TÜBİTAK

Turkish Journal of Medical Sciences

http://journals.tubitak.gov.tr/medical/

Research Article

Turk J Med Sci
(2018) 48: 980-984
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doi:10.3906/sag-1803-132

Effects of tourniquet usage in lower extremity surgery on optic nerve sheath diameter

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Received: 15.03.2018 • **Accepted/Published Online:** 27.07.2018 • **Final Version:** 31.10.2018

Background/aim: The aim of this study was to evaluate changes in intracranial pressure following tourniquet deflation using noninvasive ultrasonographic optic nerve sheath diameter (ONSD) measurements.

Materials and methods: Our study included 59 adult patients between the ages of 18 and 65 years from the American Society of Anesthesiologists (ASA) I/II risk groups who were scheduled to undergo elective orthopedic surgery of the lower extremities using a tourniquet under general anesthesia. ONSD and end-tidal CO2 (ETCO2) were measured 5 times: 15 min prior to the anesthesia induction; just prior to the deflation of the tourniquet; and at 5, 10, and 15 min after the deflation. Additionally, age, sex, weight, height, ASA score, and duration of operation and tourniquet usage were recorded.

Results: The ONSD value measured 5 min after the deflation was significantly higher than all of the remaining measurements. There was a significant correlation between the ONSD and ETCO2 measurements at 5 and 10 min after deflation ($r = 0.61$, 95% CI 0.42–0.75, $P < 0.0001$ and $r = 0.30$, 95% CI 0.04–0.51, $P < 0.05$, respectively).

Conclusion: The ultrasonographic ONSD measurements, which were obtained using a simple and noninvasive approach, increased significantly following tourniquet deflation, and this increase was correlated with an increase in ETCO2.

Key words: Tourniquet, intracranial pressure, optic nerve sheath diameter

1. Introduction

Pneumatic tourniquets have frequently been used in orthopedic surgeries in order to provide a bloodless surgical area and reduce blood loss via wrapping around the extremities. While there are many advantages to tourniquet applications, hemodynamic or metabolic changes may be observed in relation to the inflation and deflation of the tourniquet (1). When the tourniquet is inflated, metabolic changes such as increases in carbon dioxide (CO_2), lactic acid, and potassium and reductions in P_aO_2 and pH are observed in the ischemic extremity. These ischemic products are then secreted into the general circulation following deflation of the tourniquet (2).

P_aCO_2 plays an important role in the regulation of cerebral vasomotor tone in normal patients. Increase in $PaCO_2$ leads to concurrent cerebral vasodilatation and an increase in intracranial pressure (ICP) (3). The sudden increase in $ETCO_2$ also observed after the deflation is associated with a 50% increase in the middle cerebral artery blood flow velocity, which peaks at approximately 2–4 min and regresses to normal values after 8–10 min (4). Although this transient increase is well tolerated in healthy individuals, it may lead to a sudden increase in ICP in patients with a mass-occupying lesion in which intracranial compliance is reduced (5).

ICP is monitored in many clinical studies using both invasive and noninvasive methods. The standard invasive ICP monitoring technique, which is performed using an intraparenchymal probe or intraventricular catheter, is accepted as the standard. However, the clinical effectiveness of this technique is controversial since it is invasive and thus has certain risks to it, including bleeding and infection (6). Previous studies that compared optic nerve sheath diameter (ONSD) ultrasonography and invasive methods in ICP monitoring revealed a strong correlation between these two methods (7). The sheath around the optic nerve is the continuation of the dura and extends along the optic nerve within the sheath in the subarachnoid space. A possible increase in ICP is transmitted to the optic nerve and results in swelling in the optic disc and papillary edema (8). ONSD measurement via ultrasonography is a noninvasive, safe method that shows the rapid changes in ICP (9). The aim of our investigation was to define the change in ONSD and the correlation between this change and $ETCO_2$ after tourniquet deflation.

