Maxillary Pain in Correlation to Changes in Intramaxillary Sinus Pressure

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ABSTRACT

Aim: To find out the role of intramaxillary sinus pressure changes in the pathogenesis of headache and facial pain in cases of rhinosinusitis, in order to improve our understanding to the nature of rhinogenic or sinogenic headache.

Materials and methods: A cross-sectional controlled study was carried out on 195 patients with maxillary sinusitis (study group) and 12 control normal volunteers. The study group then divided based on the presence of sinus pain. Under local anesthesia, all participants were subjected to anterior antral puncture and intramaxillary sinus pressure measurement using the manometer used in central venous pressure measuring. The value of intramaxillary pressure was measured and recorded.

Results: The intramaxillary pressure median and range were 0 (−7.1 to 8) in the study group and 0 (−0.5 to 0.4) in the control group. In the study group, we had found 77 out of 195 patients (39.5%) were with negative intramaxillary pressure values and 62 patients (31.8%) were with positive intramaxillary pressure values. In subgroup A (pain), the median and ranges were −3.8 (−7.1 to 8) compared to 0 (−1.9 to 2.7) in subgroup B (no pain) (p = 0.002). There was no one in the subgroup A with 0 cm water intramaxillary pressure, while in subgroup B, 56 patients (54.9%) were with 0 cm water intramaxillary pressure.

Conclusion: The changes in the intramaxillary pressure play a significant role in producing facial pain in cases of sinusitis. Pain was significantly related to both positive and negative pressure values.

Clinical significance: Sinusitis may cause headache and facial pain, and in this case, intramaxillary pressure change is one of the main mechanisms causing this sinogenic headache or facial pain.

Keywords: Facial pain, Headache, Intramaxillary pressure, Sinogenic headache, Sinusitis.

INTRODUCTION

Facial pain remains a challenge for both physicians and patients regarding diagnosis and treatment. It is associated with significant morbidity and high levels of healthcare utilization and often diagnosed by exclusion. Patients complaining from facial pain usually undergo multiple consultations and specialists’ visits and receive different treatments, including surgery.

The management of patients with sinus headache, fullness, or facial pain is very important in the field of otolaryngology. From a clinical point of view, we should differentiate “sinogenic” from “nonsinogenic” facial pain to avoid wrong treatment.

The term “sinogenic” facial pain usually suggests a pathogenic relationship between facial pain and rhinological disorders, and several physiopathological theories have been postulated.

According to the classification of International Headache Society, chronic sinusitis is not validated as a cause of headache and facial pain unless relapsing into an acute stage. Most patients presenting with a symmetrical temporal or frontal headache, sometimes with an occipital component, have tension headache.

A “convergence hypothesis” has been postulated, suggesting that headache might be a rebound mechanism that occurs when a headache-provoking environmental trigger stimulates the central nervous system. Sinus headache may result from activation of the trigemino-vascular system by trigeminal afferents in the nasal and sinus mucosa.

Conclusion

In addition, the pressure difference created across the obstructed sinus ostia, underlying osteitis, and presence of bacterial toxins, may contribute to the etiology of headache and sinogenic facial pain and influence sensory nerve function.

When the maxillary sinus ostium obstructed, the first phase occurs by transient positive pressure due to trapping of the air inside the sinus or by nitrous oxide production during inflammation. The second phase then is caused by the respiratory function of the sinus mucosa. In a process similar to the middle ear in eustachian tube obstruction, oxygen passes through the sinus mucosa to the circulation causing negative pressure inside the sinus.

This study aimed to find out the role of pressure changes inside the maxillary sinus in the pathogenesis of headache and facial pain.
in cases of rhinosinusitis, in order to improve our understanding to
the nature of headache of rhinogenic or sinogenic origin.

Patients and Methods
This cross-sectional observational controlled study was carried out during the period from March 2018 to December 2020. In this study, 209 adults were enrolled; 195 patients in the study group, diagnosed with maxillary sinusitis based on clinical and radiological findings, were selected from out-patient clinic and 12 control normal volunteer participants.

We excluded from the study patients younger than 18 years old and patients with general diseases such as diabetes, kidney diseases, bleeding disorders, and uncooperative patients.

Detailed history was obtained including the demographic data and history of the condition stressing on symptoms of headache and facial pain. Full otorhinolaryngology examination of ear, nose, and throat was completed. Nasal endoscopy was performed to reveal mucopurulent or purulent nasal discharge which may be anterior or posterior, mucosal edema or congestion, also polyps or occlusion of ostomeatal complex, and/or sphenoid-ethmoidal recess. CT scan was done to confirm sinuses opacification, ostomeatal complex, and/or sphenoid-ethmoidal recess occlusion. Volunteer participants were free from any medical condition and had neither sinus pain nor rhinosinusitis evidence by clinical assessment, endoscopic evaluation, or radiological imaging. The small number of the control group in relation to the study group number is due to the long expensive and unusual intervention to the volunteers.

