idiyopatik burun kanaması hastalarından oluşan spesifik bir grupta *S. aureus* kolonizasyonunu doğrulamaktadır. Ancak *S. aureus* kolonizasyonu olan ve olmayan hastalar arasında NLR, PLR ve Sİİ dâhil olmak üzere sistemik inflamatuar belirteçlerde anlamlı bir fark bulunmadı. Bu durum idiyopatik burun kanamasında lokal inflamatuar etkiler yoluyla bakteriyel kolonizasyonun rol oynadığını göstermektedir.

Anahtar Kelimeler: İdiopatik epistaksis; *Stafilokokkus aureus;* bakteriyel kolonizasyon; sistemik immün inflamasyon indeksi

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Epistaxis is a common cause of presentation to ear nose and throat outpatient clinics in paediatric patients. Many things can cause this, the most common cause is trauma. There are many causes such as digital trauma, local inflammation, and infective disorders. The most common site is the anterior nasal septum. The most common place to bleed is the Little area.[1] Nosebleeds, or epistaxis, pose a potential threat to individuals of all ages, making them a matter of concern. The nose, being highly vascularized, is susceptible to minor trickles, acute bleeding episodes, or prolonged bleeding. The primary objective in managing nosebleeds is to promptly stop the bleeding, recognizing it as a sign of a potentially harmful situation.[2]

Staphylococcus aureus, commonly found in the human nasal cavity, can also exist in various other body sites, including the skin, pharynx, perineum, vagina, axilla, and gastrointestinal tract. Nasal carriage of *S. aureus* predisposes individuals to autoinfections, potentially leading to methicillin-resistant strains. Furthermore, *S. aureus* is capable of causing infections in multiple organ systems, such as skin, soft tissues, septic arthritis, and osteomyelitis. It is essential to emphasize that these infections are not confined to a specific body part. Individuals with nasal carriage may experience increased vascularity and infective discharge in the nasal mucosa. Approximately 30% of healthy individuals in the community carry *S. aureus* in their nasal cavity, potentially leading to infections.[3] Children with nosebleeds are more likely to have nasal colonization with *S. aureus*, a bacterium commonly found in the nasal cavity. In patients with epistaxis, *S. aureus* replaces the existing nasal flora, leading to changes in nasal flora that increase mucosal vascularity and local inflammation. While this carrier of *S. aureus* may not always exhibit symptoms in the body, it can sometimes manifest as idiopathic epistaxis.[1,3,4]

Idiopathic epistaxis, characterized by spontaneous nosebleeds without an obvious cause, lacks a precise definition. Laboratory investigations, notably the complete blood count (CBC), play a pivotal role in evaluating epistaxis. Idiopathic epistaxis might be linked to inflammation, making it crucial to assess indicators of inflammation and blood loss through a CBC. Mean platelet volume (MPV) serves as a straightforward measure of platelet function, indicating the average size of platelets. Elevated MPV suggests larger, more effective platelets in hemostasis due to their increased production of vasoactive and thrombotic factors. CBC, including MPV measurement, aids in estimating the risk of bleeding. Neutrophil/lymphocyte ratio (NLR) shows promise as an inflammation marker, while platelet/lymphocyte ratio (PLR) serves as an indicator for both inflammation and thrombotic events. NLR and PLR prove invaluable in differential diagnosis and prognostic predictions. Several factors can influence MPV, NLR, PLR, and other CBC parameters, which have been extensively evaluated in various diseases.[5]

NLR, calculated as the ratio of neutrophil count to lymphocyte count, stands out as a superior marker for systemic inflammation compared to other white blood cell subtype counts like neutrophils, lymphocytes, and total leukocytes.[6] The systemic immune inflammation index (SII), an innovative biomarker, integrates neutrophil, lymphocyte, and platelet counts to reflect the inflammatory state. Initially utilized for evaluating prognosis in solid cancer and coronary heart disease, SII now serves as an accurate indicator of inflammation.[7,8]

Obtaining a nasal culture from pediatric patients presenting to the clinic with complaints of idiopathic epistaxis can be challenging at times. Waiting for culture results can be time-consuming for patients in the clinic and may incur additional costs. Additionally, the presence of *S. aureus* causing epistaxis only with local inflammation may necessitate the use of topical antibiotic cream. Considering all these reasons, it becomes important to determine whether the cause of inflammation is local inflammation caused by *S. aureus* or a systemic inflammatory response.[3]

In this study, we investigated whether the presence of *S. aureus* in the nose of pediatric patients has a role in epistaxis with systemic effect.