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2. Materials and methods

Following the approval of the Karadeniz Technical University Faculty of Medicine Local Ethics Committee (Approval No. 2016/296), written informed consent was obtained from all participants between April 2016 and January 2017. This prospective observational study included 59 adult patients aged between 18 and 65 years who were classified in the I–II risk groups of the American Society of Anesthesiologists (ASA) Physical Status Classification System and were scheduled to undergo elective orthopedic surgery on a lower extremity using a tourniquet (Table 1).

Exclusion criteria were: <18 and >65 years of age, known history of orbital trauma, optic nerve pathology, history of asthma or coronary obstructive pulmonary disease, pre- or intraoperative bronchospasm, previous corneal or intraocular surgery, and/or a history of increased ICP. All patients underwent electrocardiography (ECG), heart rate (HR), noninvasive mean blood pressure (MAP), peripheral oxygen saturation (SpO_2), end-tidal CO_2 ($ETCO_2$) (Spacelabs Healthcare, Snoqualmie, WA, USA), bispectral index (BIS) (Aspect Medical Systems, Norwood, MA, USA), and neuromuscular monitoring as standard.

The induction of anesthesia was performed via premedication with 2 mg of intravenous (i.v.) midazolam and 3 min of oxygenation, followed by 2–3 mg/kg propofol (i.v.) and 1 μg/kg fentanyl for muscular relaxation, and 0.6–1 mg/kg rocuronium after the BIS value was lowered below 60 using endotracheal intubation. Mechanical ventilation was adjusted to a tidal volume of 6–8 mL/kg and a respiratory rate of 10–12/min. For the maintenance of anesthesia, the total gas flow was adjusted to 2.5 L/min of 1%–3% sevoflurane plus an O_2/medical air ratio of 1:1 and continuous remifentanil infusion of 0.1–0.5 μg/kg per minute, with BIS being 40–60. In all patients, a standard pneumatic tourniquet with an 11-cm-wide cuff was placed with the distal edge 15 cm proximal to the proximal pole of the patella, and it was inflated to a pressure of 300 mmHg throughout the surgery. The surgical intervention was started following the inflation of the tourniquet. In order to control bleeding, the tourniquet was released prior to skin closure. The sonographic measurement of ONSD was performed by two anesthesiologists with a minimum of 3 years of experience in the field, and in accordance with the studies in the literature (10).

A thin gel layer was applied to the upper eyelid with the patient in the supine position and with the eyes closed. A 7.5-MHz linear probe was placed on the gel without applying excessive pressure. In order to visualize the optic nerve, the probe was set to the proper position on the eyeball. The ONSD was then measured 3 mm behind the optic globe, with a total of four measurements obtained from each eye on the transverse and sagittal planes (Figure 1). The final ONSD value was accepted as the mean of these four measurements. ONSD measurements and vital signs were recorded by different observers. HR, MAP, SpO_2, BIS, $ETCO_2$, and ONSD values were recorded 15 min prior to the induction of the anesthesia (T_0), just prior to the deflation of the tourniquet (T_i), and at 5, 10, and 15 min after the deflation of the tourniquet (T_{d5}, T_{d10}, and T_{d15},

Table 1. Patient general characteristics and intraoperative data variables.

Patient characteristics	
Sex (M/F)	26 (44)/33 (56)
Age (years)	33.9 ± 15.5
BMI (kg/m^2)	27.4 ± 4.3
ASA (I/II)	43 (73)/16 (27)
Intraoperative data Operation time (min)	90.3 ± 26.2
Tourniquet time (min)	81.6 ± 25.0

Data of patients are expressed as numbers (%), mean ± standard deviation.
BMI; Body mass index, ASA; American Society of Anesthesiologists Physical Status Classification System

