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ARAŞTIRMA / RESEARCH

Prognostic significance of the optic nerve sheath diameter in acute respiratory failure

Akut solunum yetmezliğinde optik sinir kılıf çapının prognostik önemi

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Abstract

Purpose: The primary aim of this study was to determine the role of the optic nerve sheath diameter (ONSD) measured using ultrasonography in the differentiation of hypoxemic respiratory failure and hypercarbic respiratory failure. The secondary aim was to determine the value of ONSD in predicting the clinical outcome in hypercarbic patients.

Materials and Methods: Non-traumatic adult patients presenting with dyspnea were included in this study. Arterial blood gas and concurrent ONSD measurements were obtained from all patients included in the study during presentation and at the second hour of treatment. The patients were divided into two groups - hypoxic and hypercarbic respiratory failure. An equivalent number of healthy volunteers were enrolled as the control group.

Result: Mean ONSD values were 3.8±0.2 mm in the 24 patients with type 1 respiratory failure, 5.2 ± 0.5 mm in the 25 patients with type 2 respiratory failure, and 3.9±0.3 mm in the healthy controls. The ONSD of the hypercarbic patients was statistically significantly greater than that of the hypoxic patients and healthy volunteers. The ONSD of the hypercarbic patients was 5.2±0.5mm at baseline and 4.4±0.6 mm after treatment, the difference being statistically significant. However, the difference in ONSD was not statistically significant in predicting hospitalization and mortality.

Conclusion: Sonographically measured ONSD may be an effective parameter in the differential diagnosis of type 1 and 2 respiratory failure. ONSD values changed significantly with treatment in patients with type 2 respiratory failure.

Keywords:. Hypercarbia, ocular ultrasonography, optic nerve sheath diameter, emergency medicine

Öz

Amaç: Bu çalışmanın birincil amacı, hipoksemik solunum yetmezliği ve hiperkarbik solunum yetmezliği ayrımında ultrasonografi kullanılarak ölçülen ONSD'nin rolünü belirlemekti. Çalışmanın ikincil amacı, hiperkarbik hastaların klinik sonuçlarını tahmin etmede ONSD'nin değerini belirlemektir.

Gereç ve Yöntem: Bu çalışmaya dispne ile başvuran travmatik olmayan yetişkin hastalar dahil edildi. Çalışmaya başvuran tüm hastalardan başvuru sırasında ve tedavinin ikinci saatinde arter kan gazı ve eş zamanlı ONSD ölçümleri alındı. Hastalar hipoksik ve hiperkarbik solunum yetmezliği olmak üzere iki gruba ayrıldı. Hasta sayısı kadar sağlıklı gönüllüler çalışmaya dahil edildi.

Bulgular: Tip 1 solunum yetmezliği olan 24 hastada ortalama ONSD değerleri 3,8±0,2 mm, tip 2 solunum yetmezliği olan 25 hastada 5,2 ± 0,5 mm ve sağlıklı kontrollerde 3,9±0,3 mm idi. Hiperkarbik hastaların ONSD'si, hipoksik hastaların ve sağlıklı gönüllülerinkinden istatistiksel olarak anlamlı derecede daha yüksekti. Hiperkarbik hastaların ONSD'leri tedavi başlangıcında 5,2±0,5 mm ve tedavi sonrasında 4,4±0,6 mm idi ve aralarında istatistiksel olarak anlamlı fark vardı. Ancak ONSD farkı, hastaneye yatış ve mortaliteyi öngörmede istatistiksel olarak anlamlı değildi.

Sonuç: Sonografik olarak ölçülen ONSD, tip 1 ve tip 2 solunum yetmezliğinin ayırıcı tanısında etkili bir parametre olabilir. Tip 2 solunum yetmezliği olan hastalarda tedavi ile ONSD değerleri önemli ölçüde değişmiştir.

Anahtar kelimeler: Hiperkarbi, okuler usg, optik sinir kılıf çapı, acil tıp.

