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Ultrasonographic Evaluation of Intracranial Pressure during Rapid Maxillary Expansion

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Research Article

History

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ABSTRACT

Objectives: The objective of this research was to assess the impact of rapid maxillary expansion on intracranial pressure in individuals with maxillary transverse deficiency. This was achieved by measuring the optic nerve sheath diameter using ultrasonography (US).

Materials and Methods: This prospective observational study included 25 young patients (mean age 13.10 ± 1.20) with bilateral posterior cross bite. Acrylic cap splint hyrax appliances were given to all patients for rapid maxillary expansion (RME). Prior to the initial screw activation (T0), the patient underwent monitoring, collection of vital signs, and measurement of optic nerve sheath diameter (ONSD) using US. Subsequent measurements were taken at 1 minute (T1), 10 minutes (T2), and 60 minutes (T3) following the first activation. During the final session of the rapid maxillary expansion therapy, the same measurement procedure was repeated (T4, T5, T6, and T7) as in the initial activation session. The patients' perception of pain during screw activation (T1, T5) was also assessed using a four-category verbal rating scale (VRS-4). A p-value of less than 0.05 was considered statistically significant for the conducted analysis.

Results: The ONSD values, at T1 and T5, showed a significant increase within 1 minute following screw activation. However, there was no significant difference observed between the initial (T0) and final (T7) ONSD values throughout the active RME therapy.

Conclusions: Intracranial pressure rises immediately after screw activation, but it auto regulates at the end of the active RME therapy.

Key words: Optic Nerve Sheath Diameter, Rapid Maxillary Expansion, Intracranial Pressure, Ultrasonography.

Hızlı Üst Çene Genişletme sürecinde Kafa içi Basıncının Ultrasonografik Değerlendirilmesi

Süreç

Geliş: 17/07/2023
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ÖZ

Amaç: Bu çalışmanın amacı; üst çene transversal yetersizliği bulunan hastalarda uygulanan hızlı genişletme protokolünün kafa içi basıncı üzerine etkilerini optik sinir kılıf çapını ultrason ile ölçerek değerlendirmektir.

Yöntemler: Bu prospektif gözlemsel çalışma posterior çapraz kapanışı olan 25 genç hastadan (ortalama yaş 13.10 ± 1.20) oluşmaktadır. Hastaların tamamına hızlı üst çene genişletme için akrilik kaplı hyrax apareyi uygulandı. İlk vida aktivasyonundan hemen önce (T0) hastalar monitörize edildi, vital bulgular kaydedildi ve optik sinir kılıfı çapı ultrason ile ölçüldü. Bu ölçümler 1 (T1), 10 (T2) ve 60 dakika (T3) sonra tekrarlandı. Aktif genişletme tedavisinin son seansında aynı ölçümler ilk aktivasyon esnasındaki gibi uygulandı. (T4, T5, T6 ve T7). Hastaların vida aktivasyonları (T1, T5) sırasındaki ağrı değerleri de not edildi. (VRS-4). Sonuçlar istatiksel olarak $p<0.05$ düzeyinde anlamlı olarak kabul edilmiştir.

Bulgular: Optik sinir kılıf çapının vida aktivasyonundan sonraki ilk 1 dakika içinde anlamlı derecede arttığı görüldü. Aktif genişletme tedavi sürecinde başlangıç (T0) ve bitiş (T1) optik sinir kılıf çaplarında ise anlamlı değişim görülmedi.

Sonuçlar: Kafa içi basıncı vida aktivasyonunu takiben artmakta anca aktif genişletme tedavisinin sonunda otoregule olmaktadır.

Anahtar Kelimeler: hızlı üst çene genişletme, intrakraniyal basınç, optik sinir kılıfı çapı, ultrasonografi.