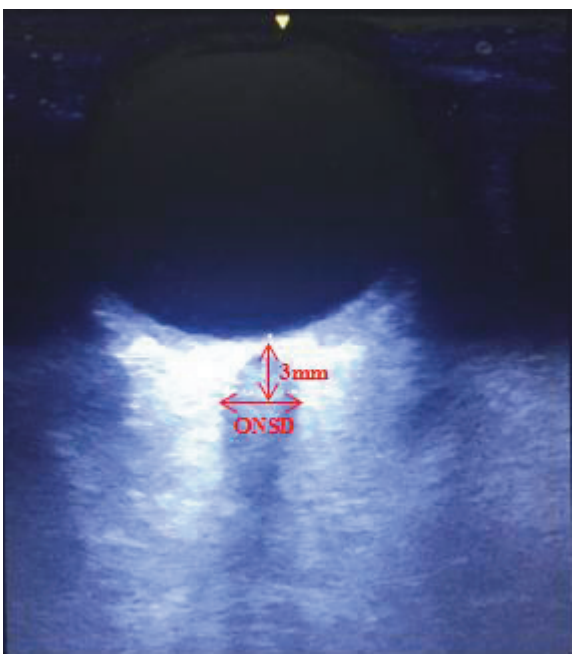

Figure 1. Sonography image of the optic nerve sheath. The optic nerve sheath is shown as a vertical hypoechoic band between echogenic retrobulbar fat tissues. Optic nerve sheath diameter is measured vertically, 3 mm behind the papilla. ONSD, Optic nerve sheath diameter.

respectively). Additionally, age, sex, body mass index, ASA score, and duration of operation and tourniquet use were recorded for each patient.

In our study, our hypothesis was that intracranial pressure increased following tourniquet deflation, as shown by ONSD. Based on our unpublished preliminary study results, the expected change of mean ONSD measurement was 1.2 ± 0.2. The minimum calculated sample size, determined by using a free online paired sample size calculator, was 12 for 0.05 type-1 error with 0.8 power.

2.1. Statistical analysis

All 59 patients' data were used for statistical analysis. SPSS 23.0 was used for statistical analysis (IBM Corp., Armonk, NY, USA). Data normality was assessed with the Kolmogorov–Smirnov test. All variables used for comparison fulfilled the parametric data criteria. Variables were HR, MAP, ONSD (T_o, T_i, T_{d5}, T_{d10}, T_{d15}), and $EtCO_2$ (T_i, T_{d5}, T_{d10}, T_{d15}). Statistical comparisons between these groups (Table 2) were performed using the repeated measures ANOVA test (with Bonferroni correction).

Pearson correlation analysis was used to explore the association of ONSD and $ETCO_2$ values (Figure 2) to suggest the causative relationship of the harmful effects of the tourniquet method.

The results were evaluated within a 95% confidence interval and at a significance level of $P < 0.05$.

3. Results

During the study years, 100 patients in total were scheduled to undergo elective orthopedic surgery of the lower extremities using the tourniquet method. Forty-one patients were not included in this study, since 22 patients were ASA III, 10 patients were >65 years old, and 9 patients

Table 2. Clinical parameters during operation.

	T_o	T_i	T_{d5}	T_{d10}	T_{d15}
HR (bpm)	76.5 ± 12.2	75.2 ± 12.3	73.2 ± 8.2	73.3 ± 8.2	74.3 ± 8.2
MAP (mmHg)	93.0 ± 11.8	90.9 ± 10.4	78.2 ± 10.9[a]	81.2 ± 12.2[ab]	81.7 ± 10.8[a]
$EtCO_2$ (mmHg)	0	32.7 ± 1.5	41.2 ± 3.2[c]	36.0 ± 1.7[c]	34.1 ± 1.7
ONSD (mm)	3.74 ± 0.02	3.82 ± 0.03	5.14 ± 0.14[e]	4.24 ± 0.03[d]	3.84 ± 0.04

Data of patients are expressed as numbers (%), mean ± standard deviation.
HR, Heart rate; MAP, mean arterial pressure; BIS, bispectral index; ONSD, optic nerve sheath diameter.
[a]: Significant reduction compared to T_0 ($P < 0.05$) and Ti ($P < 0.05$).
[b]: Significant increase compared to T_{d5} ($P < 0.05$).
[c]: Significant increase compared to T_0 ($P < 0.05$) and T_i ($P < 0.05$).
[d]: Significant increase compared to T_0 ($P < 0.05$).
[e]: Significant increase compared to other periods ($P < 0.05$).