MATERIAL AND METHODS

This study is a randomized controlled prospective study. Participants included in this study were individuals who presented to the outpatient clinic without

active epistaxis. Participants were paediatric patients (younger than 18 years) presenting to the outpatient clinic with epistaxis of unknown cause. All patients were subjected to a detailed history and clinical examination. Only patients complaining of epistaxis were included in the study and the groups were divided according to culture results. Those patients with epistaxis resulting from surgical or traumatic causes, medical conditions such as inherited bleeding disorders, liver-cell failure, or aspirin or warfarin medication, or were over 18 years old were excluded. Patients with tonsillar hypertrophy at Stage 2 or 3, and those with adenoid hypertrophy exceeding 25%, were also excluded. Patients whose culture samples showed different bacterial agents were excluded from the study. Those with co-existing medical conditions were excluded from the investigation. Patients with elevated inflammation markers in blood parameters and patients with active infection were excluded from the study. Infections such as upper respiratory tract infections cause elevated acute phase reactants. Infections can cause epistaxis. Therefore, patients with high acute phase reactants were excluded from the study.[9,10]

The intraoral, oropharyngeal, and nasal passages of each participant were evaluated through anterior rhinoscopy, and nasal endoscopy was performed to assess up to the nasopharynx. The posterior choana and nasopharynx of examined children who tolerated the examination were evaluated using 0 degrees rigid telescopes. Nasal swabs can be used to screen the general population for the detection of bacterial or viral pathogens.[9] After cleaning the area with sterile saline, cotton swab moistened with physiological sterile saline were rolled two to three times in both the anterior nasal cavity. The swab was then sent to the microbiology department where the swab was cultured on blood agar, chocolate agar and MacConckey agar. The plates were incubated for 18-24 h at 37 ºC. The colony morphology and other traits were documented, and Gram staining was performed. A coagulase test was conducted to isolate the causative species, and methicillin resistance was detected through agar screening.

CBC, coagulation profile, and liver function tests were performed on blood samples from all subjects to rule out any systemic causes of epistaxis. Demographic characteristics, blood coagulation profile, neutrophil count, white blood cell count, lymphocyte count, as well as the NLR, PLR, and SII MPV, C-reactive protein (CRP) of all patients were analyzed.

This study adhered to the ethical principles of the Declaration of Helsinki and received approval from the Ümraniye Research and Education Clinical Research Ethics Committee (date: December 22, 2022, no: 410). Informed consent form was obtained from the patients.

STATISTICAL ANALYSIS

In the descriptive statistics of the data, mean, standard deviation, median minimum, maximum, frequency, and ratio values were used. The Kolmogorov-Smirnov test was used to measure the distribution of variables. Quantitative data were analysed using independent sample t-tests and Mann-Whitney U-tests, while qualitative independent data were analysed using the chi-square test. SPSS 27.0 (SPSS Inc., ABD), was used for statistical analyses. Statistical significance was set at $p<0.05$.

RESULTS

Out of the initial 122 patients presenting with idiopathic epistaxis, 4 were excluded due to the growth of *S. epidermidis* in their nasal cultures, and 1 exhibited enterococcal growth. The patient from the orphanage was also excluded from the study because he had *enterococcus* growth. Additionally, 16 participants were excluded due to upper respiratory tract infections and acute sinusitis, while 6 patients were excluded due to hematological and endocrinological disorders. Patients with a history of hormone replacement therapy involving corticosteroids (n=2), chronic renal failure (n=2), and autoimmune diseases (n=4) were also excluded. Finally, the study comprised 86 patients (Figure 1). Among these participants, 36 were female, and 50 were male, with an average age of 11.4±5.9 years. Nasal swabs were collected from all patients, and *S. aureus* growth was detected in 28 patients (32.6%). Of these, 8 were methicillin-resistant *S. aureus*, while no *S. aureus* growth was observed in 58 patients (67.4%) (Table 1).

