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# The effect of ketofol anesthesia on intraocular pressure in pediatric strabismus surgery

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**Ethics Committee Approval**
The study was approved by the Omangazi University Ethical Committee (no: 25; November 22, 2022).
All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

**Conflict of Interest**
No conflict of interest was declared by the authors.

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## Abstract

**Background/Aim:** Keeping intraocular pressure (IOP) within normal limits is an important goal in the anesthetic management of pediatric strabismus surgery. While propofol is commonly used as an induction agent since it provides smooth laryngeal mask insertion, it has the undesirable side effect of dose-dependent cardiorespiratory depression. On the other hand, ketamine acts as a sympathetic cardiorespiratory stimulant; however, its effect on IOP is controversial. The aim of this study was to determine the effect of the combination of ketamine and propofol (ketofol) on IOP in pediatric strabismus surgery compared to propofol alone.

**Methods:** Participants included patients aged between 2 and 18 years who underwent strabismus surgery. They were divided into two groups according to type of anesthesia induction: propofol and ketofol. Patient characteristics, surgical data, hemodynamic parameters, oculocardiac reflex (OCR), and IOP were compared between the two groups.

**Results:** Forty-five children with a mean age of 7.7 years were enrolled in the study. The patients were assigned into two groups: propofol alone (n=26) and ketofol (n=19). The groups were similar in patient characteristics, surgical data, and hemodynamic parameters ($P$<0.05 for each). IOP was measured at four points: before anesthesia, at 1 minute following induction, at 3 minutes following laryngeal mask airway (LMA) insertion, and at the end of surgery. All IOP values were within normal limits. No significant differences in mean IOP values were found between the groups ($P$>0.05 for each). There was also no significant difference in OCR between the groups ($P$=1.000).

**Conclusions:** Compared to propofol alone, ketofol had a similar effect on IOP, OCR, and hemodynamic parameters. These results suggest that ketofol can be safely used in the induction of anesthesia in pediatric patients undergoing strabismus surgery.

**Keywords:** induction of anesthesia, intraocular pressure, ketamine, ketofol, propofol

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## Introduction

Strabismus surgery, a common procedure in children, involves the surgical intervention for the extraocular muscles that provide movement of the eyeball. This surgery is always performed under general anesthesia, and, as in other ophthalmic procedures, keeping intraocular pressure (IOP) within normal limits is an important goal. An increase of more than 10 mmHg in IOP reduces choroidal blood flow and ocular fundus vibrations in healthy eyes, while an increase of more than 5 mmHg may lead to choroidal and optic nerve ischemia in injured eyes [1].

All anesthetic drugs affect IOP, with varying severity depending on the depth of anesthesia [2]. Propofol is a widely preferred induction agent in pediatric strabismus surgery as it ensures smooth laryngeal mask airway (LMA) insertion by depressing airway reflexes [3]. It also has rapid induction and recovery times, with strong antiemetic effects. On the other hand, dose-dependent cardiorespiratory depression is the leading undesirable adverse effect of propofol, particularly at higher doses [4]. To minimize this potential hemodynamic instability, several combinations of pharmacological drugs have been introduced to anethesia protocols. Although some clinical studies on cataract surgery have demonstrated that anesthetic drugs such as propofol and fentanyl do not significantly change IOP, the effect of ketamine on IOP remains controversial [5]. It is widely known that ketamine is not used alone as an induction agent because it causes excessive secretions and does not adequately suppress airway reflexes. However, unlike propofol, it acts as a sympathetic cardiorespiratory stimulant and has pain-relieving properties [6]. In the literature, several studies have shown that hemodynamic stability can be achieved with the combined use of ketamine and propofol in pediatric cases under general anesthesia with LMA [7–10]. However, most of those studies involved sedation and general anesthesia for non-specific surgical indications and were conducted on different patient cohorts consisting of both pediatric and adult groups. Because changes in IOP are more important in pediatric patients undergoing ophthalmic surgery, it is crucial for anesthesiologists to understand the effects of preferred anesthesia drugs on IOP in pediatric patients.

Therefore, this study aimed to determine the effect of ketofol-based anesthesia protocol on IOP in pediatric patients who underwent strabismus surgery.

## Materials and methods

### Study design

The study, which was approved by the Local Ethics Committee of Eskişehir Osmangazi University Faculty of Medicine (protocol number: 25, date: 22 November 2022), was conducted on pediatric patients who underwent elective surgery for strabismus under general anesthesia with LMA at Osmangazi University Hospital between October 2020 and June 2021. Patient data including age, gender, American Society of Anesthesiologist (ASA) physical status, operative findings, anesthetic medications, and complications were recorded. Intraoperative hemodynamic parameters including heart rate (HR), mean arterial pressure (MAP), pulse oximetry ($SpO_2$), end-tidal carbon dioxide concentration ($etCO_2$), minimal alveolar concentration (MAC) of sevoflurane, tidal volume (TV), peak airway pressure (PAP), and IOP were also noted. The patients were randomly assigned into two anesthesia induction groups—propofol alone and ketofol—and compared in terms of patient characteristics, surgical data, hemodynamic parameters, occurrence of oculocardiac reflex (OCR), and IOP values.

### Inclusion and exclusion criteria

Inclusion criteria were that the patients were between 2 and 18 years old, had an ASA physical status of 1 or 2, and were undergoing an elective surgery. Children with an ASA physical status ≥3, high IOP, previous ocular surgery, allergy to the study medications, irregular medical records, or any ophthalmic, cardiac, or central nervous system disease were excluded from the study.

### Anesthesia management

The fasting time was at least 8 hours before the operation. No premedication was given to the children. After standard monitoring, anesthesia was administered via a face mask with inhalation of sevoflurane in 4 L/min oxygen (50%) and air (50%). Venous access was opened following sufficient loss of consciousness. The patients were randomly assigned to one of the two induction groups: remifentanil (1 μg/kg), lidocaine (0.5 mg/kg), and propofol (3–5 mg/kg) (propofol alone group) or propofol (2.5–3 mg/kg) plus ketamine (1.5 mg/kg) (ketofol group). LMA was used for airway control. Anesthesia was maintained with sevoflurane (with age-corrected 1–1.3 MAC) in 4 L/min oxygen (50%) and nitrous oxide (50%). Weight-appropriate continuous IV fluids (mixture of 0.45% NaCl and 5% dextrose) were given to the patients throughout the surgery. Standard monitoring was conducted for HR, MAP, $SpO_2$, and $etCO_2$.

OCR was defined as a 20% decrease in HR or the presence of dysrhythmia following the traction of extraocular muscle. In the case of OCR lasting for more than 5 seconds, the surgeon was warned to stop the traction of muscle and to wait until the HR returned to normal rhythm. IV atropine (0.01 mg/kg) was given to patients when a persistent OCR or rapid drop in HR <60 /min was observed. The surgeon continued the surgery after the values returned to normal limits. All surgical interventions were performed by the same surgeon using standard techniques.