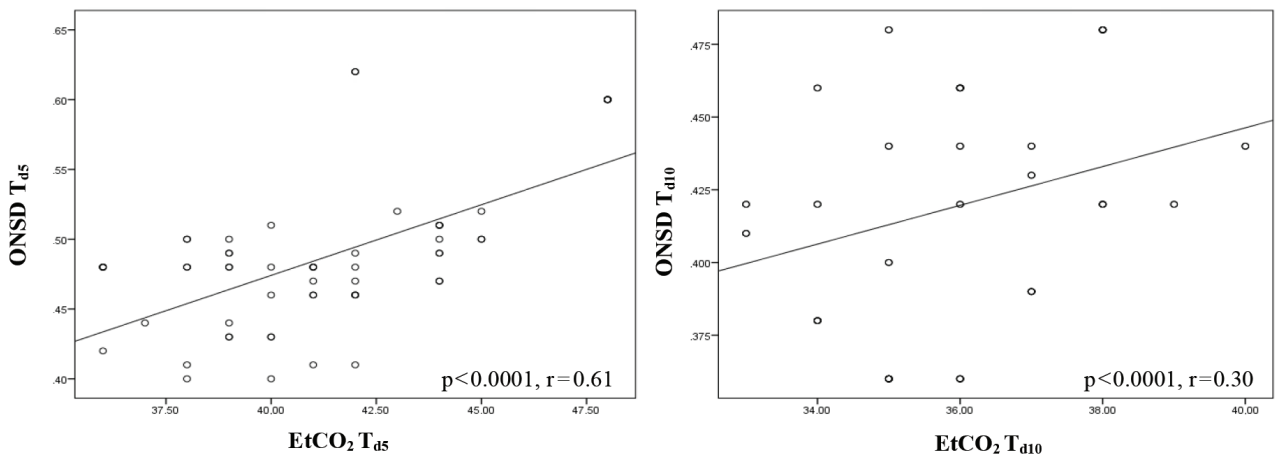

Figure 2. Correlation between ETCO2 and ONSD at 5 and 10 min after deflation of the tourniquet. ONSD, Optic nerve sheath diameter; EtCO2, end-tidal carbon dioxide.

had serious systemic illness. Following the application of the inclusion and exclusion criteria, only 59 adult patients aged between 18 and 65 years who were ASA I–II and were scheduled to undergo elective orthopedic surgery of the lower extremities using a tourniquet were included for study analysis between April 2016 and January 2017. The general characteristics and intraoperative data of the patients are presented in Table 1. Heart rate, mean arterial pressure, saturation, $ETCO_2$, ONSD, and bispectral index values are presented in Table 2. The MAP values at 5, 10, and 15 min after the deflation of the tourniquet were significantly lower than baseline values and the values obtained just prior to the deflation. The MAP value observed at 10 min after deflation was significantly higher than that observed 5 min after deflation ($P < 0.05$). BIS values were between 40 and 60 in the intraoperative period. ONSD observed 5 min after deflation was 5.14 mm (range: 4.15–6.24), and was significantly higher than all the other values observed throughout the follow-up (Table 2). ONSD observed 10 min after deflation was 4.24 mm (range: 3.68–4.84), and was significantly higher than the baseline value. $ETCO_2$ observed 5 and 10 min after deflation was significantly higher than the values at baseline and just prior to the deflation of the tourniquet (41.2 mmHg [range: 38.9–43.4], 36.0 mmHg [range: 34.2–37.8, respectively) (Table 2).

The correlation coefficients for ONSD and $ETCO_2$ values observed 5 and 10 min after deflation were 0.61 (95% CI: 0.42–0.75, $P < 0.0001$) and 0.30 (95% CI: 0.04–0.51, $P < 0.05$), respectively, which showed a significant correlation (Figure 2). However, the correlation coefficients for ONSD and $ETCO_2$ measurements obtained 15 min after deflation showed no significant changes.

No complication was observed during ultrasonographic ONSD measurement throughout the study.

4. Discussion

CO_2 increase as a result of tourniquet deflation-related reperfusion is an important risk factor for an increase in ICP in tourniquet-guided surgeries. Many studies exist in the literature on tourniquet-related complications; however, to our knowledge, no study has been completed on the ultrasonography-mediated detection of an increase in ICP via ONSD, which is measured noninvasively.

