



## Velopharyngeal Insufficiency after Dental Trauma: A Case Report

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### Abstract

**Objective:** Velopharyngeal insufficiency is usually defined as failure of the velopharyngeal sphincter (mainly: velum, lateral and posterior pharyngeal walls) to separate the oropharynx from the nasopharyngeal space during speech and deglutition resulting in swallowing and speech difficulties with adverse effects on communication skills and the psychological status of the patient.

**Methods:** We present the first case of VPI after injudicious dental extraction that resulted in extensive, left side, full thickness palatal trauma jeopardizing the posterior part of the hard palate, the adjacent area of the soft palate, the inferior and posterolateral maxillary walls and the pterygoid plates.

**Results:** After discussion of modalities of treatment, the non-surgical approach was the preferred choice of the patient, and a palatal prosthesis was designed.

**Conclusion:** Injudicious dental extraction could be considered as a cause of VPI. Prosthetic rehabilitation might be a simple choice leading to satisfactory results for the patient.

**Keywords:** Traumatic velopharyngeal insufficiency; Velopharyngeal insufficiency after dental trauma

### Introduction

Velopharyngeal Insufficiency (VPI) is usually defined as failure of the velopharyngeal sphincter (VPS; namely: velum, lateral and posterior pharyngeal walls) to functionally separate the oropharynx from the nasopharyngeal space during speech and deglutition [1]. Hyper nasality and regurgitation of food and fluids are the main problems [2]. In the literature, acquired VPI is usually described after surgery e.g. post-adenoidectomy especially in cases of sub mucosal cleft palate or after cleft palate repair; or after surgical resection of a tumor of the palate. By literature review, we could not find dental trauma as a cause of VPI, which was only referred to postoperative trauma of the palate especially for oncological etiologies [3]. To the best of our knowledge, this is the first case of velopharyngeal insufficiency (due to extensive tissue trauma) after tooth extraction. Different modalities of treatment were mentioned.

### Case Presentation

A previously healthy 73-years-old male was referred to the ENT clinic, from the Neurology Department with a provisional diagnosis of unilateral palatal weakness of unknown etiology; presumably viral or localized (undetectable) vascular suggestions, for more than 3 months. The general and neurological examinations revealed no systemic diseases and all his laboratory data were within normal values except for a low hemoglobin level (7.1 gm/dl). The report of the brain MRI was negative and offered no explanation for his complaint. At the ENT clinic, data were reviewed and everything was fine except the severe nasality of voice. The patient had regurgitation of fluids and food that was improved with time but still a problem. The patient mentioned earache and egophony. History and patient's speech were not clear. Neck examination was negative for lumps; also nose and ear showed no abnormality. With oral examination, a large (about 2.5 cm × 3.5 cm) full thickness, triangular defect was detected at the posterior part of the hard palate on the left side, the gingival and the adjacent part of the soft palate (Figure 1). At that time, the patient mentioned a left upper wisdom tooth extraction at a traditional, non-medical dental center, which was followed by severe pain and bleeding. Bleeding lasted for 3 weeks and lessened gradually. The patient denied any other traumatic issues.

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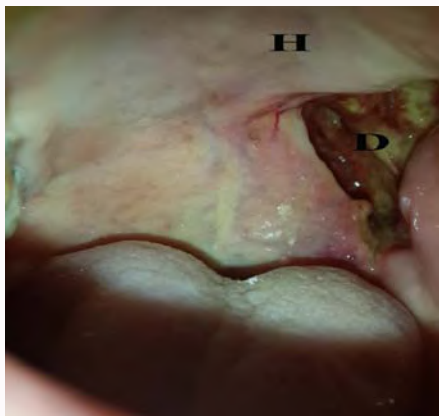


Figure 1: Oral photo showing palatal defect; H: hard palate, D: defect.

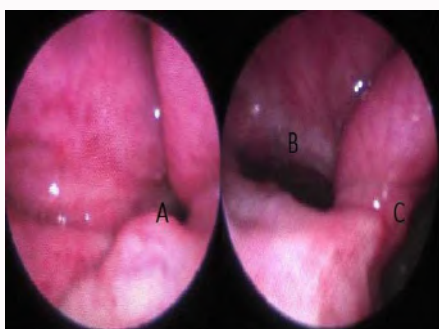


Figure 2: VPV during speech (left) and rest (right); A: (left sided insufficiency), B: (velopharyngeal opening) and C: (gap of dental trauma).

High fidelity audio-recordings including a sample of patient's speech was assessed by 3 phoniatricians blindly and their perceptual assessment revealed: moderate hyper-nasality, mild to moderate imprecision of anterior sounds e.g., (t, d, l and r) with compensatory mechanisms in the form of severe pharyngeal articulation of (s, z and s, h) sounds and facial grimace. There was audible nasal emission of air. Overall speech showed moderate unintelligibility.