### Measurement of IOP

IOP was measured four times during the procedure. The first measurement (baseline) was performed when spontaneous breathing was lost and the patient was fully immobilized. The other measurements were performed at 1 minute following induction of anesthesia, at 3 minutes following insertion of LMA, and at the end of the surgery. All measurements of IOP were performed using the same tonometry device (Tonopen XL, Reichert Technologies, Depew, NY) by the same ophthalmologist, who was blinded to the type of anesthesia protocol being used.

### Statistical analysis

A power analysis based on the study by Aydoğan et al. [11] showed that a sample size of 37 patients was required to achieve 95% power with a 5% significance level to assess the differences between the two anesthesia groups. Statistical

analyses were performed using the Statistical Package for the Social Sciences (SPSS 23.0 software). Descriptive data were presented as numbers (%) for the categorical variables and as mean (standard deviation) for the continuous variables. Chi-square test, Mann-Whitney U test, and Fisher's exact test were used to assess the differences between the groups. A *P*-value <0.05 was accepted as the level of significance.

## Results

Forty-five patients with a mean age of 7.7 years were included in the study. There were 23 (51.1%) boys and 22 (48.9%) girls. All patients had a preoperative ASA physical status of 1 or 2. All patients underwent strabismus surgery, including 19 (42.2%) single-eye and 26 (57.8%) double-eye procedures. The patients were classified into two anesthesia induction groups: propofol alone (n=26) and ketofol (n=19). The two groups were similar in basic characteristics and surgical data (Table 1).

Table 1: Comparison of two groups in terms of basic patient characteristics and surgical data

| Characteristics | Propofol alone (n= 26) | Ketofol (n=19) | *P*-value |
|---|---|---|---|
| **Age (y)** | 8.5 (3.9) (3-17) | 6.5 (3.4) (2-14) | 0.108 |
| **Weight (kg)** | 31.8 (14.8) (14-65) | 23.7 (11.4) (11-60) | 0.053 |
| **Gender (F/M)** | 15 (57.7%)/11 (42.3%) | 7 (36.8%)/12 (63.2%) | 0.231 |
| **ASA status (ASA 1/ASA 2)** | 20 (76.9%)/6 (23.1%) | 17 (89.5%)/2 (10.5%) | 0.211 |
| **Laterality** | | | 0.241 |
| Unilateral | 13 (50%) | 6 (31.6%) | |
| Bilateral | 13 (50%) | 13 (68.4%) | |
| **Operated muscle** | | | 0.493 |
| Medial rectus | 11 (42.3%) | 8 (42.1%) | |
| Lateral rectus | 8 (30.7%) | 9 (47.4%) | |
| Others | 6 (23%) | 2 (10.5%) | |
| **Type of surgery** | | | 0.624 |
| Recession | 18 (69.2%) | 16 (84.2%) | |
| Resection | 6 (23%) | 2 (10.5%) | |
| **Duration of procedure** | 36.7 (13.1) (15-60) | 41.5 (13.5) (10-55) | 0.192 |

y: year, kg: kilogram, F: female, M: male. Data are presented as mean (standard deviation) (minimum-maximum) for age, weight, and duration of procedure; n (%) for other variables.

Hemodynamic parameters including HR, MAP, $SpO_2$, $etCO_2$, MAC of sevoflurane, TV, and PAP were continuously monitored during the procedure. These parameters were recorded at four points: before anesthesia (baseline), 1 minute following induction, 3 minutes following LMA insertion, and at the end of the surgery. All hemodynamic parameters were similar between the patients in the propofol group and the patients in the ketofol group (Table 2).

Table 2: Comparison of hemodynamic parameters between the two induction groups

| Variables | Propofol alone (n= 26) | Ketofol (n=19) | *P*-value |
|---|---|---|---|
| **HR (baseline)** | 103.5 (18.6) (72-141) | 106.9 (19.8) (85-160) | 0.704 |
| **MAP (baseline)** | 81.2 (12.9) (56-110) | 81.3 (11.4) (61-112) | 0.740 |
| **$SpO_2$ (baseline)** | 99.4 (0.6) (98-100) | 99.5 (0.7) (98-100) | 0.404 |
| **HR (1st min of induction)** | 93.5 (15.2) (58-121) | 101.3 (17.7) (67-131) | 0.110 |
| **MAP (1st min of induction)** | 73.2 (14.5) (53-107) | 71.3 (10.8) (50-92) | 0.927 |
| **$SpO_2$ (1st min of induction)** | 99.4 (0.6) (98-100) | 99.6 (0.6) (98-100) | 0.338 |
| **$etCO_2$ (1st min of induction)** | 38.8 (2.7) (32-42) | 38.6 (3.3) (28-42) | 0.932 |
| **MAC (sf) (1st min of induction)** | 0.7 (0.1) (0.6-1) | 0.8 (0.1) (0.4-1.1) | 0.225 |
| **TV (1st min of induction)** | 270.9 (81) (136-429) | 237.9 (88.4) (113-512) | 0.110 |
| **PAP (1st min of induction)** | 12.2 (1.8) (9-16) | 11.8 (1.5) (10-16) | 0.490 |
| **HR (3rd min of LMA insertion)** | 94.5 (15.1) (68-121) | 112.1 (21.1) (72-150) | 0.180 |
| **MAP (3rd min of LMA insertion)** | 64.1 (7.2) (55-84) | 68.2 (8.4) (55-91) | 0.071 |
| **$SpO_2$ (3rd min of LMA insertion)** | 99.5 (0.5) (98-100) | 99.4 (0.9) (97-100) | 0.587 |
| **$etCO_2$ (3rd min of LMA insertion)** | 38.9 (2.7) (32-42) | 39.3 (2.3) (33-42) | 0.675 |
| **MAC-sf (3rd min of LMA insertion)** | 1.1 (0.1) (0.9-1.3) | 1.1 (0.1) (0.8-1.3) | 0.320 |
| **TV (3rd min of LMA insertion)** | 267.3 (84.9) (152-426) | 244.4 (85.6) (142-511) | 0.358 |
| **PAP (3rd min of LMA insertion)** | 12.3 (1.5) (10-16) | 12.1 (1.5) (10-16) | 0.608 |
| **HR (end of the surgery)** | 103 (10.9) (80-119) | 112.7 (21.1) (73-152) | 0.174 |
| **MAP (end of the surgery)** | 73.5 (16.1) (52-119) | 70.8 (13.2) (54-104) | 0.651 |
| **$SpO_2$ (end of the surgery)** | 97.8 (7) (65-100) | 99.2 (1) (97-100) | 0.821 |
| **$etCO_2$ (end of the surgery)** | 39.4 (3.5) (32-45) | 40.4 (3.7) (33-50) | 0.431 |
| **MAC-sf (end of the surgery)** | 1.1 (0.1) (0.7-1.4) | 1.1 (0.1) (0.7-1.3) | 0.116 |
| **TV (end of the surgery)** | 267.4 (97.9) (132-486) | 241.2 (90.6) (145-505) | 0.552 |
| **PAP (end of the surgery)** | 12.2 (1.9) (10-16) | 12.8 (1.4) (10-16) | 0.132 |

sf: sevoflurane. All continuous variables were presented as mean (standard deviation) (minimum-maximum).