The study group then divided based on the presence of sinus pain which had to fulfill the following criteria to be considered: facial pain or pressure of a dull, constant, or aching sort over the affected sinuses and may worsen when the affected person bends over or when lying down. Pain often starts on one side of the head and progresses to both sides. It had to be of mild-to-moderate intensity and last 3 days or more. It might be unilateral or bilateral according to the site of sinusitis. Frequently, it is associated with nasal blockage, congestion or discharge, and variation of smell. Subgroup A included 93 rhinosinusitis patients with sinus pain, while subgroup B included 102 rhinosinusitis patients without the defined sinus pain.

Procedure
All cases were subjected to anterior antral puncture and pressure measurement. The patient sat on the examination chair; sometimes the head was moved forward or backward to attain best manipulation position of the patient.

Trap door valved trocar and cannula “Sklar-Sklartech 5000 Standard Trap Door Cannula 35 mm diameter” were used to penetrate anterior maxillary sinus wall (when the trocar removed, the valve automatically closes to keep the air and pressure unchanged). After administration of general analgesia (paracetamol) and local anesthesia to its anterior wall at the canine fossa with lidocaine and adrenaline 2%, the trocar and cannula were inserted in the anterior maxillary wall of the affected sinus through the canine fossa in the subgroup with sinus pain and to the left sinus in other participants. Then, an adapter was inserted, one side to the cannula and the other side to central venous measuring manometer to measure the pressure inside the maxillary sinus.

The manometer set consisted of stand, reservoir containing fluid (sterile water), U-shaped manometer, three-way valve connected to the manometer reservoir and to the patient connection through an adaptor attached to the cannula. The manometer was fixed to the stand in the same horizontal level of the cannula inside the maxillary sinus, and the fluid level is adjusted to zero assuring there were no air bubbles included in the tube; then, the connection of the patient side to the manometer is secured (Fig. 1).

After switching the valve of the manometer, if the pressure inside the sinus is positive, then the fluid in the manometer is raised up giving above zero reading and vice versa. Readings were taken after the movement of the fluid had stopped completely. The value of intramaxillary pressure was measured by centimeter of water.

Results
The intramaxillary pressure median and range were 0 (−7.1 to 8) in the study group (195 patients with sinusitis) and 0 (−0.5 to 0.4) in the control group (12 participant). In subgroup A (93 patients with pain), the median and ranges were −3.8 (−7.1 to 8) compared to 0 (−1.9 to 2.7) in subgroup B (102 patients without pain) (p = 0.002) (Table 1).

Figure 2 shows the number of cases with negative, positive, or zero intramaxillary pressure in each group. As shown in Figure 2, there was a statistically significant difference between subgroup
**Table 1:** The intramaxillary pressure medians and ranges in each group

<table>
<thead>
<tr>
<th>Study group (n = 195)</th>
<th>Subgroup A (pain) (n = 93)</th>
<th>Subgroup B (no pain) (n = 102)</th>
<th>Control group (n = 12)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intramaxillary pressure (cm water)</td>
<td>0 (−7.1 to 8)</td>
<td>−3.8 (−7.1 to 8)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0 (−1.9 to 2.7)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0 (−0.5 to 0.4)&lt;sup&gt;a,b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Data are presented as medians and ranges. Kruskal-Wallis test was used. Post hoc analysis was done, and different letters indicate significant pair. All post hoc were Bonferroni-adjusted.

**Fig. 2:** The percent of cases with negative, positive, or zero intramaxillary pressure in each group.

**Table 2:** The intramaxillary pressure medians and ranges for those with negative pressure in the study group

<table>
<thead>
<tr>
<th>Pain (n = 59)</th>
<th>No pain (n = 18)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intramaxillary pressure (cm water)</td>
<td>Median (range)</td>
<td>Median (range)</td>
</tr>
<tr>
<td>−4.9 (−7.1 to −0.3)</td>
<td>−0.4 (−1.9 to −0.2)</td>
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</tbody>
</table>

Mann-Whitney U test was used.

**Table 3:** The intramaxillary pressure medians and ranges for those with positive pressure in the study group

<table>
<thead>
<tr>
<th>Pain (n = 34)</th>
<th>No pain (n = 28)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intramaxillary pressure (cm water)</td>
<td>Median (range)</td>
<td>Median (range)</td>
</tr>
<tr>
<td>3.6 (0.6–8)</td>
<td>0.5 (0.2–2.7)</td>
<td></td>
</tr>
</tbody>
</table>

Mann-Whitney U test was used.