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INTRODUCTION

Respiratory failure derives from problems occurring in the oxygenation and/or carbon dioxide (CO2) elimination functions of the respiratory system. Although hypoxia is a parameter that varies depending on age, atmospheric pressure, and height above sea level, it is generally defined as a decrease in PaO2 below 80 mm Hg for individuals under the age of 65. A value below 60 mmHg is defined as type 1 (hypoxemic) respiratory failure. PaCO2 values in these patients are either normal or low[1,2,3,4]. Type 2 (hypercarbic) respiratory failure is defined as PaCO2 exceeding 45 mmHg and is most frequently seen in acute or chronic obstructive airway diseases. Hypoxemia in room air is frequently determined in patients with type 2 respiratory failure[1,2,3,4].

The clinical characteristics of type 2 respiratory failure may vary depending on the level of increase in CO2. Gradually developing mild-moderate hypercarbia can result in anxiety, dyspnea, malaise, headache, somnolence and confusion. If CO2 levels continue to rise, a clinical manifestation capable of progressing to coma and seizures develops[2,4,5]. An increase in CO2, a powerful cerebral vasodilator, results in an increase in intracranial pressure (ICP)[6,7]. Papilledema scanning with fundoscopic examination, and advanced imaging techniques such as cerebral computed tomography (CT) or magnetic resonance imaging (MRI) and direct intrathecal ICP monitoring can be employed in the detection of increased ICP[8]. However, several recent studies have suggested that the optic nerve sheath diameter (ONSD) can be successfully used to reveal ICP in an indirect manner[9,10].

The optic nerve develops from the growth of the diencephalon during embryogenesis[11]. It is an extension of the central nervous system, and is surrounded by a dural sheath containing a subarachnoid space around cerebrospinal fluid (CSF)[12]. In case of an increase in ICP for any reason, the CSF is displaced toward the subarachnoid space surrounding the optic nerve. The resulting increase in pressure in the optic nerve leads to expansion of the dural sheath and an increase in ONSD[11,12]. Numerous previous studies have shown that ONSD increases in conditions resulting in a rise in ICP, such as trauma and mass effect, and that sonographic ONSD measurement can be used as a repeatable, rapid, and non-invasive diagnostic tool in detecting and monitoring increases in ICP[9,10,13]. However, there has been no previous investigation of the success of ONSD ultrasound in evaluating increases in ICP developing in hypercarbic respiratory failure, in the differential diagnosis of the type of respiratory failure, and in predicting the clinical outcomes of patients with hypercarbic respiratory failure. The primary aim of this study was to determine the value of ONSD measurement in the differential diagnosis of type 1 and 2 respiratory failure. The secondary objective was to determine the value of ONSD in predicting hospitalization and in-hospital mortality in patients with type 2 respiratory failure.

MATERIALS AND METHODS

This prospective observational study was performed at the İzmir Katip Çelebi University Atatürk Education and Research Hospital in Turkey. Patient records in this center are transferred to an electronic environment and archived. Access is possible when required. Approval was granted by the İzmir Katip Çelebi University Non-Invasive Clinical Research Ethics Committee before the study commenced (protocol 2021-GOKAE-0057, decision no. 0041). Signed, written consent was obtained from all patients before enrolment in the study. The authors declared no conflict of interest.

Study population

One thousand and nineteen patients presented with dyspnea to the İzmir Katip Çelebi University Atatürk Education and Research Hospital, Turkey, emergency department during the study period. Respiratory failure based on arterial blood gas analysis was determined in 313 of these patients. One hundred two of these 313 patients who presented during the working hours of the senior resident conducting the research were included in the study. Ten of these 102 were excluded due to blood gas values representing venous blood, 12 due to history of ocular surgery and disease capable of increasing intraocular pressure (IOP), two due to having undergone brain surgery capable of increasing IOP, six due to arriving from an external center with endotracheal intubation, eight due to receipt of cardiopulmonary resuscitation because of arrest prior to ocular USG, three due to concurrent diagnosis of diabetic ketoacidosis, two due to pregnancy, three due to recent head-face trauma, and seven due to refusal to participate or absence of a sufficient degree

of consciousness to provide consent or of a suitable accompanying relative. The remaining 49 were included in the patient group, and a 27-member control group consisting of healthy volunteers was also established. The study design is shown in Figure 1.