IOP was measured at four points: at 1 minute following induction of anesthesia, at 3 minutes following insertion of LMA, and at the end of the surgery. All IOP values were between 5 and 25 mmHg. In both groups, the mean IOP values were between 10 and 13 mmHg at all four measurement times. When comparing the mean IOP values between the two groups, no significant differences were found ($P>0.05$) (Table 3).

Table 3: The comparison of IOP values between the groups

| IOP values (mmHg) | Propofol alone (n= 26) | Ketofol (n=19) | *P*-value |
|---|---|---|---|
| **IOP of right eye (baseline)** | 12.9 (3.5) (8-20) | 13.6 (5.1) (8-25) | 0.899 |
| **IOP of left eye (baseline)** | 12.1 (3.1) (6-17) | 13.3 (4.4) (8-24) | 0.494 |
| **IOP of right eye (1st min of induction)** | 11 (2.9) (6-18) | 12.1 (5.6) (5-25) | 0.945 |
| **IOP of left eye (1st min of induction)** | 10.7 (3.7) (5-24) | 11.6 (4.5) (6-22) | 0.676 |
| **IOP of right eye (3rd min of LMA insertion)** | 11 (3.5) (5-19) | 10.7 (4.7) (5-25) | 0.443 |
| **IOP of left eye (3rd min of LMA insertion)** | 10.4 (3.7) (5-21) | 10.7 (39) (5-20) | 0.905 |
| **IOP of right eye (end of the surgery)** | 11.1 (3.7) (5-21) | 10.4 (5.7) (5-25) | 0.187 |
| **IOP of left eye (end of the surgery)** | 10 (3.5) (6-19) | 10.1 (4.1) (4-19) | 0.990 |

IOP values were presented as mean (standard deviation) (minimum-maximum).

OCR was observed in 3 (15.8%) patients in the propofol alone group and in 5 (19.2) patients in the ketofol group. There was no significant difference in OCR between the two groups ($P=1.000$).

## Discussion

The present study showed that, compared with the use of propofol alone, the combination of ketamine and propofol did not cause any significant changes in IOP values in pediatric strabismus surgery. Moreover, the addition of ketamine to propofol reduced the frequency of OCR and provided a positive effect on basic hemodynamic parameters, although these differences were not statistically significant.

As is well known, keeping IOP within normal limits is of great importance in eye surgery, especially in pediatric cases. IOP can be affected by various factors such as tracheal intubation, MAP, hypercapnia, coughing, vomiting, and patient position on the operating table [12]. In fact, most anesthetic drugs can help to decrease IOP via mechanisms such as decreasing choroidal blood volume and the formation of aqueous humor [12,13]. On the other hand, there is a concern among anesthesiologists regarding the use of ketamine, especially in patients undergoing ophthalmic surgery, due to the general belief that this agent may increase IOP [10]. In daily practice, ketamine is considered a safe and reliable anesthetic agent, with limited cardiorespiratory suppression and few adverse outcomes. In addition, some previous studies have found that ketamine had no significant effect on IOP [14,15]. However, in a study conducted on 60 children with healthy eyes, the authors reported mild yet clinically important increases in IOP [1].

Although the criteria used to define normal IOP values vary between clinical studies, the largest work on this topic reported a mean value of 15.5 mmHg and a normal range of diurnal variations from 5 to 25 mmHg [16]. The baseline mean IOP value of 12–13 mmHg in our cohort was consistent with that value. It should be noted that mean IOP values at other times decreased according to the baseline mean IOP values in both the propofol alone group and the ketofol group. Statistically, the two groups were similar in terms of IOP values measured at the four time points during the procedure. These results are important because they demonstrate that ketofol anesthesia does not cause significant increases in IOP.

At this point, it is necessary to mention the doses of ketamine and propofol used in the ketofol protocol, as the primary goal of the present study was to obtain a smooth anesthesia induction with favorable hemodynamic parameters and to decrease adverse effects of both propofol and ketamine. It is known that cardiorespiratory suppression is related to the increased use of propofol, whereas insufficient doses may lead to difficult LMA insertion. As for ketamine, higher doses are associated with excessive secretions and inadequate suppression of airway reflexes. The doses of propofol and ketamine used in the ketofol protocol vary due to the type of procedure and anesthesia. Here, ketamine was used at the dose of 1.5 mg/kg since the study was conducted on patients under LMA anesthesia. In addition, we aimed to observe the effects of ketamine on IOP and hemodynamic parameters. Ketamine is a sympathomimetic and can theoretically elevate IOP as it causes an increase in HR and blood pressure. However, the present study showed that the combined use of ketamine and propofol at the mentioned doses did not affect IOP.

Hemodynamic indicators such as MAP and HR were found to be statistically similar between the ketofol and propofol alone groups, which is consistent with previous studies. In a study conducted by Yousef et al. [6], ketofol was found to improve hemodynamic stability compared to propofol in children. In another study, ketofol was found to be an alternative induction agent to propofol for LMA insertion in pediatric patients and to provide better hemodynamic parameters [9].

In the present study, another positive contribution of ketamine to hemodynamic stability was in terms of OCR occurrence. Although the difference was not statistically significant, the OCR rate was observed less in patients in the ketofol group. OCR is defined as at least a 20% decrease in HR or a new dysrhythmia after compression of eyeball or traction of extraocular muscles, and is often encountered during strabismus surgery, with a reported rate of up to 90% [17,18]. This complication is more often seen in children than in adults. Pediatric patients are also more susceptible to the dangerous effects of OCR, due to a higher dependence on HR to maintain cardiac output. In a study investigating the effect of four anesthetic regimens—propofol plus alfentanil, sevoflurane, ketamine plus midazolam, and halothane—on OCR during pediatric strabismus surgery, ketamine anesthesia was found to be associated with the least hemodynamic changes induced by OCR [19]. In another study conducted on patients undergoing strabismus surgery, it was shown that ketamine obtunds OCR and prevents the unwanted effects of dysrhythmias [20].

**Limitations**

This study had several limitations. The fact that it was conducted in a single center may limit the generalizability of the results. The relatively small number of patient groups may also lead to difficulty in interpreting subgroup findings. However, the prospective design, the lack of any premedication that may affect the hemodynamic parameters and OCR, and the standardized IOP measurement may be considered the strengths of the study.

**Conclusion**

The findings demonstrated that, compared to propofol anesthesia, ketofol anesthesia provided similar results in terms of IOP values, occurrence of OCR, and basic hemodynamic parameters such as MAP and HR. Therefore, ketofol can be considered a safe induction agent in pediatric patients undergoing strabismus surgery with LMA anesthesia.