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Introduction

In RME therapy, high level orthopedic forces lead to a splitting of mid-palatal suture, causing maxillary halves to separate from one another.[1] These heavy forces affect not only adjacent structures in the face but also the structures in the cranium caused by unpredictable transmission of forces.[2] Research studies have indicated that utilization of RME has been associated with notable alterations in cranial and circummaxillary sutures, as well as mid-palatal sutures, in developing individuals.[3,4] Therefore, several researchers have focused on potential effects associated with RME therapy upon visceral structures and the neurocranium.[5,6] According to a study, the application of significant forces during expansion exerts stress on the structures of the cranial base, which results in the widening of various areas such as the superior orbital fissure, spinous foramen, oval foramen, optical foramen, round foramen, and carotid sulcus. Consequently, there is a possibility of micro-fractures and potential damage to nerves and blood vessels in these regions.[5] *Li et al.*[6] determined that intensive anatomical stresses on craniofacial structures during the expansion process can induce cerebral hemodynamic changes, including increased cerebral blood volume (CBV) and cerebral blood flow (CBF). *Romeo et al.*[7] observed that the use of RME may increase intracranial pressure, impairing venous drainage by affecting cerebral venous circulation on magnetic resonance imaging (MRI).

The total volume of the brain, which consists of cerebrospinal fluid (CSF) and blood, influences the intracranial pressure (ICP).[8] Within the central nervous system, the optic nerve is surrounded by the infraorbital subarachnoid space that exhibits pressure closely resembling and correlating with the ICP.[9]

Optic nerve sheath diameter (ONSD) has been suggested as a dependable method for detecting elevated ICP.[10] Research studies have demonstrated that changes in ONSD occur almost simultaneously with rapid fluctuations in ICP, reflecting an immediate response.[9,10] Today, invasive and radiological methods have been introduced to measure raised ICP such as direct ICP monitoring via an intracranial catheter and a computerized brain tomography (CBT).[10] Nevertheless, more recent researches have also documented an alternative approach, which is non-invasive, for assessing ICP by means of US to measure the diameter of the ONSD. The measurement of ONSD for determining ICP is also easy-to-apply, reliable, and non-invasive, in comparison to other methods.[9]

There are many clinical and simulative works that provide clinicians with valuable information about skeletal and dental effects of RME therapy as a part of orthodontic treatment.[3] While earlier investigations have predominantly concentrated on examining the impact of forces on craniofacial structures in patients undergoing RME treatment, the potential secondary consequences on the brain hemodynamics, intracranial vascular compartment and intracranial pressure remain unexplored and undefined.[5,11] Thus, the aim of this research was to assess the potential impact of RME therapy on intracranial pressure through the measurement of the ONSD.

Material and Methods

This research received approval from the Scientific Research Ethics Committee at Karadeniz Technical University Faculty of Medicine (2020/296, 06/11/2020), and parental consent was obtained for all patients. The sample size was established using existing data as a reference.[6] By considering an alpha level of 0.05, beta of 0.20, and effect size of 0.65, it was calculated that a total of 22 participants would be needed. To eliminate potential data loss, final sample size was designed with at least 25 patients (15 females and 10 males). The average age of the participants was 13.10 ± 1.20 years, falling within a range of 10 years and 11 months of age to 14 years and 11 months of age. Every patient exhibited both bilateral posterior cross bite and maxillary constriction, indicating suitability to undergo RME treatment. Their skeletal maturity varied between the CS1 and CS3 stages according to the Cervical Vertebral Maturation (CVM) index. The exclusion criteria were pathologic periodontal conditions, major skeletal asymmetries, previous or ongoing orthodontic treatment, and acute or chronic ophthalmic diseases.

RME treatment was administered using a bonded acrylic splint and a hyrax screw appliance following the method outlined by McNamara and Brudon (Figure 1). The appliance was activated by turning the screw a quarter of a rotation (0.25 mm) every 12 hours, which was initiated immediately after it was placed.[12] On the days when intracranial pressure (ICP) measurements were conducted, the researcher activated the screws, while the patients or their parents performed daily activations throughout the expansion process. The average duration of the treatment period was 14 days.

