REVIEW
FACIAL PLASTIC AND RECONSTRUCTIVE SURGERY
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Guidelines for the Initial Management of Acute Facial Nerve Palsy

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ABSTRACT

The aim of this study was to provide a concise review of international standards in the initial management of facial palsy. This culminates in guidelines and indications for referral to a tertiary facial nerve center. Facial nerve palsy is a relatively rare condition, and most practitioners outside specialized centers will only see a few cases per year. While most patients will ultimately show spontaneous and full recovery of facial nerve function, the importance of accurate diagnosis and early treatment cannot be underestimated. Incorrect diagnosis can be harmful to patients, resulting in worse facial function outcomes as a result of delay in diagnosis of occult neoplasms and failure to treat patients within a timely fashion. Management of facial palsy is complex and requires a multidisciplinary board with specific focus.

Keywords: Bell's palsy, facial movement disorder, facial paresis, facial weakness, guideline

Introduction

Facial palsy (FP) includes the entire spectrum of facial movement disorders, including flaccid facial palsy (FFP), facial paresis, and postparalytic facial palsy. Facial palsy is a severe condition with a serious impact on both functional and cosmetic outcomes, resulting in a significant loss in quality of life (QOL).[1,2] Reversible Bell's type palsy is by far the most common etiology in acute unilateral FP, accounting for 60-80% of cases.[3] Timely diagnosis and treatment is key in recovery of facial nerve function. However, the diagnosis of Bell's palsy (BP) should be made with caution: literature suggests misdiagnosis rates may be as high as 20%. Moreover, these misdiagnoses are populated by benign and malignant tumors that require intervention.[4-7]

This article serves as a concise review of international standards in the initial management of FP and culminates in guidelines and indications for referral to a tertiary facial nerve center. The late management options for FP will be discussed in a subsequent article.

History and Physical Examination

Performing a thorough history is of utmost importance in evaluating patients with acute FP, with particular attention paid to time course of onset, progression, recurrence, associated symptoms, oncological history, head trauma, subjective hearing loss, and recent travel. Differential diagnosis of acute FP is provided in Table 1.

The time course of onset of facial weakness is critical. Facial weakness usually develops over a period of a few hours to 2 days, although some etiologies cause paralysis over weeks to months. Bell's palsy typically presents with a prodrome but fully evolves over 1- 3 days. Start of recovery in BP is expected to occur within 6 months after onset. If the onset of facial weakness exceeds over 72 hours, or there are no signs of recovery after 6 months, the diagnosis of BP is highly unlikely and alternative diagnoses should be considered. A history of infection or exposure to ticks may suggest infectious etiology while otovestibular symptoms such as hearing loss or vertigo indicate otitis or temporal bone trauma. Inflammatory conditions such as uveitis or parotitis and known autoimmune conditions may be important, especially in bilateral FP.

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Table 1. Causes of facial palsy

Infectious	Bell's palsy, Ramsay Hunt syndrome, acute otitis media, malignant otitis externa, cholesteatoma, skull base osteomyelitis, Lyme, and HIV
Developmental	Mobius, hemifacial macrosomia, pontine malformation, or anomaly
Benign tumor	Facial nerve schwannoma
Malignancy	Parotid carcinoma, head & neck carcinoma, and leptomeningeal carcinomatosis
Trauma	Temporal bone fracture, penetrating trauma, and birth trauma
Iatrogenic	Parotid, soft tissue, otologic, or orthognathic surgery
Systemic/autoimmune	Sarcoidosis, Melkersson–Rosenthal syndrome, Guillain–Barre, multiple sclerosis, amyloidosis, granulomatosis with polyangiitis, Sjögren, systemic lupus erythematosus, Behçet
Metabolic	Hypothyroidism, pregnancy

A complete head and neck examination is essential in the workup of FP. Palpation of the neck and parotid area to rule out neck or parotid masses, assessment of all cranial nerves to evaluate related nerve deficit, and skin examination may show vesicles (Zoster), a rash (systemic, autoimmune), malignant skin lesions or scars from prior surgery for skin cancer. Microotoscopy should be performed to rule out any vesicles in the ear canal and audiometry or tuning fork test can rule out other otologic causes.