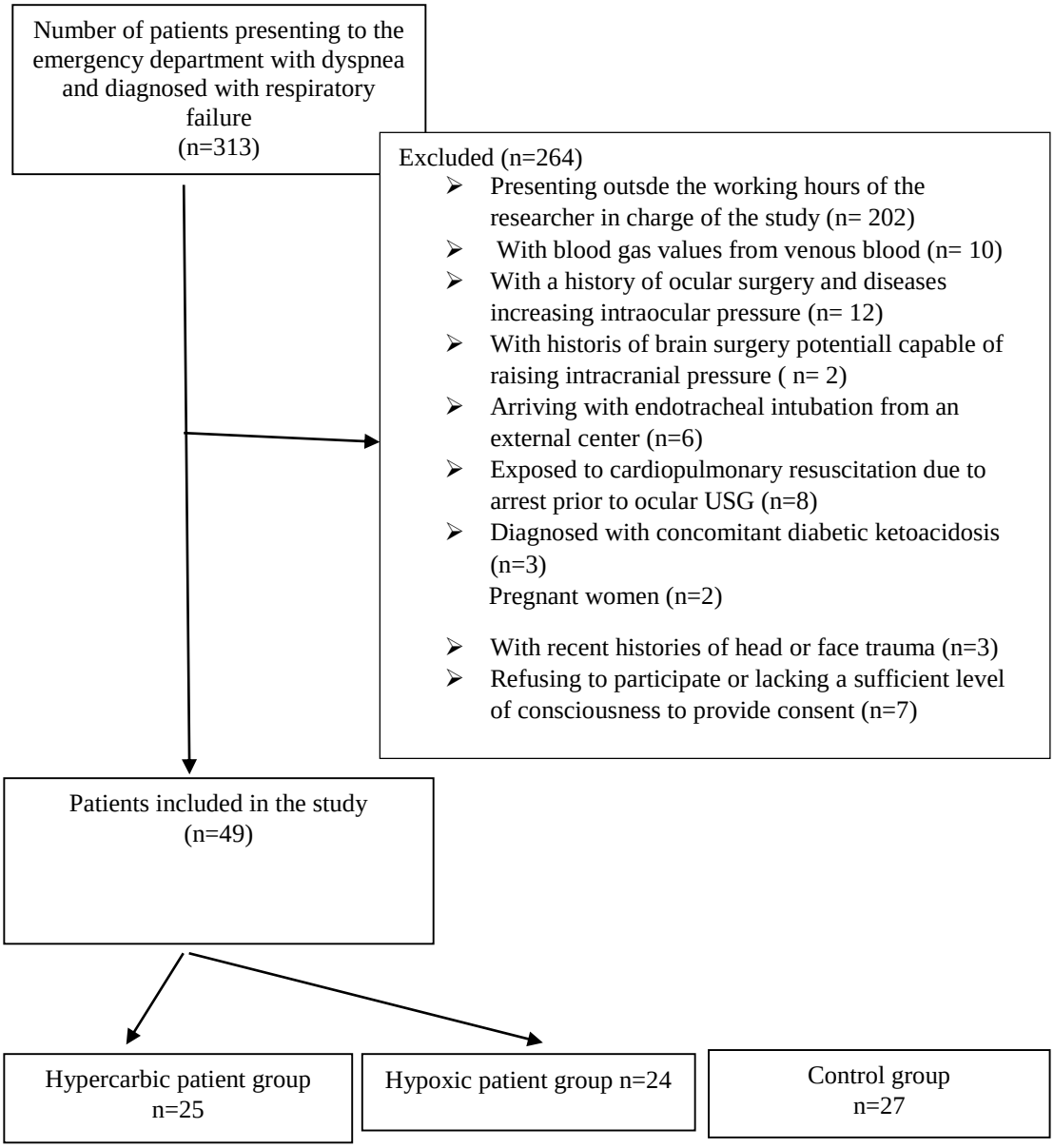

Figure 1. Study flow chart

Patients presenting to the emergency department with dyspnea were classified based on definitions of type 1 and 2 respiratory failure[1,2,3]. Patients with PaO2 <60 mmHg and normal or low PaCO2 based on arterial blood gas (ABG) results were included in the type 1 respiratory failure group, and those with PaCO2>45 mmHg in the type 2 respiratory failure group. Patients aged under 18, arriving already intubated and/or undergoing cardiopulmonary arrest before the completion of measurements, with known

additional lesions increasing ONSD, with any traumatic or non-traumatic deformity in either of the globes of the eye, with histories of head trauma or previous brain surgery, with diabetic ketoacidosis, or with suspected intoxication, and pregnant women, were excluded from the study. A control group was established consisting of healthy individuals aged over 18, consenting to participate, and with no optic neuritis known to affect the optic nerve sheath, arachnoid cyst of the optic nerve, previous history of optic nerve trauma, anterior orbital mass, cavernous sinus mass, history of eye trauma, or history of brain surgery.

Study protocol

The vital signs, oxygen saturation, demographic characteristics, initial ABG values at the time of presentation, and laboratory findings obtained during follow-up of the dyspneic patients included in the study were recorded onto case forms. After initial reception, oxygen support was initiated with a nasal cannula, and invasive or non-invasive mechanical ventilation requirements were determined. Criteria for the initiation of invasive mechanical ventilation were[14]

1. Cardiac or respiratory arrest
2. Hemodynamic instability
3. Severe encephalopathy (GCS <10)
4. Severe upper gastrointestinal bleeding