Nasoendoscopy revealed a left full thickness palatal defect, through which the endoscope could pass freely to the left pterygopalatine fossa; and the oral cavity and upper airway were seen. No suspicious masses or ulceration were detected, and the margins of the defect showed healing edges. It showed also mild VPI (mainly on the left side) during speech (Figure 2). CT showed a large defect at the left posterior end of the hard palate, the posterior part of the floor, the posterior wall, and the posterior part of the lateral wall of the left maxillary sinus, and the whole medial pterygoid plate i.e. left maxillary tuberosity trauma (Figure 3 and 4). Treatment options were clearly discussed with the patient in details, as regard operative (including hazards and limitations) and conservative managements. The patient refused any surgical intervention, and then he was referred to the dental clinic for a palatal prosthesis. The protocol of the investigation has been approved by the Institutional Review Board, and that the investigators have obtained written informed consent from the patient.

**Discussion**

Velopharyngeal Insufficiency (VPI) is the inability of the VPS to close the velopharyngeal port during various oropharyngeal functions e.g. speech and deglutition. VPS is a functional sealing

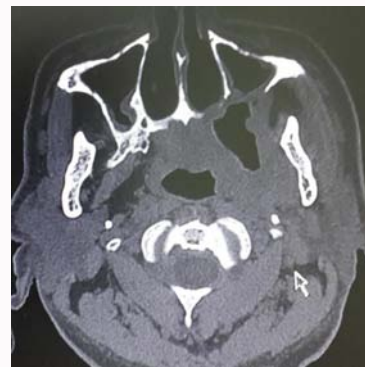


Figure 3: CT (axial cut) showing the defect area.



Figure 4: CT (coronal cut) showing the defect area.

area between the oropharynx and the nasopharynx, thus playing an essential role during deglutition, nasal breathing, and speech. The effect of VPI on speech and resonance ranges from mild speech distortion to a catastrophic disruption of speech intelligibility, leading to breakdown of the ability to communicate verbally with negative impacts on the psychological status of the patient [4,5]. The soft palate with the lateral and posterior oropharyngeal walls represents the anatomical components of the VPS. Proper functions of VPS are achieved through tissue integrity and the functional coordination of palatal and pharyngeal muscles. Velopharyngeal Insufficiency (VPI) indicates loss of tissues and results in nasal regurgitation and open nasal speech (rhinolalia aperta), and could participate in otologic complaints [6]. Loss of tissue integrity might be congenital e.g. cleft palate or acquired e.g. surgical resection of a tumor. By searching in the available internet websites, we could not find a dental trauma as a cause of VPI.

In the above mentioned case, an injudicious dental removal resulted in extensive, left side tissue loss involving the posterior part of the soft palate, adjacent parts of the hard palate and the maxillary tuberosity (pterygoid plates and the posterior walls of the maxillary sinus). Another surprising point is that poor patient did not consider post-extraction pain and bleeding (for few weeks) as a reason to seek a medical advice. No suspicious areas for malignancy or inflammatory granulomas that could explain a tissue weakness or vulnerability to dehiscence were detected. Reconstruction of the palatal defects could be considered a major challenge because of the functional consequences in this active area. The main goal of surgery is to maintain a functional separation between the oral and nasal/ nasopharyngeal cavities. Options entail surgical and conservative tools. Surgical repair of the defect could be performed via different flaps: local (V-Y push back and buccal flap); regional (the

buccinator and the pedicled temporalis muscle flaps) and free flaps (radial forearm free flap). Temporary palatal obturators could be used to support the flaps in the postoperative period to avoid flap drooping and wound dehiscence [7-9]. After discussing details and various options with the patient, he chose the non-surgical approach. Then, he was referred to the dental clinic for a custom-made prosthesis. The aim of the prosthetic rehabilitation in this patient was to fabricate well-fitted obturator prosthesis in order to close communication of the oral cavity with nasal/nasopharyngeal cavities, replace lost soft/bone tissue and improve oral function (swallowing and hyper nasality) at the lowest possible cost in a non-invasive technique. After using the palatal obturator, food regurgitation became minimal. Speech assessment showed mild hyper-nasality, mild imprecision of sounds and mild pharyngeal articulation. There was audible nasal emission of air and intelligibility of speech improved. The patient was satisfied about swallowing and speech results at 6 months follow up.

### Conclusion

This is the first case report of post-traumatic velopharyngeal insufficiency, after dental extraction. Injudicious dental trauma resulted in extensive loss of tissue and affection of the VPS components. Prosthetic rehabilitation might be a simple choice leading to satisfactory results for the patient.

### Declaration of Conflicting Interests

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