No significant differences in age and gender distribution were noted between the groups with and

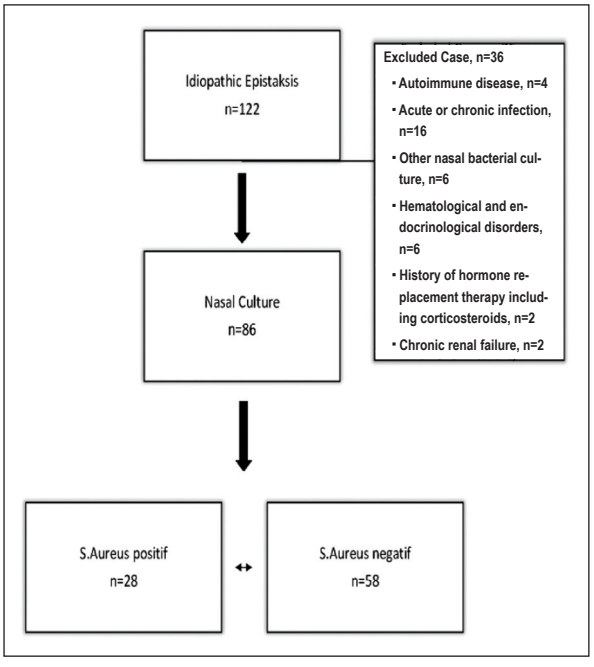

FIGURE 1: Selection of patients.

without *S. aureus* (p>0.05) (Table 2). Hemoglobin, hematocrit, red blood cell count, white blood cell count, mean corpuscular volume, alanine transaminase, aspartate transaminase, CRP, and prothrombin time values did not show any significant differences between the groups with and without *S. aureus* (p>0.05) (Table 2).

However, the activated partial thromboplastin time was significantly higher (p<0.05) in the group with *S. aureus* compared to the group without it (Table 2). International normalized ratio, platelet count, neutrophil count, lymphocyte count, MPV, NLR, PLR, and SII values showed no significant differences between the groups with and without *S. aureus* (p>0.05) (Table 2). No difference was observed in the inflammation markers between patients with methicillin-resistant *S. aureus* and those with methicillin-sensitive *S. aureus*.

TABLE 1: Descriptive analyzes and blood results.

		Minimum-Maximum	Median	X̄±SD/n-%	
Age		3.0-17.0	11.0	11.4±5.9	
Gender	Female			36	41.9%
	Male			50	58.1%
Additional illness	(-)			86	100.0%
S. aureus	(-)			58	67.4%
	(+)			28	32.6%
HG		11.7-16.4	13.5	13.8±1.3	
HCT		35.2-50.3	40.4	41.2±3.8	
RBC		4.0-6.0	5.0	5.0±0.4	
WBC		4.0-14.3	7.7	7.8±2.4	
MCV		67.4-95.2	82.6	82.1±5.7	
ALT		4.9-47.7	14.0	16.6±9.0	
AST		9.9-35.0	22.3	22.6±6.1	
CRP		0.1-5.4	0.6	1.3±1.4	
PT		11.8-16.5	13.8	14.0±1.1	
aPTT		20.6-39.0	30.7	31.1±3.4	
INR		0.9-1.8	1.1	1.1±0.1	
PLT		154.0-477.0	296.0	292.1±69.7	
Neutrophil		1.8-10.3	3.5	3.9±1.8	
Lympocyte		1.3-5.0	3.0	3.0±0.9	
MPV		7.0-11.3	9.3	9.2±0.9	
N/LR		0.4-4.8	1.2	1.4±0.8	
P/LR		51.9-262.6	97.9	103.6±34.2	
SII (PxN)/L		141.2-1259.0	356.7	391.7±229.6	

Hg: Hemoglobin; HCT: Hematocrit; RBC: Red blood cell; WBC: White blood cells; MCV: Mean corpuscular volume; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; CRP: C-reactive protein; PT: Protrombin time; aPTT: Activated partial thromboplastin time; INR: International normalized ratio; PLT: Platelet; MPV: Mean platelet volume; N/LR: Neutrophil lymhocyte ratio; P/LR: Platelet lymphocyte ratio; SII (PxN/L): Systemic Inflammation Index; SD: Standard deviation.

TABLE 2: Comparative analyzes of *Staphylococcus aureus* positive and negative patients.