5. Facial trauma
6. Upper airway obstruction
7. Inability to protect the airway, and
8. Acute respiratory acidosis and hypercarbia (pH $\leqslant$7.35; Pa CO_2 >45 mmHg)

Criteria for initiation of non-invasive mechanical ventilation were[15]

1. Presentation of acute respiratory acidosis and hypercarbia (pH $\leqslant$7.35; Pa CO_2 >45 mmHg)
2. Tachypnea, and
3. Arterial oxygen tension/inspiratory oxygen fraction ratio (PaO2/FIO2) $\leqslant$200.

Treatment for the primary cause was initiated with oxygen among patients with no invasive or non-invasive ventilation requirements in the type 1 respiratory failure group. Patients in the type 2 respiratory failure group were evaluated primarily in terms of invasive and non-invasive mechanical ventilation. Treatment in the type 2 respiratory failure group was subsequently initiated with beta 2 agonists via nebulizer inhaler, anti-cholinergics, and intravenous glucocorticoid therapy, as recommended in the guideline[16]. ONSD was measured following initial assessment by the primary physician and initiation of emergency treatment within the first five minutes of presentation. The results were recorded onto the case forms. Following emergency treatment of the patient's dyspnea, the two-hour maximum recommended duration for non-invasive ventilation[17,18] was adopted as the time frame for control blood gas and ONSD measurement. Control blood gas values were determined after two hours, and ONSD measurement was repeated in the same way using the first protocol. Concurrently obtained blood gas measurement values and ONSD results were recorded onto the data collection form. Patients with PaCO2 > 45 mmHg at ABG analysis were recorded as the hypercarbic patient group, and those with PaO2 <60 mmHg as the hypoxic patient group.