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# Sarcopenia prevalence between obese and morbid obese patients in an obesity center

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**Ethics Committee Approval**
The study was approved by the Ethics committee of Dr Lutfi Kırdar Kartal City Hospital (Date: October 26, 2022, Number: 20221514/236/8). All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

**Conflict of Interest**
No conflict of interest was declared by the authors.

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## Abstract

**Background/Aim:** Sarcopenia and obesity are independent diseases that result in decreased muscle strength and function. Few studies have been conducted on the association of sarcopenia and obesity, especially in women. This study aims to measure the possibility of sarcopenic obesity in women with obesity.

**Methods:** Our study was organized using a prospective cross-sectional study in Turkey**.** A total of 135 volunteer were included in the study. Inclusion criteria required the patients to have a BMI >35 kg/m$^2$ or BMI >40 kg/m$^2$ and no current comorbid disease. The exclusion criteria included: age (<18 and >70 years were excluded), history of muscle disease, malignancy, psychiatric disorders such as bipolar disease and psychosis, malnutrition, and recent corticosteroid (CS) use (within the last three months). Probable sarcopenia is determined by low skeletal muscle strength, and confirmed sarcopenia is defined if there is both low skeletal muscle mass and low skeletal muscle quality. Muscle strength was measured with isometric dynamometry using the handgrip method. A six-minute walk test (6MWT), in which we measured walking speed, was performed to determine the physical performance of the patients. We adjusted appendicular skeletal muscle (ASM) using height squared (ASM/height$^2$) bioelectrical impedance analysis (BIA) to measure the muscle mass.

**Results:** Patients' mean age was 43 (11.4) (20-69) years. Of the total participants, 64.6% were in the age range of 40-59; 19.2% of patients were defined as possible sarcopenia; and 2.2% had confirmed sarcopenia. A total of 78.5% of patients did not meet any of the sarcopenia criteria. We determined that there was no difference in anthropometric measurements between sarcopenic and non-sarcopenic patients ($P$>0.05), except for waist and hip circumferences. However, we did observe a noteworthy distinction in waist and hip circumference measurements between the two groups, with sarcopenic patients exhibiting larger circumferences ($P$=0.05 and $P$=0.032, respectively). Our study revealed a significant disparity in the results of the six-minute walk test and handgrip strength values between sarcopenic and non-sarcopenic patients ($P$<0.001). Specifically, non-sarcopenic patients demonstrated higher values in both tests..

**Conclusion:** Obesity and sarcopenic obesity will continue to be a public health problem in the future among middle-aged women. It should be considered that the prevalence of decreased muscle strength was high in our study group, and physical performance decreased due to muscle strength. We concluded that as success in the six-minute walk test and handgrip values increased, the diagnosis of sarcopenia decreased, and each increase in platelet count increased the risk of sarcopenia in obese female patients.

**Keywords:** sarcopenia, sarcopenic obesity, middle aged women, EWGSOP

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## Introduction

When body fat increases and muscle mass and strength decrease with aging, sarcopenic obesity (SO) will occur. For a young healthy population, a muscle mass index, which is two standard deviations below the norm, was first named SO by Baumgartner [1]. SO leads to an increased risk of metabolic deterioration together with physical impairment rather than either sarcopenia or obesity alone [2].

Sarcopenia is associated with inflammatory, hormonal, and muscle cell alterations in response to aging and pathological factors, leading to muscle weakness, increased fat mass, and relatively decreased lean mass [3]. When the balance of muscle growth shifts toward muscle inhibitors, normal muscle quantity and function are disrupted. This is a mechanism in the pathogenesis of sarcopenia [4].

Low muscle mass is triggered together with many comorbid conditions [4,5]. In women with obesity, when fat tissue increases and muscle mass decreases with aging, it can cause resistance to insulin. If those women have hypertension, hyperlipidemia and type 2 diabetes mellitus (T2DM), metabolic syndrome will appear [6]. Defective lipolysis occurs in skeletal muscle due to insulin resistance in obese and T2DM individuals. Fatty acids from triglyceride storage are lipolyzed via adipose triglyceride lipase and hormone sensitive lipase [7,8]. In addition, obesity increases the risks of arthritis, some types of cancers, and sleep apnea [9]. Sarcopenia can develop in young adults due to factors including autoimmune disorders, inflammatory diseases, and endocrine dysfunctions [10].

Our aim is to examine the sarcopenic obesity prevalence between the group of Turkish obese and morbidly obese patients in this study.

## Materials and methods

### Patients

This cross-sectional study registered patients who were followed up with a diagnosis of obesity in the Obesity Center outpatient polyclinic of our hospital from April 2022 until January 2023. The study was approved by the Ethics Committee of Dr. Lutfı Kırdar Kartal City Hospital in Turkey (Decision number: 202215l4/236/8, Date: October 26, 2022). Patients were classified into two groups: women 20 to 39 years old and women 40 to 69 years old). The study was continued if the patients' values were BMI >35 kg/m2 or BMI >40 kg/m2 and they had no comorbid diseases.

The exclusion criteria included: age (<18 and >70 years were excluded), history of muscle disease, malignancy, psychiatric disorders such as bipolar disease and psychosis, malnutrition, and recent corticosteroid (CS) use (within the last three months). We obtained a written informed consent form from the patients. The management of the study adheres to the World Medical Association Declaration of Helsinki.

### Skeletal muscle mass/quantity/quality assessment

Weight units are given in kilograms (kg), height is measured in meters (m), and BMI is measured using the formula kg/m2. Tanita MC-580 body composition analysis (TANITA, MC-580, Japan) was used in the anthropometric measurements. We adjusted the appendicular skeletal muscle (ASM) using height squared (ASM/height$^2$), because muscle mass is related to body size [11,12]. In our study, we used the cutoff values of 9.2 kg/m2 for males and 7.4 kg/m2 for females for ASM/h2, defined by Bahat et al. [13] for the Turkish population.

According to the revised definition of the European Working Group on Sarcopenia in Older People (EWGSOP), probable sarcopenia is determined in the presence of low skeletal muscle strength. Confirmed sarcopenia is concluded in the presence of both low skeletal muscle mass and low skeletal muscle quality. Finally, in the presence of low physical performance in addition to these two findings, sarcopenia is defined to be “severe”[11].

### Skeletal muscle strength assessment

We used the handgrip strength (HGS) test for muscle strength assessment [14]. A strain-gauged dynamometer (TKK 5001, Takei Scientific Instruments, Tokyo, Japan) was used to measure HGS (kg). During measurements, the subject was in a standing position with the arms parallel to the body but without contact with the body. Patients repeated grip force at least three times with both their left and right hands and the maximum value was recorded. In the study, the cutoff reference values defined by Bahat et al. [13] for low HGS, <32 kg in male and <22 kg in female, were used. Low HGS values define low muscle strength.

### Physical performance

The six-minute walk speed test (6MWST), a widely used assessment for measuring walking speed, was used to measure physical performance. Patients were asked to walk at a normal pace without interruption on a long corridor with flat, hard and smooth floors for six minutes. Walking speed was calculated using distance in meters and time in seconds for each participant (m/s). Low gait speed cutoff value was defined as <0.8 m/s [11].