A and subgroup B, denoting that negative intramaxillary pressure was significantly associated with pain 59/93 (63.4%) in subgroup A (with pain) compared to 18/102 (17.6%) in subgroup B (without pain) (p <0.001). There was no one in the subgroup A with 0 cm water intramaxillary pressure, while in subgroup B, 56 patients (54.9%) were with 0 cm water intramaxillary pressure.

In the study group, we had found 77 out of 195 patients (39.5%) were with negative intramaxillary pressure values, 59 out of those 77 patients were with pain, and their pressure median and range were −4.9 (−7.1 to −0.3). Eighteen patients out of those 77 patients were without pain, and their pressure median and range were −0.4 (−1.9 to −0.2) (p <0.001) (Table 2).

In the study group, we had found 62 out of 195 patients (31.8%) were with positive intramaxillary pressure values, 34 out of those 62 patients were with pain, and their pressure median and range were 3.6 (0.6–8). Twenty-eight patients out of those 62 patients were without pain, and their pressure median and range were 0.5 (0.2–2.7) (p <0.001) (Table 3).

**Discussion**

Sinusitis is overdiagnosed as a cause of headache because of the belief that pain over the sinuses must be related to the sinuses. There is a controversy whether nasal obstruction can lead to chronic headache. Paradoxically, sinus disease also tends to be underdiagnosed, as sphenoid sinus infection frequently is missed.

 Conditions like atrophy of sinus membranes, hypertrophy of nasal turbinates, mucosal contact, and deviation of nasal septum are not sufficiently validated as causes of headache. Chronic sinusitis is only validated as a cause of headache or facial pain when relapsing into an acute stage.

Jankowski et al. said that the role of the paranasal sinus ostium dysfunction in facial pain should now be considered. Complete absence of opacity in a closed sinus does not exclude pain due to sinus disease and questions the more conventional concepts concerning ventilation and drainage of the paranasal sinuses.
We are convinced that cases of sinusitis do not necessarily cause facial pain, but when sinogenic headache or facial pain is encountered, the aim of our study was to clarify whether pressure changes inside the maxillary sinus have a role in the pathogenesis of headache and facial pain.

Our results showed that 102 out of 195 patients in the study group (52.3%) did not present with facial pain or headache, and this is consistent with many previous studies stated that it is not common for facial pain/theadache to present in the cases of sinusitis unless during an acute exacerbation.10-12

In the comparison between subgroup A (93 patients with pain) and subgroup B (102 patients without pain) regarding intramaxillary pressure median and ranges, there were statistically significant difference (p = 0.002). Also, there was a statistically significant difference between subgroup A and subgroup B as regards the number of cases with negative, positive, or zero intramaxillary pressure in each group (p <0.001). There was no one in the subgroup A with 0 cm water intramaxillary pressure, while in subgroup B, 56 patients (54.9%) were with 0 cm water intramaxillary pressure.

Bertrand and Robillard13 found excellent correlation (82.09%) when the morphologic appearance of the ostium had been studied in conjunction with the sinusomanometric findings, but they did not study the relationship between sinogenic headache and intramaxillary sinus pressure.

The results of our study declared that the changes in the intramaxillary pressure play a significant role in producing facial pain in cases of sinusitis presented with pain. Pain was significantly related to both positive and negative pressure values, also the subgroup of sinusitis without pain showed significantly smaller range of pressure changes than that of the subgroup with pain.

The limitation in our study was the rather small number of the control group, but by referring to the nature of the procedure followed, we found a great deal of difficulty in subjecting volunteers for such a procedure. However, during the study period, a number of 12 volunteers were actually accepted and we saw that it was not logical to ignore their results even if their number is small.

Conclusions
Closed ostium is the main pathology sequence which is responsible for acute and chronic rhinosinusitis causing sinus pain. Sinus pain is highly correlated to intramaxillary pressure changes either negative or positive pressures. Nasal pathology that does not affect the intramaxillary pressure does not cause sinus pain.

Clinical Significance
There is a need to understand whether headache and facial pain in cases of rhinosinusitis are related to sinus pressure imbalances. Sinusitis may cause headache and facial pain, and in this case, intramaxillary pressure change is one of the main mechanisms causing this sinogenic headache or facial pain.

Ethics and Consent
The study was performed in accordance with the Helsinki Declaration of 1975 and its amendments, and the study protocol was approved by the corresponding local ethical committee. All participants signed their written informed consent to participate in the study.

Availability of Data and Material
The data that support the findings of this study are available from the corresponding author upon reasonable request.

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References