Sonographic measurements

Measurements were performed by a senior emergency medicine physician with formal training in ultrasonography. This registrar received 30 min theoretical instruction on the subject of ONSD measurement and practical training involving 20 patients before the measurements were performed. ONSD measurements were carried out using a linear transducer with a frequency range of 7-11 MHz on a GE LOGIQ A5 ultrasound device with depth set to 7 cm in the nerve imaging module. Measurements were performed with the patient in the supine position, eyes closed, and the head in a neutral position. Following the application of a protective gelatin barrier, both globes were filled with a water-soluble USG gel. The probe was held transversely in the coronal plane, with the notch pointing to the patient's right side. The image was frozen at the point where the entrance of the optic nerve to the globe was best visualized, and the transverse diameter was measured by placing the cursors on the outer contours of the optic nerve, 3 mm below the entrance to the eyeball. Measurements were recorded (Figure 2). Measurements were repeated using the same method on both eyes, and the average values of the two measurements were used for the statistical analyses. ONSD measurements in hypercarbic patients were repeated in the same manner on the second hour of treatment (Figure 3), and the values were again recorded. The operator who performed the measurements was blinded to the patient's clinical information and laboratory results, and apart from

the healthy individuals, was unaware of which group the patients belonged to.

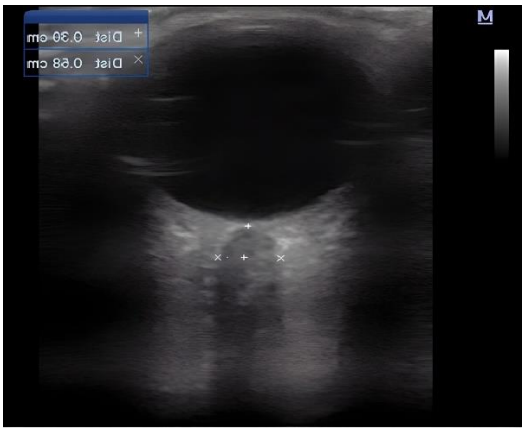

Figure 2. ONSD measured before treatment in the hypercarbic group

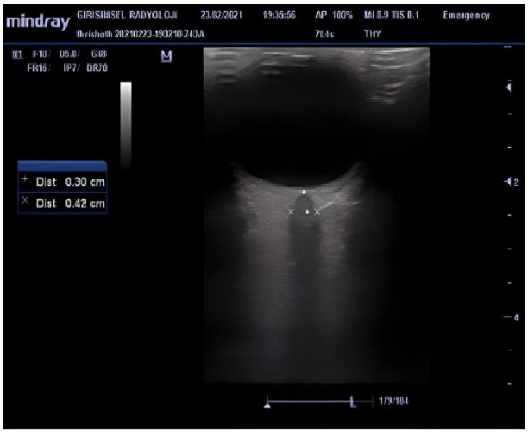

Figure 3. ONSD measured at the second hour of treatment in the hypercarbic group.

Outcome measures

This study investigated the value of the ONSD measured at presentation in the differential diagnosis of hypoxic and hypercarbic patients and, secondarily, the value of measurements at presentation and after two hours, and the difference between the two, in predicting hospitalization and in-hospital mortality.

Statistical analysis

Komut et al.'s[19] study was employed to calculate the sample size before the research commenced. The GPower3.1 program was used for this calculation; power analysis performed with 0.95 power and a 0.05 α margin of error based on that study determined an effect size of 1.54 and a minimum patient number of 22.

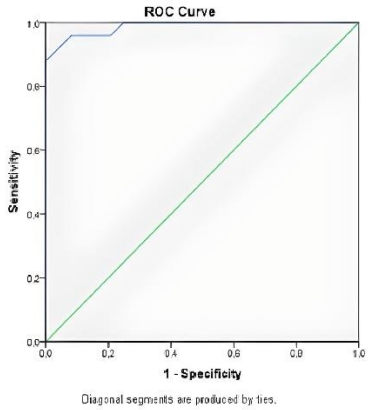

Figure 4. ROC CURVE showing the place of ONSD in the differential diagnosis of hypercarbic and hypoxic patients.

Descriptive statistics were expressed as frequency, percentage, mean, standard deviation, median, minimum, and maximum values. Number and percentage values were calculated for categorical variables, and mean, median, standard deviation, minimum, maximum, and interquartile range (IQR) for numerical variables.