### Statistical analysis

Analyses were performed with the statistical package software, SPSS (version 23.0, IBM Corp. Armonk, NY). Frequency and percentage were given for categorical data; mean (standard deviation) or median, minimum and maximum descriptive values were given for continuous data. For comparisons between groups, the independent samples t-test or the Mann-Whitney U test was used for the two groups, and chi-square or Fisher's exact test was used to evaluate categorical variables. Logistic regression analysis was used to examine the risk factors affecting the development of sarcopenia. Variables with a *P*-value less than 0.10 in univariate analysis were included in the logistic regression analysis. The results were considered statistically significant when the *P*-value was 0.05 or less.

## Results

The study was conducted with 135 female patients who met the criteria and whose mean age (SD) was 43 (11.4) (20-69) years. Of the patients, 64.6% were in the age range from 40 to 59. All the laboratory and clinical evaluations and demographic values are shown in Table 1 and Table 2.

One hundred six (78.5%) patients did not meet any of the sarcopenia criteria (no sarcopenia); 26 (19.2%) patients were determined to have possible sarcopenia, and 3 (2.2%) patients had confirmed sarcopenia. None of the patients were diagnosed with severe sarcopenia. We used the Bahat’s study cutoff values

Table 1: Demographic and clinical findings of the patients

| Variables | Total (n=135) | | Non-Sarcopenic (n=106) | | Sarcopenic (n=29) | | *P*-value |
|---|---|---|---|---|---|---|---|
| | Mean (SD) | Median (Min-Max) | Mean (SD) | Median (Min-Max) | Mean (SD) | Median (Min-Max) | |
| **Age (years)** | 43.3 (11.4) | 43 (18-69) | 42.6 (11) | 42 (22-69) | 46.2 (12.9) | 48 (18-66) | 0.146 |
| **Height (cm)** | 159.6 (6.8) | 159 (145-180) | 159.6 (6.9) | 158 (145-180) | 159.7 (6.5) | 160 (147-168) | 0.537 |
| **Waist circumference (cm)** | 114.5 (13.4) | **112 (82-158)** | **113.4 (13.6)** | **110.5 (82-158)** | **118.3 (12.4)** | **114 (99-140)** | **0.050** |
| **Hip circumference (cm)** | 126.1 (11.8) | **125 (101-155)** | **125.1 (11.8)** | **123 (101-155)** | **129.8 (11.1)** | **130 (110-150)** | **0.036** |
| **Arm circumference (cm)** | 35.4 (4.7) | 34.8 (27-55) | 35.1 (4.5) | 34 (27-50) | 36.5 (5.2) | 36 (31-55) | 0.178 |
| **Weight (kg)** | 102.4 (18.4) | 99 (70.6-157) | 101 (18.2) | 97.3 (70.6-157) | 107.6 (18.8) | 105.7 (75.5-153.1) | 0.089 |
| **BMI (kg/m$^2$)** | 40.2 (6.5) | 39.3 (30-58.9) | 39.6 (6.3) | 38.2 (30-58.9) | 42.3 (7) | 42.9 (30.3-55.8) | 0.055 |
| **PBF (%)** | 42.1 (4.2) | 41.6 (32.9-54.5) | 41.7 (4.1) | 41.3 (32.9-53.7) | 43.4 (4.7) | 42 (35.7-54.5) | 0.060 |
| **SLM (kg)** | 53.6 (7.7) | 52.5 (39-75.2) | 53.1 (7.4) | 52.3 (41.5-75.2) | 55.4 (8.4) | 54.6 (39-72.6) | 0.153 |
| **ASMM (kg)** | 26.6 (4.6) | 25.9 (17.4-39.1) | 26.3 (4.4) | 25.7 (17.4-38.5) | 27.8 (5.2) | 27.1 (18.8-39.1) | 0.194 |
| **ASMM/H$^2$ (kg/cm$^2$)** | 10.4 (1.7) | 10.1 (3.5-15) | 10.3 (1.5) | 10.1 (6.9-15) | 10.8 (2.4) | 10.7 (3.5-15) | 0.307 |
| **6 min walking test (m)** | 422.1 (61.6) | 416 (275-580) | **441.1 (53.5)** | **435.8 (300-580)** | **352.6 (32.2)** | **350 (275-425)** | **<0.001** |
| **Handgrip (kg)** | 22.5 (5.2) | 21.9 (11.8-38.9) | **23.6 (5.2)** | **23.2 (13.1-38.9)** | **18.6 (2.5)** | **19.3 (11.8-21.8)** | **<0.001** |

BMI: body mass index, ASMM: appendicular skeletal muscle mass, ASMM/h$^2$: appendicular skeletal muscle mass/height$^2$, SLM: smooth lean mass. Values are given as mean and median (range). The Mann-Whitney U test was performed. *Chi-squared test was used.

Table 2: Laboratory findings of patients

| Variables | Total (n=135) | | Non-Sarcopenic (n=106) | | Sarcopenic (n=29) | | *P*-value |
|---|---|---|---|---|---|---|---|
| | Mean (SD) | Median (Min-Max) | Mean (SD) | Median (Min-Max) | Mean (SD) | Median (Min-Max) | |
| **Glucose (mg/dl)** | 103.6 (21.9) | 100 (68-243) | 103.6 (22.6) | 100 (68-243) | 103.4 (19.6) | 97 (68-166) | 0.849 |
| **Insulin (IU)** | 18.6 (11.6) | 15.7 (3.5-67.9) | 18.5 (10.5) | 15.9 (3.5-61.4) | 19.1 (15.3) | 15 (5.3-67.9) | 0.493 |
| **TC (mg/dl)** | 207.9 (39.6) | 207 (120-314) | 206.5 (38.7) | 206.5 (120-305) | 213 (42.9) | 211 (140-314) | 0.439 |
| **HDL (mg/dl)** | 50.5 (10.5) | 50 (27-88) | 51 (10.8) | 50 (27-88) | 48.6 (9) | 47 (30-75) | 0.293 |
| **TG (mg/dl)** | 138.9 (81.2) | 130 (39-831) | 141.8 (88.3) | 131.5 (39-831) | 128.2 (47.2) | 126 (43-232) | 0.688 |
| **LDL (mg/dl)** | 132.2 (35.6) | 128 (64-233) | 129.1 (34.4) | 123.5 (64-229) | 143.6 (38.5) | 145 (78-233) | 0.053 |
| **TSH (mIU/L)** | 2.5 (1.6) | 2.2 (0.3-10.5) | 2.5 (1.7) | 2.2 (0.3-10.5) | 2.6 (1.5) | 2.4 (0.8-7.9) | 0.491 |
| **HOMA** | 4.8 (3.1) | 4.1 (0.9-19.3) | 4.6 (2.6) | 3.9 (1-14.2) | 5.6 (4.4) | 4.8 (0.9-19.3) | 0.492 |
| **Iron (mcg/dl)** | 65.5 (24.1) | 61 (28-139) | 66.7 (24.7) | 62 (28-139) | 61.2 (21.8) | 58 (29-107) | 0.309 |
| **Ferritin (mcg/L)** | 36 (31) | 26.8 (3-146) | 35 (31) | 25.4 (3-146) | 39.7 (31.2) | 29.3 (3.6-111) | 0.377 |
| **25OHD3 (ng/ml)** | 16.5 (7.6) | 15.4 (4.1-49.1) | 16.3 (7.2) | 15.1 (4.1-37.7) | 17.3 (9) | 16.1 (4.5-49.1) | 0.734 |
| **Hgb (g/dL)** | 13 (1.1) | 13.1 (9.8-16.3) | 13 (1.1) | 13.1 (9.8-15.2) | 12.9 (1.2) | 13 (10.2-16.3) | 0.453 |
| **HTC (%)** | 39.1 (4.6) | 39.4 (9.2-49.5) | 39.4 (3.9) | 39.5 (15.3-46.6) | 38.1 (6.4) | 39.2 (9.2-49.5) | 0.168 |
| **PLT (10$^3$/ul)** | 294.2 (90.5) | 273 (102-774) | 285.2 (80.2) | 270 (102-524) | 326.3 (116.3) | 300 (191-774) | 0.056 |
| **HBA1C (mmol/L)** | 6 (0.9) | 5.8 (4.9-9.9) | 5.9 (0.8) | 5.8 (5-9.9) | 6.2 (1) | 6 (4.9-8.6) | 0.157 |
| **Vitamin B12 (ng/mmol)** | 243.6 (116.4) | 221.5 (85-944) | 242.3 (116.7) | 219 (85-944) | 248.5 (117.1) | 224 (133-764) | 0.658 |