ICP measurements were conducted by a single operator using an US device from General Electric (GE, Vivid-e, Wauwatosa, USA) equipped with a 7.5 MHz linear probe. During the US imaging, patients were positioned supine with their head elevated at ~30°. To protect the closed upper right eyelid, a transparent film dressing was applied followed by a layer of water-soluble US gel. The operator carefully moved the probe to obtain the optimal image of the optic nerve entering the eyeball (Figure 2). The operator measured the ONSD behind the globe, approximately 3 mm, using the ultrasound images (Figure 3). Each bulb was examined two times by the operator and the mean values of the ONSD were used for data.

The measurement of ONSD was carried out immediately prior to the initial screw activation (T0). Subsequently, measurements were repeated at intervals of 1 minute (T1), 10 minutes (T2), and 60 minutes (T3) after the first activation. On the day when the expansion process concluded, the same measurement procedure was repeated, mirroring the protocol employed during

the initial activation appointment (T4, T5, T6, and T7). The study design and measurement times are provided in Figure 4.

Physiological parameters including heart rate, mean arterial pressure (MAP), and peripheral oxygen saturation (SpO2) were documented both before and after the screw activation intervals (T0, T1, T4, and T5) using conventional monitoring techniques (Spacelabs Healthcare, USA).

The patients' experience of pain during screw activation was assessed using a verbal rating scale (VRS-4) at T1 and T5. Each patient was requested to rate their pain level on the VRS-4, which consisted of self-explanatory categories ranging from no pain to mild, moderate, or severe pain.

Statistics

The statistical analysis was conducted utilizing the NCSS (Number Cruncher Statistical System) Statistical Software, developed in Utah, USA. The study data were assessed using descriptive statistical techniques, including calculation of mean and standard deviation. Additionally, the Shapiro-Wilk test was employed to evaluate the normality of the data. Repeated one-way analyses of variance and binary evaluations with a Bonferroni correction were used for comparisons among the time points of more than two normally distributed quantitative variables. A paired sample t-test was used to evaluate the differences between the each time point during the study period. A significance level of less than 0.05 was deemed significant for the conducted analysis.

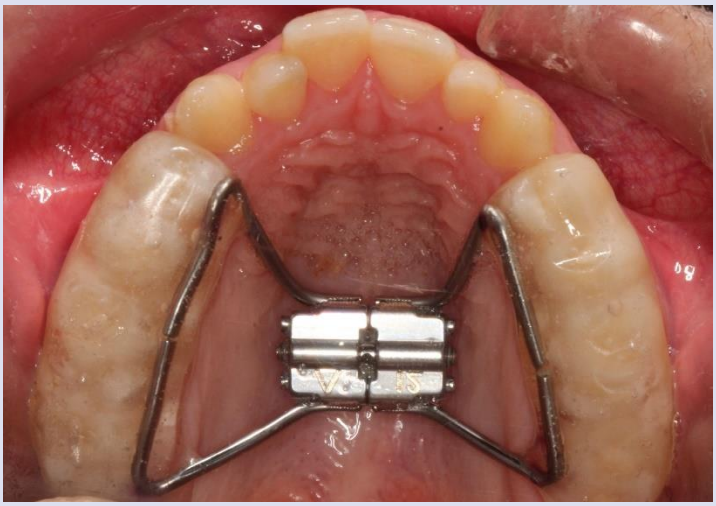

Figure 1: Bonded rapid maxillary appliance (Hyrax screw, Dentaurum, Germany).

Figure 2: Measurement of optic nerve sheath diameter ultrasonographically.

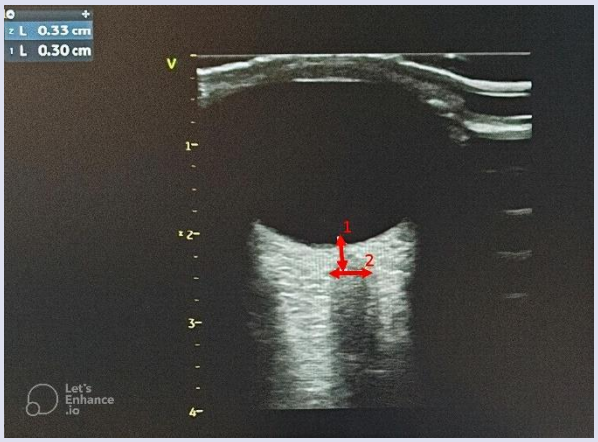

Figure 3: Ultrasonographic image of optic nerve sheath diameter measurement (1.Distance behind the optic disc where the optic nerve sheath diameter (ONSD) is measured in its width, 2. ONSD measurement).