Histogram curves and the Shapiro-Wilk test were used to determine whether continuous variables were normally distributed. The chi-square and Fisher's tests were applied to compare categorical variables between the control and patient groups, while the Mann Whitney U test was used to compare numerical variables, since these were not compatible with normal distribution. ROC curves were drawn to calculate the ideal cut-off values differentiating the groups and those values' sensitivity and specificity. ONSD values before and after treatment were compared in a dependent group using the Wilcoxon test.

Correlation between PaCO2 values and concomitantly measured ONSD values was evaluated using Spearman's correlation test. All statistical calculations were performed on SPSS 24.0 software and at a 95% confidence interval. p values <0.05 were regarded as statistically significant.

RESULTS

Seventy-six individuals were included in the study, 49 patients and 27 healthy controls. The mean age of the patient group was 69±12 years and 53% were men. The mean age of the healthy control group was 64±8 years and 48% were men. The demographic characteristics and vital findings of the patient and control groups are shown in Table 1.

Table 1. The general characteristics of the patient and control groups and first blood gas analysis data during presentation

Demographic characteristics and vital findings	Values during presentation	
	Patient group	Control group
Number	49	27
Age/years (Mean ± SD)	69 ± 12	64 ± 8
Male / M (%)	26 (53 %)	13 (48 %)
MAP (mmHg) (mean ± SD)	86± 15	81 ±11
Heart rate (/min) (mean ± SD)	86 ± 20	71± 20
Respiratory rate (/min))(mean ± SD)	22 ± 4	12 ± 3
Blood gas results		
Ph	7.36 ± 0.11	
PaO_2 (mmHg)	67 ± 22	
$PaCO_2$ (mmHg)	51 ±21	
Sat O_2 (%)	88 ±7	
Lactate (mmol/L)	2.8 ±3.4	
History		
CHF number (%)	20 (41%)	
COPD (%)	13(27%)	
DM number (%)	18 (37 %)	
HT number (%)	29 (60 %)	
CKF number (%)	5 (10 %)	
Cause of dyspnea		
DCHF	16 (33 %)	
DCHF+ pneumonia	4 (0.8 %)	
COPD attack	7 (16 %)	
COPD + pneumonia	6(12 %)	
Pneumonia alone (7 viral pneumonia)	11 (22 %)	
Asthma attack	4 (0.8 %)	
ARDS	1(0.2 %)	

SD: standard deviation, MAP: mean arterial pressure, PaO_2: partial oxygen pressure, $PaCO_2$: partial carbon dioxide pressure, CHF: congestive heart failure, DM: diabetes mellitus, HT: hypertension, CKF: chronic kidney failure, DCHF: decompensated congestive heart failure, COPD: chronic obstructive pulmonary disease, ARDS: acute respiratory distress syndrome

The patient group was divided into two subgroups - 25 hypercarbic individuals and 24 hypoxic patients. ONSD levels at baseline were subjected to two-group comparisons between the hypoxic, hypercarbic, and control groups. ONSD measurements differed significant between the hypercarbic group and both the hypoxic group and the control group, but there was no significant difference between the hypoxic patient group and the control group (0.123). ONSD measurements and ABG analysis results are shown in Table 2.

An ROC curve was drawn to determine the value of ONSD measurements in differentiating hypercarbic and hypoxic patients (Figure 4). An area under the curve (AUC) value of 0.98 for ONSD measurements was found to successfully differentiate the two groups (Table 3). The ideal cut-off point for differentiating the groups was determined at 4.3 mm, exhibiting 96% sensitivity, 91.7% specificity, a 92.3% positive predictive value (76.05% - 97.84%) and a 95.6% negative predictive value (76.26% - 99.34%)

Table 2. Concomitant arterial gas analysis and ONSD results in the patient and control groups

Measurements	Hypercarbic group	p	Hypoxic group	p	Control group	p	Hypercarbic group[
ONSD (mm)	5.3 ± 0.5	<0.001	3.8 ± 0.2	0.123	3.9 ± 0.3	<0.001	5.3 ± 0.5
pH	7.29 ± 0.09	<0.001	7.44 ± 0.05				
PaO_2 (mmHg)	71 ± 29	0.276	52 ± 7				
$PaCO_2$ (mmHg)	67 ± 18	<0.001	34 ± 6				
Sat O_2 (%)	85 ± 11	0.230	90 ± 3				
Lactate (mmol/L)	2.7 ± 2.8	0.733	3 ± 4				