TC: Total cholesterol, HDL: High density cholesterol, TG: triglycerides, LDL: low density lipoprotein, TSH: thyroid stimulant hormone, HOMA: homeostasis model assessment, HTC: hematocrit, PLT: platelet, HBA1C: glycosylated hemoglobin

adapted from EWGSOP for the Turkish people [15]. In this diagnostic method, low grip strength with certain cutoff points (handgrip 22 kg for female) defines possible sarcopenia. Sarcopenia was confirmed if the low muscle quantity (SMMI cutoff value 7.2 kg/m$^2$) was combined with the first criteria [13].

It was identified that there were no significant differences in anthropometric measurements between patients with sarcopenia and those without sarcopenia ($P$>0.05 for each), except for waist and hip circumferences. However, we did observe a notable contrast in waist and hip circumference measurements between the two groups, with sarcopenic patients showing larger circumferences ($P$=0.05 and $P$=0.032, respectively). Furthermore, our study unveiled a significant difference in the results of the six-minute walk test and handgrip strength values between sarcopenic and non-sarcopenic patients ($P$<0.001). Specifically, non-sarcopenic patients exhibited higher values in both tests.

The distribution of comorbidities detected along with the diagnosis of sarcopenia is given in Table 3. According to the table, it was determined that there was no relationship in the groups in terms of comorbidity distributions and additional diseases ($P$=0.316).

Table 3: The distribution of comorbid diseases in patients who diagnosed sarcopenia or not

| Variables | Total (n=135) | Non-Sarcopenic (n=106) | Sarcopenic (n=29) | *P*-value |
|---|---|---|---|---|
| | n (%) | n (%) | n (%) | |
| **Comorbidity** | | | | 0.316 |
| No | 84 (62.2) | 69 (65.1) | 15 (51.7) | |
| 1 comorbidity | 33 (24.4) | 25 (23.6) | 8 (27.6) | |
| ≥2 comorbidity | 18 (13.3) | 12 (11.3) | 6 (20.7) | |
| **HT** | 19 (14.1) | **11 (10.4)** | **8 (27.6)** | **0.031** |
| **DM** | 20 (14.8) | 14 (13.2) | 6 (20.7) | 0.376 |
| **HL** | 1 (0.7) | 0 (0) | 1 (3.4) | 0.215 |
| **Hashimoto** | 2 (1.5) | 2 (1.9) | 0 (0) | 1.000 |
| **Thyroid** | 11 (8.1) | 9 (8.5) | 2 (6.9) | 1.000 |
| **Insulin resistance** | 4 (3) | 3 (2.8) | 1 (3.4) | 1.000 |
| **PCOS** | 1 (0.7) | 1 (0.9) | 0 (0) | 1.000 |
| **Hyperprolactinemia** | 1 (0.7) | 0 (0) | 1 (3.4) | 0.215 |
| **Pituitary deficiency** | 1 (0.7) | 1 (0.9) | 0 (0) | 1.000 |
| **GER** | 2 (1.5) | 2 (1.9) | 0 (0) | 1.000 |
| **Rheumatologic diseases** | 1 (0.7) | 1 (0.9) | 0 (0) | 1.000 |
| **CAD** | 1 (0.7) | 0 (0) | 1 (3.4) | 0.215 |
| **Fibromyalgia/Osteoporosis** | 2 (1.5) | 1 (0.9) | 1 (3.4) | 0.385 |
| **Epilepsy** | 2 (1.5) | 2 (1.9) | 0 (0) | 1.000 |
| **Psoriasis** | 1 (0.7) | 1 (0.9) | 0 (0) | 1.000 |
| **Asthma** | 1 (0.7) | 1 (0.9) | 0 (0) | 1.000 |
| **COPD** | 2 (1.5) | 1 (0.9) | 1 (3.4) | 0.385 |

HT: hypertension, DM: diabetes mellitus, HL: hyperlipidemia, PCOS: polycystic over disease, GER: gastroesophageal reflux, CAD: coronary artery disease, COAH: chronic obstructive pulmonary disease

Risk factors affecting the development of sarcopenia in patients are provided in Table 4. Among all variables, the six-minute walk test, handgrip, and PLT values included in the model in the univariate analysis, affected the development of sarcopenia, respectively ($P$<0.001, $P$<0.001, $P$=0.040). It was determined that the diagnosis of sarcopenia decreased as the success of the patients in the six-minute walk test and the handgrip values increased, and each increase in the PLT value increased the risk of sarcopenia. Variants, which had significant changes in univariate analysis, were re-evaluated in multivariate analysis. We observed a difference in the six-minute walk test and handgrip values ($P$<0.001). It was found that an increase in

the six-minute walk test in the patients decreased the risk of sarcopenia 0.94 times, and an increase in the handgrip value decreased the risk of sarcopenia 0.63 times.