		S. aureus (-)		*S. aureus (+)*			
		X̄±SD/n-%	**Median**	**X̄±SD/n-%**	**Median**	**p value**	
Age		11.5±6.7	11.0	11.3±3.9	11.0	0.919	t
Gender	Female	22	37.9%	14	50.0%	0.452	X^2
	Male	36	62.1%	14	50.0%		
HG		13.8±1.3	13.5	13.9±1.2	13.8	0.730	t
HCT		40.7±3.9	39.8	42.3±3.7	41.8	0.201	t
RBC		5.0±0.4	5.1	5.0±0.5	5.0	0.809	t
WBC		8.3±2.3	8.1	6.9±2.1	6.1	0.063	t
MCV		81.5±5.9	81.3	83.5±5.2	83.5	0.284	t
ALT		17.6±9.2	14.1	14.4±8.5	12.7	0.262	m
AST		23.0±6.4	22.0	21.7±5.7	23.0	0.520	t
CRP		1.4±1.5	0.6	1.3±1.2	0.7	0.856	m
PT		14.1±1.0	14.1	13.8±1.3	13.4	0.289	t
APTT		30.4±2.5	29.9	32.4±4.7	31.9	0.029	m
INR		1.1±0.1	1.1	1.1±0.2	1.1	0.603	m
PLT		293.8±69.1	305.0	288.6±73.5	281.5	0.821	t
Neutrophil		4.2±1.9	3.7	3.4±1.2	3.4	0.294	m
Lymphocyte		3.1±0.9	3.1	2.8±0.9	2.7	0.287	t
MPV		9.1±0.8	9.3	9.4±0.9	9.5	0.255	t
N/L R		1.4±0.8	1.2	1.3±0.6	1.3	0.856	m
P/L R		99.4±25.5	97.9	112.4±47.4	100.0	0.659	m
SII(PxN)/L		401.4±252.9	355.1	371.6±178.7	363.3	0.938	m

[t]: Independent sample t-test; [m]: Mann-Whitney U test; X^2: Chi-square test; Hg: Hemoglobin; HCT: Hematocrit; RBC: Red blood cell; WBC: White blood cells; MCV: Mean corpuscular volume; ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; CRP: C-reactive protein; PT: Protrombin time; aPTT: Activated partial thromboplastin time; INR: International normalized ratio; PLT: Platelet; MPV: Mean platelet volume; N/LR: Neutrophil lymhocyte ratio; P/LR: Platelet lymphocyte ratio; SII (PxN/L): Systemic Inflammation Index; SD: Standard deviation.

DISCUSSION

Epistaxis is a condition that can be caused by many diseases that affect all age groups. Epistaxis can be triggered by mucosal dryness, inflammation in the local area, and manual nasal cleaning. Epistaxis affects 30% of children aged 0-5, 56% aged 6-10, and 64% aged 11-15. Nosebleeds typically arise from the anterior septal area, frequently from the Kisselbach's plexus (Little's region) among young patients, and generally result from non-malignant circumstances - a deviation from their presentation in adult patients. Little's area is covered by a thin mucosal septum and possesses a fragile network of blood vessels that can be readily damaged, resulting in bleeding even with minor trauma. Extensive vascular structure in the nasal mucosa and heightened incidence of upper respiratory tract infections in children render it vulnerable to epistaxis. Trauma is undoubtedly the leading cause, with nose rubbing or digital trauma commonly associated with inflammation or infection being a significant contributor. Clear diagnosis is crucial in identifying the most likely cause of trauma, given that this can assist in determining the best possible course of treatment. The process of colonization can lead to inflammation, crusting, and the development of new blood vessels which in turn results in epistaxis. There is a significant deficiency in literature concerning the influence of nasal bacterial colonization and blood parameters in children.[10] Previous studies have revealed that nasal colonization with *S. aureus* is associated with an increased frequency of epistaxis. It was believed that *S. aureus* could cause epistaxis as a result of local inflammation. Our study examined the relationship between blood parameters in patients with and without bacterial colonization. However, no significant differences were found between the two groups in terms of NLR, PLR, and SII ratios.[1,5,11]

In patients with adenoid hypertrophy and tonsillar hypertrophy, increased dryness or local inflammation in the nose is possible due to factors such as infection and snoring. The susceptibility to bacterial infection rises in patients suffering from adenoid hypertrophy. Patients with acute or chronic adenoid and tonsillar hypertrophy have been excluded from the study, as blood parameters could be affected during acute adenoiditis.[12]