Then the facial nerve function will be observed in rest[1] and motion and compared with the contralateral side. This examination will start superior to inferior: first, the patient is asked to raise the eyebrows to assess the action of the frontalis muscle.[2] When the upper third of the face is spared, a central cause should be excluded. Next, the examiner asks the patient to close the eyes softly and relax,[3] then close the eyes as hard as possible.[4] The inability to close the eye with minor effort is known as lagophthalmos, though often the eye can be closed with effort. By holding the eyelids open when the patient is asked to close them, the clinician can often notice the eye rolling upward. This is known as Bell's phenomenon. Severe lagophthalmos and the absence of Bell's phenomenon should be noted as they confer a higher risk of corneal drying exposure keratopathy, particularly when the patient is asleep. The patient is asked to smile without[5] and with showing teeth[6] to assess zygomaticus major muscle function. Finally, the patient is asked to pucker and press the lips together to evaluate orbicularis oris muscle function[7] and then to evert the lower lip to examine the lower lip depressors.[8] During these facial movements, it is important to check for synkinesis movements, for example, closing of the eye with lip pucker. The phenomenon of synkinesis is caused by aberrant reinnervation among facial nerve fibers in the late recovery of FP. Documentation of these facial movements using video- or photography at presentation and relevant follow-up visits is useful in evaluating recovery or interventions.

The House–Brackmann (HB) scale is one of the most commonly used tools for the clinical evaluation of facial nerve function. It is useful in the initial assessment and decision-making of FP. However, it is not sensitive to detect small changes and there is no specific evaluation of synkinesis. Therefore, HB scale is inadequate in evaluating rehabilitation or interventions. We prefer to use the eFACE, a clinician-graded facial function scale based on videography of dynamic movements.[8] It is a validated clinician objective assessment of facial paralysis, and in contrast to the widely used HB scale, it is sensitive for synkinesis and flaccid facial paralysis and facial reanimation interventions. We therefore recommend its use in staging, during follow-up, and after facial reanimation procedures.

Main Points

- Bell's palsy is a tentative diagnosis and should be made with caution.
- Facial nerve palsy requires close follow-up to confirm a time course consistent with BP.
- In traumatic or iatrogenic FP, urgent referral is important as treatment delay is a negative predictive factor.

Management of Facial Palsy

Guidance on the management of FP is summarized in the flowchart (Figure 2). An essential item in the initial assessment of FP is differentiation between an incomplete and complete paralysis of peripheral or central origin. Urgent referrals are critical not only to rule out stroke in the case of central paralysis but also for the potential surgical management of complete peripheral FP.

Trauma

The first and most urgent variable to be determined is whether the facial weakness resulted from trauma.

- In the case of facial weakness after penetrating trauma (with complete and immediate paralysis), surgical nerve repair by coaptation should be performed as soon as possible. Distal nerve axons undergo Wallerian degeneration (WD) over 3 days, so it is important to explore the wound in an expedient fashion. If the exploration is performed within 3 days, the distal nerve stumps can still be stimulated. This allows efficient localization of severed distal nerve stumps facilitating nerve co-adaptation.
- Iatrogenic FP can often occur after parotidectomy for benign lesions. It is important to bear in mind that anesthetic infiltration of the external auditory canal or

retro-auricular region can cause a temporary paralysis of the extratemporal portion of the facial nerve. Lidocaine can last for 4-5 hours, while marcaine can last over 12 hours. If the paralysis persists longer, it is important to discuss the case with the operating surgeon to understand if the facial nerve was put at significant risk of inadvertent division—revision parotid—main trunk not found—lumpectomy. If so, early exploration can be considered. However, the vast majority of cases are related to traction of the nerve. If the surgeon is sure of identification and preservation of the main trunk and branches through nerve stimulation, it is reasonable to administer oral steroids and await spontaneous recovery.