Table 4: Risk factors in the development of sarcopenia

| Variables | Univariate | | Multivariate | |
|---|---|---|---|---|
| | Odds ratio (95% GA) | *P*-value | Odds ratio (95% GA) | *P*-value |
| **Waist circumference (cm)** | 1.03 (1.00-1.06) | 0.089 | | |
| **Hip circumference (cm)** | 1.04 (1.00-1.07) | 0.057 | | |
| **Weight (kg)** | 1.02 (1.00-1.04) | 0.089 | | |
| **BMI (kg/m$^2$)** | 1.06 (1.00-1.13) | 0.052 | | |
| **PBF (%)** | 1.09 (1.00-1.21) | 0.063 | | |
| **6 min walking test (m)** | **0.95 (0.93-0.97)** | **<0.001** | **0.94 (0.91-0.96)** | **<0.001** |
| **Handgrip (kg)** | **0.76 (0.67-0.87)** | **<0.001** | **0.63 (0.49-0.80)** | **<0.001** |
| **LDL (mg/dl)** | 1.01 (1.00-1.02) | 0.056 | | |
| **PLT (10$^3$/ul)** | **1.01 (1.00-1.02)** | **0.040** | 1.00 (0.99-1.01) | 0.721 |
| **DM** | 1.71 (0.59-4.95) | 0.319 | | |

BMI: body mass index, PBF: percent body fat, LDL: low density lipoprotein, PLT: platelet, DM: diabetes mellitus

## Discussion

Our aim in this study was to examine sarcopenic obesity among a group of obese and morbidly obese Turkish patients. Our population consisted of female patients, and sarcopenia was detected in 21.5%. According to BMI values, sarcopenia was detected in six patients with a BMI of 30-35; five patients with a BMI of 35-40, and in 18 patients with a BMI >40. This shows that sarcopenia due to low muscle strength increases with age in female patients. In addition, there was a difference in waist and hip circumference, the six-minute walk test and handgrip values among all patients.

Low muscle strength in the elderly has been determined by the European Working Group on Sarcopenia as the main criterion for investigating sarcopenia. If the grip strength is low, cause of death due to functional limitation risks will be increased [10]. Sarcopenia diagnosis is confirmed if muscle mass and quality are both low. According to these criteria, in our study probable sarcopenia was detected in 26 women and the confirmed sarcopenia was detected in 3 patients. Additionally, in this study, skeletal muscle mass was adjusted for height$^2$. Although this is the most widely used method in sarcopenia definitions, it has been shown to fail in obese individuals with sarcopenia [16,17].

Kemmler et al. [17] studied sarcopenic obesity and sarcopenia in a group of German females over 70 years of age, and their results were almost identical to the prevalence rate of sarcopenia due to EWGSOP (4.9% versus 4.5%). Additionally, Beadurt et al. [18] also applied the same EWGSOP sarcopenia criteria to a CDW (cohort of community-dwelling) cohort of young multimorbid Belgian men (n=157) and identified much higher prevalence rates. The Korean study by Kim et al. [19] also found that the SO prevalence according to different definition indices ranged from 0.8 to 11.8% in women aged 40 to 59 years.

According to the World Health Organization's definition, the obesity prevalence in older adults in the United States is reported to be nearly 37.9% [20]. The mean age of our obese patients was 46.2 (12.9), and their distribution was: BMI 30-35 20.7%, BMI 35-40 17.2%, BMI >40 62.1%.

In this study, we tried to examine the possibility of sarcopenia in young and middle-aged women and the evaluation criteria in severely obese patients in Turkey. SO figures were low due to the age range of the selected study group. It is known that among European countries, the prevalence of obesity in the elderly has increased the most in Germany [21].

The reduced energy expenditure as a result of decreased muscle mass and physical activity level causes visceral fat and general body fat, which is especially significant. Loss of skeletal muscle, which is the largest target tissue sensitive to insulin, together with visceral fat, which appears as fatty liver, causes insulin resistance. This results in the onset of metabolic syndrome [22].

The variation in physical performance of sarcopenic and non-sarcopenic obese women is due to differences in muscle mass. As emphasized by Newman et al., we believe that it is important to identify obese individuals who do not appear sarcopenic but have decreased muscle mass masked by obesity [23].

In the comorbidity evaluation of our data, the rate of diabetes was higher in individuals with sarcopenia and there was no statistical relationship between the two groups in all diseases except DM as shown in Table 3. Sarcopenia prevalence was substantially higher in non-obese patients (48.1% vs 29.3%), and obesity and sarcopenic obesity were more common in patients with DM in a recent study conducted among nursing home patients [24].

The current study observed that the increase in the six-minute walking test and in the handgrip value decreased the risk of sarcopenia 0.94 and 0.67 times in the patients, respectively. In addition, Silva et al. indicated that sarcopenia prevalence varied between 11.1% and 13.9% due to low level of muscle quantity and muscle quality [25].

Our results align with similar studies. Our ASM (26.6 [4.6] kg) is comparable to Silva et al. [26] who determined no statistical difference in the adequacy of the total ASM (mean 24.9 [4.7] kg) of all subjects when comparing BMI degrees or age groups. When ASM (kg) was analyzed, it was seen that as BMI increased, the ASM(kg) value also increased a bit.

### Limitations

There are some limitations in our research. First, standard protocols have not been established for the diagnosis of SO. Our study was a single center study and the low patient counts in a number of comparisons considerably limit the generalizability of our findings. Further studies with a larger number of patients are needed to compare sarcopenic obesity depending on body composition. The absence of a control group is another limitation. In addition, our study group age was a bit younger for the determination of sarcopenic obesity.

The strength of this study is that sarcopenic obesity is a condition rarely studied in middle-aged women. Another strength of this study is that the methods we used to measure patients' physical performance and body composition were valid, inexpensive, and noninvasive.

### Conclusion

In our study, young and middle-aged obese female patients were evaluated. We concluded that as the success in the six-minute walk test and handgrip values increased, the diagnosis of sarcopenia decreased and each increase in PLT value increased the risk of sarcopenia. If patients have sufficient muscle strength, the prevalence of sarcopenia will decrease.

Low muscle mass and physical activity level reduce total energy expenditure, which leads to the accumulation of visceral fat and obesity. Among middle-aged women, obesity and sarcopenic obesity (SO) will be a public health problem in the future. Sarcopenic obesity has been studied mostly in older adults. More research is needed regarding the prevalence and reasons for SO among middle-aged female patients.

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# How to manage a congenital heart defect in a patient with thrombocytopenia-absent radius syndrome?

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**Abstract**

Ventricular septal defect (VSD) can be repaired using cardiopulmonary bypass, resulting in a favorable postoperative outcome with minimal bleeding. Thrombocytopenia-absent radius (TAR) syndrome is rare, occurring in approximately 0.42 out of 100,000 live births. This syndrome is characterized by hypo-megakaryocytic thrombocytopenia and bilateral absent radii. TAR syndrome can be life-threatening within the first 14 months of life due to severe bleeding. In this report, we present the case of a 4-month-old male patient diagnosed with both VSD and TAR syndrome. We describe the surgical management of the VSD as well as the perioperative treatment for hemorrhagic diathesis.

**Keywords:** thrombocytopenia-absent radius syndrome, TAR syndrome, ventricular septal defect, infant

## Introduction

Thrombocytopenia-absent radius (TAR) syndrome is characterized by thrombocytopenia, along with specific skeletal abnormalities primarily affecting both upper limbs. It was initially described by Hall et al. [1] in 1969 as a condition characterized by thrombocytopenia, bilateral absence of the radius bones, and the presence of both thumbs. This exceedingly rare syndrome is associated with a high risk of severe hemorrhage due to significant thrombocytopenia, with a platelet count of less than 50 × 109/L during the first year of life. Here, we present an uncommon association between TAR syndrome and congenital heart surgery, detailing our perioperative management of the patient.