There is a correlation between platelet activation in the pathophysiology of thrombosis and conditions susceptible to inflammation. Elevated levels of MPV, which are linked to thrombosis, signify enhanced platelet activation. Whilst the primary purpose of platelets is to facilitate haemostasis, they are also capable of discharging inflammatory mediators. Thus, heightened platelet activation is correlated with inflammation. High MPV levels have been linked with low-grade inflammatory conditions. Individuals colonised with *S. aureus* may experience chronic and mild inflammation accompanied by irritation.[5] In our study, no difference in MPV was found between the two groups. *S. aureus* is the primary cause of community-acquired sepsis and a significant nosocomial pathogen. It leads to a wide range of diseases, including lung and urinary tract infections, toxin-induced diseases, skin and soft tissue infections, and deep tissue infections.[13] According to Montague et al. in paediatric epistaxis, *S. aureus* is the first bacterium to colonise the child's nose. This results in mild inflammation accompanied by crusting and irritation. Nosebleeds in this instance could possibly be attributed to digital trauma, increased vascularity resulting from inflammation and trauma resulting from detachment of the crusts. Prolonged inflammation results in the growth of new vessels, a process known as neovascularization, which often leads to the formation of prominent vessels. The growth of these vessels is stimulated by the release of inflammatory mediators.[4] Colonisation of *S. aureus* in the nasal vestibule is more probable to happen in individuals experiencing recurrent nosebleeds than in those who do not. It appears that this colonization may play a part in the development of epistaxis. However, it is also possible to consider that this colonization may be caused by epistaxis, given the changing environment of the nasal vestibule after each period of nosebleed.[14] The study found that the frequency of *S. aureus* colonising the nasal vestibule was similar to the general population. We evaluated the signs of inflammation in the blood in patients with *S. aureus* growth due to colonization and in patients without colonization. Whilst the overall population showed a 25% average of *S. aureus* carriage, our study identified S. aureus growth in 14 out of 43 idiopathic epistaxis patients.[4,15] The reproduction rate, which stood at 32.6%, was marginally greater than that of the general populace. Although carriage of *S. aureus* was higher in patients with idiopathic epistaxis in comparison to the general population, there was no alteration in SII, NLR, or PLR. The prevalence of *S. aureus* carriage varies across countries, with Konatrat et al. estimating a rate of 20-30% depending on the sample collection method used.[15] The small discrepancy in the observed frequency suggests that further inquiry should be made into the occurrence of *S. aureus* in patients experiencing idiopathic epistaxis.[15]

The study faced significant limitations, primarily due to the small sample size utilized. This limitation underscores the need for future studies to adopt larger patient cohorts, serving as a model for more comprehensive research. In this study, it is a limitation that the patients were not retrospectively questioned about their digitalization history, their history of admission to another hospital due to epistaxis, and that their previous bleeding history and frequency were not questioned. Additionally, our study did not assess local inflammation, posing another limitation. Further research endeavors are necessary to establish a clear correlation between local inflammation and blood parameters. Despite the absence of differences in blood parameters between the *S. aureus* positive and negative groups, it is essential to note that this lack of alteration does not negate the importance of nasal culture. Nasal culture remains valuable, especially for individuals experiencing local inflammation and idiopathic epistaxis that does not respond to conventional treatment methods. However, to provide a more reliable evaluation, it is imperative to conduct further investigations through numerous additional studies. Another limitation worth mentioning is that nasal

swab samples were obtained only once during the study.[15] To address this limitation, future studies should consider collecting swab samples at various intervals. This approach would enable the assessment of both permanent and temporary carriers, leading to a more comprehensive understanding of the subject matter.

CONCLUSION

Our study affirms the colonization of *S. aureus* in a specific group of idiopathic epistaxis patients. However, no significant differences in systemic inflammatory markers, including NLR, PLR, and SII, were found between patients with and without *S. aureus* colonization. This implies a complex interplay between bacterial colonization, local inflammation, and systemic inflammatory markers. We emphasize that local inflammation resulting from *S. aureus* colonization is effective in nosebleeds.

Source of Finance

During this study, no financial or spiritual support was received neither from any pharmaceutical company that has a direct connection with the research subject, nor from a company that provides or produces medical instruments and materials which may negatively affect the evaluation process of this study.

Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: *Fatih Savran;* ***Design:*** *Fatih Savran;* ***Control/Supervision:*** *Yaşar Kemal Duymaz;* ***Data Collection and/or Processing:*** *Fatih Savran, Yaşar Kemal Duymaz;* ***Analysis and/or Interpretation:*** *Fatih Savran;* ***Literature Review:*** *Yaşar Kemal Duymaz;* ***Writing the Article:*** *Fatih Savran;* ***Critical Review:*** *Fatih Savran;* ***References and Fundings:*** *Yaşar Kemal Duymaz;* ***Materials:*** *Fatih Savran.*

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