ONSD: Optic nerve sheath diameter; $SatO_2$: Oxygen saturation

Table 3. Area under the ROC curve drawn for the differentiation of hypercarbic and hypoxic patients

Area under the curve				
Area under the curve	Standard deviation	p	95% confidence interval	
			Lower bound	Upper bound
0.988	0.011	<0.001	0.965	1.000

The initial ONSD value (ONSD-1) in patients with hypercarbic respiratory failure, 5.2 ± 0.5 mm, decreased significantly to 4.4 ± 0.6 mm after treatment (ONSD-2) (p<0.001). Powerful correlation was determined at blood gas analyses at the time of presentation and after two hours' treatment between PaCo2 results and concomitant ONSD measurements (correlation coefficient 0.733). Investigation of the value of ONSD in differentiating hospitalized and discharged patients with hypercarbic respiratory failure showed that the difference (delta) in ONSD-1 measured at baseline and ONSD-2 measured after two hours was not statistically significant (Table 4). Investigation of the value of ONSD in differentiating surviving and exitus patients with hypercarbic respiratory failure revealed no significant difference (delta) in or between ONSD-1 and ONSD-2 between the surviving and exitus groups (Table 5).

Table 4. Comparison of ONSD values in the discharged and hospitalized cases

	Discharged subjects N:7	Hospitalized subjects N:18	p
ONSD-1 (mm)	5.2 ± 0.5	5.3 ± 0.5	0.739
ONSD-2 (mm)	4.4 ± 0.6	4.4 ± 0.4	0.762
Delta_ONSD (mm)	0.8 ± 0.2	0.9 ± 0.3	0.395

Table 5. Comparison of ONSD measurements in the exitus and surviving cases

	Surviving subjects n:17	Exitus subjects n:8	p
ONSD-1 (mm)	5.3 ± 0.5	5.4 ± 0.5	0.618
ONSD-2 (mm)	4.3 ± 0.5	4.4 ± 0.5	0.837
Delta_ONSD (mm)	0.8 ± 0.2	0.9 ± 0.3	0.596

DISCUSSION

The two main factors that increase ICP are CSF and the vasogenic component[20]. High PaCO2, increased cerebral metabolism, and cerebral hyperemia can cause increased ICP of vasogenic origin. Numerous studies have shown that increases in PaCO2 values, which are known to exert a very strong vasodilator effect on cerebral vascular structures, are correlated with increased intraoperative ICP.[21,22,23,24] To the best of our knowledge, no clinical studies have investigated the use of increased ICP in the differential diagnosis of diseases progressing with hypercarbia and those progressing with hypoxia. Publications examining the ICP-raising effect of

diseases progressing with hypercarbia have been limited to case reports[25]. From that perspective, this study is the first to evaluate the value of ONSD in clinical outcomes and differential diagnosis of dyspneic patients by indirectly evaluating ICP using ONSD measurement. ONSD values in patients with hypercarbic respiratory failure in the present study were significantly higher than those in patients with hypoxic respiratory failure and in the healthy controls. In addition, at a cut-off value of 4.3 mm, ONSD powerfully differentiated type 1 and type 2 respiratory failure with 96% sensitivity and 91.7% specificity. However, ONSD did not emerge as an effective parameter in predicting hospitalization and mortality in hypercarbic patients. The study population consisted of patients presenting to the emergency department with dyspnea. The mean age of the study population was 69 years, and 53% were men. Similarly, mean ages of 70-72 and male predominance between 59% and 65% have been reported in previous studies involving acute dyspneic patient populations[26,27]. In terms of age and gender, and considering the inclusion criteria, a similar distribution was observed in the present study, with a larger male population. This may be due to the prevalence of chronic obstructive pulmonary disease (COPD) in Turkey being significantly higher in males (male 20%, female 8%)[28]. In terms of acute respiratory failure diagnoses in the present study, decompensated heart failure was present in 33% of patients, COPD attack in 16%, COPD+pneumonia in 12%, and viral or bacterial pneumonia in 22%. In a study investigating the etiology of respiratory failure in elderly patients, Patrick Ray et al. reported acute pulmonary edema in 43% of patients, COPD in 32%, and pneumonia in 35%, findings consistent with the present study[29].