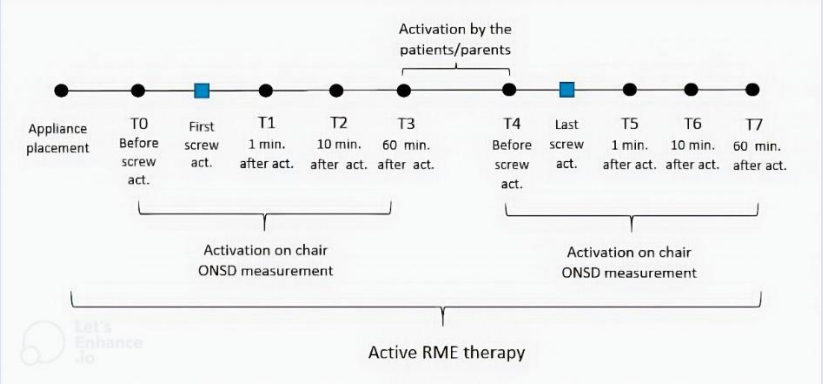

Figure 4: Study design and measurement times.

Results

A cohort of 25 individuals participated in the study. The average age was 13.10 ± 1.20 years. Detailed demographic information of the patients can be found in Table 1.

Mean ONSD measurements were 0.35 ± 0.03 at T0, 0.37 ± 0.05 at T1, 0.36 ± 0.05 at T2, 0.35 ± 0.04 at T3, 0.33 ± 0.031 at T4, 0.36 ± 0.04 at T5, 0.34 ± 0.03 at T6, and 0.34 ± 0.03 at T7. The results of the pairwise comparisons showed that the ONSD values, 1 minute after screw activations (T1, T5), were significantly higher compared to the ONSD values before screw activations (T0, T4) ($p = 0.001$). Nonetheless, a notable and statistically significant decline was observed in the ONSD measurements taken 60 minutes after screw activation (T3, T7) in comparison to the ONSD measurements taken 1 minute after activation (T1, T5). In contrast, changes in ONSD values at both 10 minute and 60 minutes following screw activations (T0-T2, T0-T3, T4-T6, T4-T7) were not statistically significant ($p>0.05$). Additionally, no significant alteration was observed between the initial (T0) and final (T7) ONSD measurements ($p>0.05$). Detailed comparisons of the mean ONSD values and corresponding descriptive statistics at different time points can be found in Figure 5 and Table 2.

After comparing the heart rate, SpO2, and MAP values of the patients before the initial activation (T0) with those after the first activation (T1), no statistically significant differences were observed ($p>0.05$). Similarly, there were no significant variations in these vital parameters between T4 and T5. A comprehensive depiction of the potential influence of hemodynamic parameters on ICP can be observed in Figure 6.

A total of 3 patients had severe pain score during screw activation at both the initial and final appointments of active RME treatment. 22 patients did not have mild or moderate pain score during the activations. Pain scores of the patients were insignificant at T0 and T1, and T4 and T5 ($p>0.05$).

Table 1. Demographic data of the patients

	Min - Max	Mean ± SD
Age	11 - 15	13.10 ± 1.20
Gender	Number	%
Female	15	60.0
Male	10	40.0

Table 2. Comparison of optic nerve sheath diameter (ONSD) values at each time point

	Mean ± SD	Median (Min/Max)	P
T0-T1	0.03 ± 0.03	0.03 (-0.03/ 0.10)	0.001
T4-T5	0.02 ± 0.02	0.02 (0/ 0.07)	0.001
T1-T3	-0.03 ± 0.02	-0.02 (-0.08/ 0.02)	0.001
T5-T7	-0.02 ± 0.03	-0.02 (-0.06/ 0.03)	0.007
T0-T2	0.02 ± 0.03	0.01 (-0.03/ 0.09)	0.113
T0-T3	0.01 ± 0.03	0.01 (-0.05/ 0.07)	1.000
T4-T6	0.01 ± 0.01	0.01 (-0.01/ 0.04)	0.110
T4-T7	0 ± 0.02	0 (-0.03/ 0.05)	1.000
T0-T7	-0.01 ± 0.03	0 (-0.08/ 0.03)	1.000