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**Informed Consent**
The authors stated that the written consent was obtained from the parents of the patient presented with images in the study.

**Conflict of Interest**
No conflict of interest was declared by the authors.

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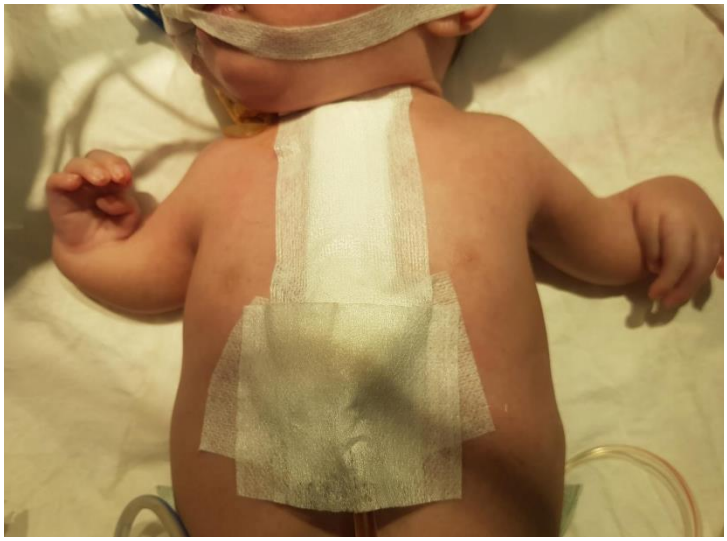
## Case presentation

A 4-month-old male patient weighing 4.3 kg, previously diagnosed with ventricular septal defect (VSD) and TAR syndrome, was referred to our clinic due to a cardiac murmur. During the physical examination, we observed syndactyly of digits 1–4 on both hands and low-set ears bilaterally. Furthermore, bilateral absence of the radii was noted, along with varus angulation of the metacarpal bones in relation to the ulna and diminutive second and third metacarpal bones on the left hand (Figure 1). The lower extremities appeared normal. A cardiac examination revealed a grade 2/6 murmur on the left parasternal side. The VSD was classified as perimembranous, and no additional heart defects were detected via a two-dimensional echocardiogram. The patient had previously received a TAR syndrome diagnosis based on physical findings and had been hospitalized every two weeks since birth for thrombocyte replacement due to a platelet count of less than $50 \times 10^9$/L. Upon admission, laboratory results showed a platelet count of 48,000/mm$^3$ and a hemoglobin level of 9 g/dL. The size of the platelets appeared normal. Genetic analysis confirmed the diagnosis of TAR syndrome, revealing an interstitial microdeletion in 1q21.1 and a hypomorphic *RBM8A* allele. Informed consent was obtained from the patient's relatives.

Figure 1: Specific deformities on both hand and absence of radii in TAR syndrome.



VSD repair using cardiopulmonary bypass (CPB) is typically performed at the age of 4 months. However, considering the patient's fluctuating platelet count, we anticipated challenges in managing perioperative hemorrhage. Therefore, we opted to initially perform pulmonary banding to reduce pulmonary blood flow and delay the VSD repair until the platelet count increased. One hour before the operation, the patient received a 15 mL/kg platelet suspension through a peripheral catheter, resulting in a platelet count exceeding $100 \times 10^9$/L. Arterial and central venous catheters were inserted, and the procedure was performed via sternotomy following standard protocols. Postoperatively, an additional 15 mL/kg platelet suspension was administered at the 5th hour. The patient received a low dose of an inotropic agent and exhibited postoperative drainage of 30 mL on the first day. By the second postoperative day, the platelet count reached $65 \times 10^9$/L, and the total drainage was 40 cc. Consequently, the mediastinal tube was removed on this day. Routine thrombocyte infusions were administered to mitigate bleeding complications, and the patient was successfully extubated on the third postoperative day, with a platelet count consistently above $50 \times 10^9$/L. Steroids were not required to improve the platelet count. Unfortunately, the patient developed catheter-related sepsis on the fifth postoperative day, leading to septicemia and subsequent demise on the eighth postoperative day.

## Discussion

TAR syndrome is an extremely rare genetic disorder characterized by hypo-megakaryocytic thrombocytopenia and radial aplasia or hypoplasia in both thumbs, as described by Hall et al. [1]. This condition impairs the maturation of megakaryocyte progenitor cells in the bone marrow, resulting in hypo-megakaryocytic thrombocytopenia. Various biological and molecular studies conducted on TAR syndrome have indicated elevated levels of the cytokine thrombopoietin (TPO) and suboptimal differentiation of megakaryocyte progenitor cells in response to TPO *in vitro*, suggesting a potential defect in the TPO signaling pathway [2]. Most TAR syndrome patients develop thrombocytopenia within the first week of life, with approximately 95% of cases being diagnosed within the initial 4 months of life and exhibiting platelet counts typically below $50 \times 10^9$/L [2]. Initially, thrombocytopenia tends to be severe (less than $30 \times 10^9$/L), but the platelet count gradually increases over time and reaches near-normal levels by 1–2 years of age [3]. The severity of thrombocytopenia correlates with symptoms such as petechial rash and bleeding. Severe spontaneous hemorrhagic events, most commonly in the brain, gastrointestinal tract, or other organs, are the leading cause of mortality in these patients. However, major hemorrhages are predominantly observed during the first 2 years of life [4].

Patients diagnosed with TAR syndrome may exhibit facial dysmorphism, macrocephaly, renal malformations, and skeletal abnormalities. It is important to investigate the presence of additional cardiac defects, such as atrial septal defect, VSD, tetralogy of Fallot, patent ductus arteriosus, and atrioventricular septal defect [4,5]. While there are limited case reports discussing TAR syndrome in conjunction with heart surgery, Kumar et al. [4] described a rare association between TAR syndrome and tetralogy of Fallot. They reported a successful surgical repair in a 3-month-old male patient in their study. However, the authors did not provide detailed information regarding postoperative drainage in the case report.

### Conclusion

During heart surgery, the use of cardiopulmonary bypass (CPB) and heparin can lead to thrombocyte dysfunction and increased bleeding tendency in the general population. However, there is limited information in the literature regarding managing congenital heart defects in patients with TAR syndrome [4]. Given our understanding of TAR syndrome and its associated complications, we anticipated that performing VSD repair under CPB could result in higher-than-expected postoperative bleeding due to thrombocyte dysfunction. Therefore, we opted for a palliative procedure, specifically pulmonary banding, which involves a sternotomy but not a complete repair under CPB with heparin. This approach was chosen to minimize bleeding risks. Despite the unfortunate outcome of sepsis in our patient, we believe that adopting a conservative procedure offers maximum

safety in terms of bleeding for patients with congenital heart defects and TAR syndrome who undergo surgery within their first 1.5 years of life.

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