- In case of blunt head trauma, a computed tomography (CT) scan of the ipsilateral mastoid should be executed. If CT scan shows a fracture through the facial nerve, we would recommend urgent referral for consideration of decompression.
- If CT scan is normal, our policy depends on the HB grade of facial weakness. With an FP HB grade of maximum 4, we would recommend a conservative treatment and watchful waiting. In case of flaccid FP—HB grade 5-6—after blunt head trauma, we recommend urgent referral to a tertiary facial nerve center for consideration of decompression of the compromised segment. In this case, we recommend a gadolinium-enhanced MRI of the course of the facial nerve for localization of the affected segment to further guide surgical decompression if needed.

Central Facial Palsy

It is of vital importance to rule out a central FP that is characterized by palsy of the lower half of the face and non-involvement of the forehead. If this is the case, the patient should be referred urgently to rule out stroke.

Bell's Palsy

All initial diagnoses of Bell's are tentative. This point cannot be stressed strongly enough.

All patients with symptoms and signs consistent with BP must be closely followed to confirm signs of recovery. In BP, some recovery should be seen within 4 months and certainly within 6 months. The patient's recovery of facial function should then proceed either to full recovery or to a hypertonic/synkinetic postparalysis FP state by 12 months, which is roughly the expected time for facial nerve function to recover. Persistent signs of facial flaccidity after 12 months also effectively rule out the diagnosis of Bell's palsy. Making a definitive clinical diagnosis of BP at the first consultation therefore is dangerous and should be strongly discouraged for various reasons.

Once a diagnosis of BP is made, the patient is often then placed in a watch-and-wait holding pattern/scenario with no further active investigations being performed. Other specialist clinicians and GPs assume a benign diagnosis that will improve over some period of unspecified time. It is not infrequent in a facial nerve center setting to find patients being referred with "Bell's palsy or atypical Bells" following 3-4 years of monitoring with non-resolving flaccid facial paralysis. This is a disaster for the patient as the optimal therapeutic window has often passed.

It is recommended to use the term *Bell's-type picture* until the facial function can be measured at the above critical time points, to confirm a time course and a facial function recovery pattern consistent with Bells. Failure of recovery at 6 months should lead to a reappraisal of the diagnosis. The disease course is either that of a Bell's type picture or it is not. If it is not, then diagnosis remains unknown and requires active investigation. Recommended terminology is listed below:

1. Acute flaccid facial paralysis 1-6 months (Bell's-type picture or not—if other associated signs or symptoms that are inconsistent with Bell's palsy);
2. Persistent flaccid facial paralysis 6 months to 2 years (by definition cannot be of a Bell's-type picture)—cause unknown;
3. Chronic flaccid facial paralysis >2 years (by definition cannot be of a Bell's-type picture)—cause unknown.

It is not unsurprising that the literature suggests misdiagnosis rates as high as 20%. What is of more concern is that many of these misdiagnoses are populated by benign and malignant tumors.[4-7]

In cases where the history and physical examination are consistent with Bell's-type picture, no further investigations are mandatory. When history and physical examination are inconsistent with a Bell's-type picture (i.e., red flags for BP), imaging studies—preferably a gadolinium-enhanced MRI of the course of the facial nerve including the entire parotid gland and computed tomography of temporal bone and neck—are performed to rule out neoplastic or infectious causes like osteomyelitis. Blood tests are indicated in recurrent cases with suspicion for autoimmune or infectious etiologies like HIV. Serology tests are justified in endemic areas for Lyme. Consequently, underlying pathologies causing the FP should be managed accordingly.

Management of Incomplete Bell's Palsy

Peitersen[9] described spontaneous full recovery of BP in about 70% of all patients. Full recovery was significantly associated with less severity of weakness, early first signs of recovery, and young age. This study also stated that facial function recovered fully in 85% of patients within 3 weeks and in the remaining 15% after 3-5 months.