Repeated Measure Test & Post-Hoc Bonferroni Test *p < 0.05

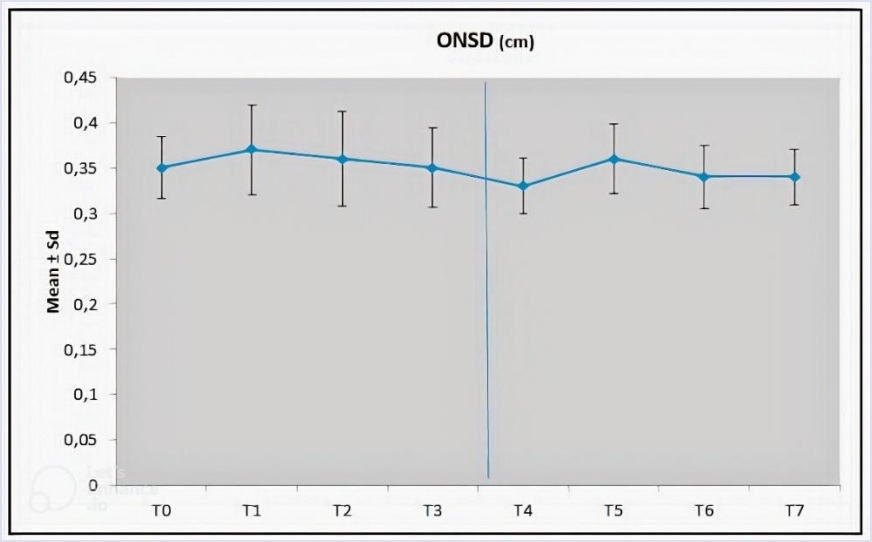

Figure 5: Changes in optic nerve sheath diameter (ONSD) values at each time point.

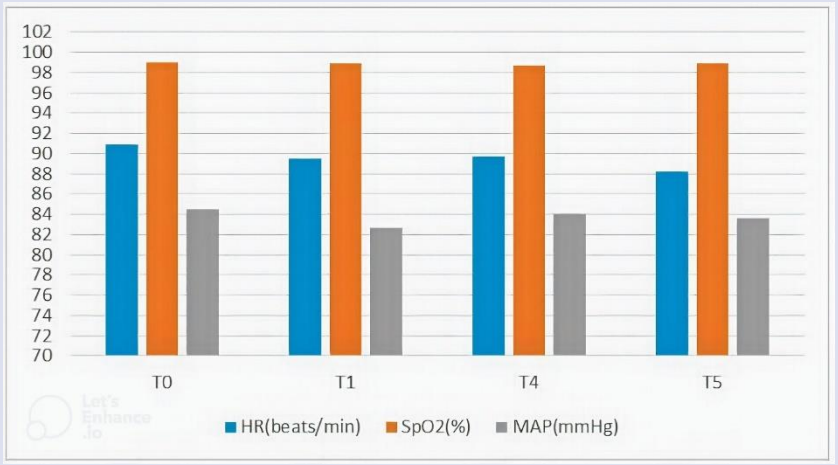

Figure 6: The hemodynamic parameters (HR, Spo2, MAP) before and after screw activation periods.

Discussion

In this current prospective observational study, we discovered that the activation of the screw during active RME therapy leads to a noteworthy enlargement of the optic nerve sheath diameter and a subsequent compensation in ICP over the course of one hour. In our extensive literature search, no previous study assessing intracranial pressure through ultrasonography-guided ONSD measurements in patients undergoing RME therapy was identified.