The major findings in the present study are that the ONSD values obtained via ultrasonography were increased following tourniquet deflation and that this change was associated with an increase in $ETCO_2$.

Ventriculostomy is the gold-standard technique for the direct measurement of ICP, although it has several complications, such as bleeding and infection, and requires sterile conditions (11,12). Studies in the literature address the effects of ICP on ONSD in various surgeries, such as laparoscopic surgeries, in the intensive care unit (where neurosurgery is performed), or in pain management (7,9,13). However, no study in the literature addresses the measurement of ICP change via ONSD following tourniquet deflation in surgeries of the lower extremities.

When a tourniquet is inflated, ischemic products are observed in the ischemic extremity and are then secreted into the general circulation following the deflation of the tourniquet (14). CO_2, which is a potent vasodilator, affects ICP. The rapid increase in CO_2 observed as a result of postdeflation reperfusion leads to a 50% increase in the velocity of the middle cerebral arterial blood flow, which reaches a peak at 2–4 min and returns to normal values within 8–10 min (15). This increase is well tolerated in healthy individuals, but it can lead to a serious increase in ICP in patients with secondary brain injury (5). In our study, 15 min after the deflation of a patient's tourniquet, normal ONSD values were observed and the patient was extubated and sent to a postanesthesia care unit. Thus, no later ONSD measurements were made. The clinical importance of later measurements has not been considered.

Studies investigating the effects of tourniquet application times on ICP are limited. In a case of multiple traumas, where the ICP was measured via invasive methods (a fiberoptic epidural transducer), a lower-extremity tourniquet was applied with a pressure of 350 mmHg for approximately 2 h. Within 15 s after deflation, a 7-mmHg increase in $ETCO_2$ was accompanied by a sudden increase in ICP from 17 to 58 mmHg (16). In our study, we found a good positive correlation between $ETCO_2$ and ONSD in the fifth minute and a weak positive correlation in the tenth minute (Figure 2). We believe that the increase in $ETCO_2$ plays an important role in the increase in ONSD, which is a sign of ICP increase. Therefore, our results suggest that during the postdeflation period, the serial ultrasonographic measurement of ONSD contributes to the evaluation of ICP. Thus, both serial monitoring and the early detection of a significant change in ICP may prevent destructive complications with a mass-occupying lesion in which intracranial compliance is reduced and where brain injury occurs in a patient.

In many studies, an ICP value of over 20 mmHg was considered to represent intracranial hypertension. The optimal cut-off value for ONSD is ≥5.0 mm. However, the literature features various ONSD cutoff values of between 5.2 and 5.9 mm obtained via invasive and noninvasive measurements. In these studies, sensitivity ranged between 74% and 95%, and specificity ranged between 73% and 100% (1,9,10,17,18). In the present study, the ONSD value observed 5 min after deflation was 5.1 mm. Although ONSD values were high in other deflation periods, they were within normal limits.

Inhaled anesthetics may influence the cerebral blood flow and ICP due to vasodilatation (19). This effect is accompanied by elevated ICP. In our study, sevoflurane

was used as an inhaled anesthetic. Matta et al. reported that sevoflurane has a direct dose-dependent cerebral vasodilatory effect, and no increase in ICP occurred at 0.5, 1.0, and 1.5 minimum alveolar concentrations (20). Thus, the sevoflurane concentration did not exceed 3% throughout the surgery and therefore may not have influenced the ONSD level.

Our study had the following limitations: first, ONSD was not compared with an invasive method, such as ventriculostomy, in which ICP is directly evaluated. Although invasive methods are more accurate than ONSD in the determination of real ICP values, such a comparison was not possible due to the surgical methods and the ethical concerns that could arise. Second, our study did not include a control group because we investigated our protocol as an observational study. Further prospective randomized studies are required.

In conclusion, we observed that ultrasonographic ONSD measurements, which are obtained using a simple and noninvasive approach, were increased following tourniquet deflation, and this change correlated with the increase in $ETCO_2$ that occurred. We suggest to clinicians that normocapnia maintained through hyperventilation after tourniquet deflation can prevent increased ICP.

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