Previous research has shown that hypercarbia increases ICP.[21,22,23,24]. Numerous previous studies have shown that an increase in ICP can be successfully evaluated using ONSD measurement[21,30]. Although hypercarbia is known to increase ICP, the acute effect of PaCO2 on ONSD is still unknown. Experience on this subject is limited to a few studies performed with patients under anesthesia[2,31]. In the present study, the ONSD values of hyperbaric patients presenting to the emergency department with dyspnea were higher than those in the normal population and hypoxic patients. ONSD values then decreased with treatment, together with the diminution observed in PaCO2. In their experimental study, M. Dinsmore et al. examined the effects on ONSD of changes in carbon dioxide at the end of inspirium. Consistent with the results of the present research, those authors showed that ONSD increased together with CO2 values, and that ONSD returned to normal values when hypercarbia decreased[30]. Powerful correlation was observed in the present study between PaCO2 blood gas values in hypercarbic patients and concomitantly measured ONSD (cc: 0.733). Consistent with our findings, Seo et al. observed correlation between PaCO2 and concomitantly measured ONSD during reperfusion in liver transplantation patients (cc: 0.746)[20]. Although these results support the idea that ONSD measurements can be used in the follow-up of hypercarbic patients, ONSD was unsuccessful in predicting hospitalization and mortality in our population.

Further studies showing the applicability of ONSD in the differentiation of type 1 and type 2 respiratory failure are now needed. We also think that this measurement and its use in clinical practice will increase with studies involving critically ill patients in the emergency department and showing the change in PaCO2 and ONSD.

The principal limitations of this study are its single center nature and relatively low patient number. Another limitation is that ultrasonographic measurements were performed by a single operator.

Powerful correlation was determined between PaCO2 and ONSD values in hypercarbic patients in this study. In addition, ONSD was significantly higher in the hypercarbic patients than in the hypoxic patients and the healthy population, and this may represent an effective parameter in the differential diagnosis of type 1 and 2 respiratory failure. However, ONSD may not be an effective parameter in predicting hospitalization and mortality in hypercarbic patients.

Yazar Katkıları: Çalışma konsepti/Tasarımı: ZK, AY, MGE; Veri toplama: MGE; Veri analizi ve yorumlama: AY, AK; Yazı taslağı: MGE, AY; İçeriğin eleştirel incelenmesi: ZK, AK; Son onay ve sorumluluk: MGE, ZK, AY, AK; Teknik ve malzeme desteği: AK; Süpervizyon: ZK; Fon sağlama (mevcut ise): yok.
Etik Onay: Bu çalışma için İzmir Katip Çelebi Üniversitesi Girişimsel Olmayan Klinik Araştırmalar Etik Kurulundan 18.02.2021 tarih ve 41 sayılı kararı ile etik onay alınmıştır.
Hakem Değerlendirmesi: Dış bağımsız.
Çıkar Çatışması: Yazarlar çıkar çatışması beyan etmemişlerdir.
Finansal Destek: Yazarlar finansal destek beyan etmemişlerdir.
Author Contributions: Concept/Design : ZK, AY, MGE; Data acquisition: MGE; Data analysis and interpretation: AY, AK; Drafting manuscript: MGE, AY; Critical revision of manuscript: ZK, AK; Final approval and accountability: MGE, ZK, AY, AK; Technical or material support:AK; Supervision: ZK; Securing funding (if available): n/a.

Ethical Approval: For this study, ethical approval was obtained from Izmir Katip Celebi University Non-Interventional Clinical Research Ethics Committee with the decision dated 18.02.2021 and numbered 41.
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