RME therapy has been initially applied during circumpubertal age, and the closure of median palatal suture usually occurs at ages 11 to 16 years.[13,14] *Narula and colleagues*[15] reported a substantial correlation between the CVM index and the maturation of sutures. The participants in the current study had an average age of 13.10 ± 1.20, and their skeletal maturity ranged from CS 1 to CS 3 stages, as determined by the CVM index. Considering the disruptive factors of midpalatal suture fusion and possible high stress accumulation on craniofacial structures, patients going through the CS 4 stage and above were excluded from the study.

Enlarged ONSD is a robust predictor of raised ICP.[10] Several studies have documented the rapid changes in intracranial pressure (ICP) associated with immediate variations in ONSD.[10] Several methods have been introduced in prior research to measure raised ICP.[16,17] Invasive methods such as intraventricular catheter are the gold standard for ICP monitorization.[2] However, these methods can lead to complications such as bleeding and infection.[18] In our investigation, we utilized ultrasound imaging to measure optic nerve sheath diameter (ONSD) and assess the impact of active RME therapy on intracranial pressure (ICP). Notably, no complications were detected throughout the active treatment procedure.

Several researches have hypothesized that the forces generated by palatal expansion have the potential to extend to deeper anatomical structures. Previous studies have highlighted the proximity of these structures to critical and delicate vessels that are crucial for cerebral blood supply.[6,11,19] Importantly, the strain exerted around various cranial base foramina can lead to the enlargement of adjacent blood vessels. [7]

In a study on rabbits by *Li et al*[6], it was reported that anatomical stresses on craniofacial structures during RME therapy cause cerebral hemodynamic changes, including increases in CBF and CBV. In a case report, it was reported that RME treatment may cause raised intracranial pressure in patients with impaired venous drainage and pseudotumor cerebri syndrome (PTCS) by affecting cerebral venous circulation on MRI.[7]

In our current investigation, there was a notable increase in ONSD one minute after screw activations (T1, T5) in comparison to the ONSD values prior to screw activations (T0, T4) (p=0.001). While the precise mechanisms underlying the elevation of ICP are not yet fully understood, we speculate that this phenomenon might be attributed to varying levels of stress and cerebral hemodynamic alterations within the intracranial region.

Our study revealed a significant decrease in ONSD values 60 minutes after screw activation (T3, T7) when compared to the ONSD values 1 minute after activation (T1, T5) (p<0.05). This observation suggests the involvement of the brain's autoregulatory capacity and mechanisms. A prior investigation utilizing perfusion CT reported an initial increase in CBF during the initial stages of RME therapy followed by a return to normal levels.[6]

When comparing the ONSD values immediately before activations (T0 - T4) to those 60 minutes after activations (T3 - T7), no statistically significant differences were observed (p>0.05). Furthermore, the correlation between the initial (T0) and final (T7) ONSD values was also found to be non-significant (p>0.05). These findings suggest that the optic nerve sheath diameter, which serves as an indicator of ICP, increases during activation and subsequently returns to its baseline value within an hour. Moreover, repetitive activations do not appear to have an impact on this process.

Researchers have pointed out that pain raises ICP by increasing blood pressure and causing respiratory irregularity.[20,21] In our study, patients' perception of pain during screw activation was evaluated by a four-category verbal rating scale (VRS-4) at T1 and T5. In both initial and final appointment of active RME treatment only 3 patients had noticeable pain during screw activation, and the pain scores were insignificant at T0 - T1 and T4 - T5 (p>0.05). Therefore, we suspect that pain was not the reason for the raised ICP.

In the study, ICP measurements were performed only during a short period of the active RME treatment, and only among adolescents. There is therefore a need to investigate the long-term effects of total RME treatment on intracranial pressure, and to assess these factors in adults.

Conclusions

This study has contributed to the assessment of rising intracranial pressure in patients undergoing RME treatment. Raised ICP is likely normalized by autoregulatory mechanisms of the brain and body for healthy people. We propose that healthcare professionals should take into account the potential hazards associated with elevated ICP, and ask patients about previous conditions before RME treatment, especially for adolescent patients.

Acknowledgements

Not applicabble.

Conflicts of Interest Statement

All authors declare that no conflict of interests.

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