For medical treatment, the single most important factor affecting the outcome is delay in treatment, so early diagnosis and treatment are key. The following guidelines for medical management are specific for BP and were developed in accordance with the methods proposed by the GRADE Working Group.[10]

- Corticosteroids

Literature review comparing oral steroids versus placebo shows strong evidence for the use of oral steroids in early FP.[11] Corticosteroids reduce the risk of unsatisfactory facial recovery and this risk reduction is more powerful in patients with severe FP. Oral steroids should be started in the first 72 hours after the onset of symptoms. After 14 days of onset of

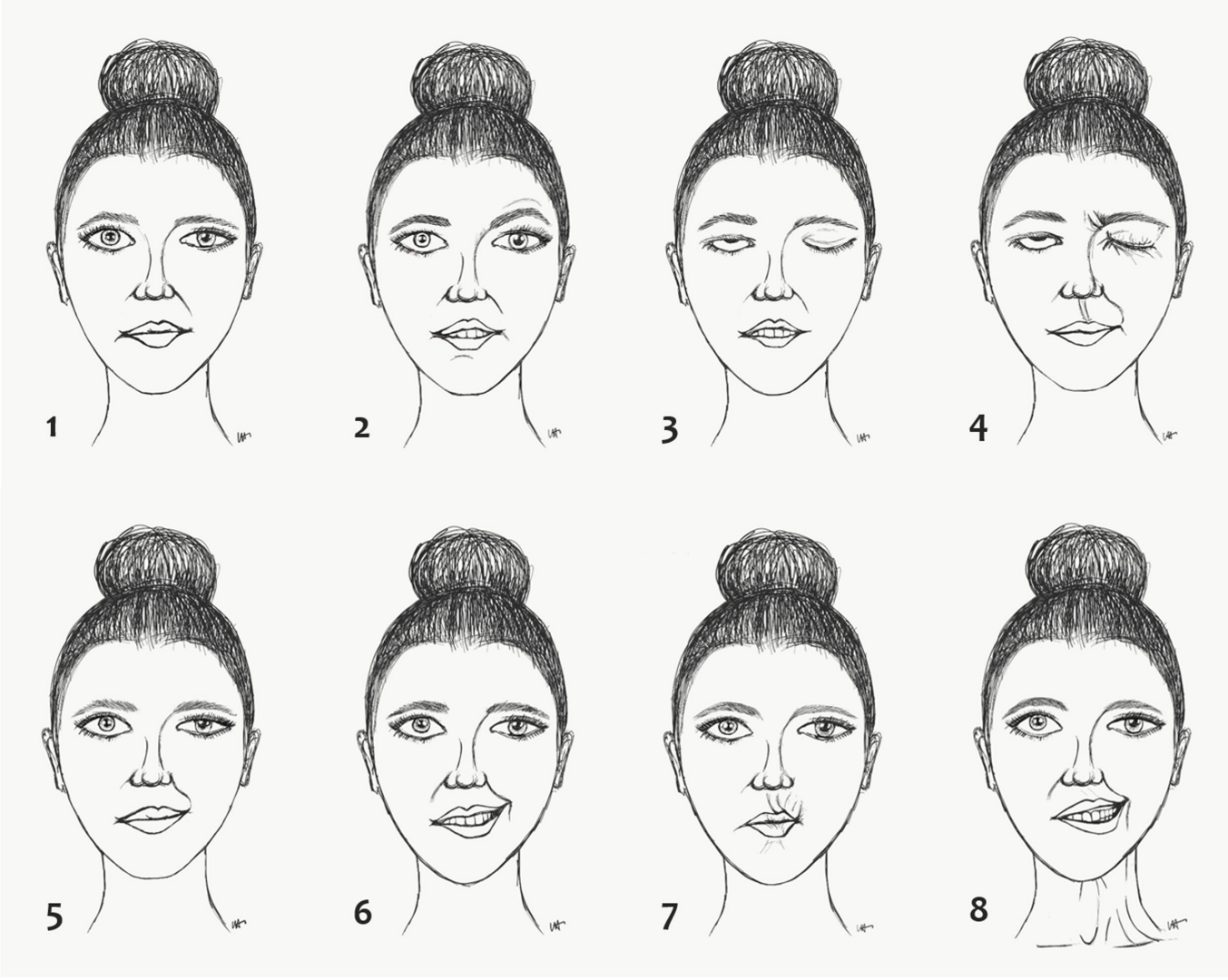

Figure 1. Facial movement examination.

symptoms, no treatment will have any impact on recovery. Most studies used prednisolone 60 mg per day for the first 5 days followed by a 5-day taper, usually this short-term dosing is well tolerated. Intravenous delivery of steroids was not shown to be superior to oral.

● Antivirals

There is strong evidence that monotherapy with antivirals has no role in BP.[12] However, a Cochrane review showed that combining steroids with antivirals was beneficial compared to monotherapy with steroids.[13] Antivirals are contraindicated in pregnancy and liver or kidney failure, and valacyclovir or famciclovir are preferred over acyclovir because of higher oral bioavailability. There is no consensus on dosage, but we recommend valacyclovir 3 g/day over 5 days or famciclovir 1 g/day over 5 days. The benefit of antivirals after 72 hours after onset of symptoms is arguable except in immunocompromised cases where it is recommended.

● Corneal protection

Closure of the eye is compromised, resulting in lagophthalmos, because normal retraction of the levator palpebrae superior muscle, innervated by the oculomotor nerve, is unopposed due to the paralysis of the orbicularis oculi muscle. This may result in corneal epithelial lesions, exposure keratopathy, ulceration, and in extreme cases, corneal perforation. In addition, proximal facial nerve lesions or BP can impair the parasympathetic innervation of the lacrimal gland compounding the risk of corneal dryness and lesions. Patients are encouraged to use protective glasses and regular use of natural tears during the day and a thicker ointment at night. Night-time requires particular attention as there is no active tear production at night, and the cornea can easily dry and ulcerate, particularly in the absence of the Bell's phenomenon. Careful taping of the eyelids may help in protecting against exposure keratopathy. Again, clinical examination for associated cranial nerve dysfunction is essential, and exposure keratitis in patients with combined facial and trigeminal dysfunction can be painless; therefore, we recommend urgent referral of these patients to a tertiary referral facial nerve center as these patients are particularly vulnerable to corneal blindness.

● Physiotherapy/electrical stimulation/acupuncture

There is no evidence for adding physiotherapy or electrical stimulation in the early management of FP.[14] It does not provide any benefit, to the contrary, it may facilitate aberrant

reinnervation and excess motor unit recruitment, favoring abnormal movement patterns, synkinesis, mass movements, and hypertonic areas. Systematic reviews by Pereira et al[15] and La Touche et al[16] suggest that physical therapy might prevent and decrease synkinesis in longstanding facial paralysis. However, a Cochrane analysis by Teixeira et al[17] was unable to find a significant benefit or harm from any physical therapy.

Complete Facial Palsy

In case of sudden and complete FP or sudden flaccid facial paralysis, we recommend referral to a tertiary facial nerve center for further investigation to consider urgent surgical decompression. In the absence of any red flags or underlying pathology, electrodiagnostic testing is indicated to stratify those patients for nonsurgical versus surgical management.

Electrodiagnostic Testing

Electroneuronography (ENoG) and electromyography (EMG) are the 2 most reliable electro-physiological tests currently. These tests provide prognostic information for likelihood of recovery in patients with complete paralysis and help identify those who might benefit from surgical decompression in the acute setting. In patients with incomplete paralysis or early recovery, electrodiagnostic testing is not indicated.

Electroneuronography measures muscle action potentials evoked by supramaximal stimulation of the facial nerve by placing a stimulating electrode at the stylomastoid foramen and a recording electrode at the nasolabial groove.[18,19] The amplitude of the reaction is compared between the affected and normal sides. The result is expressed as a percentage, estimating the relative proportion of nerve fibers that have undergone WD. Electroneuronography is not performed before the fourth day of paralysis because WD does not occur in the first 3 days after the pathological event. Patients with degeneration of more than 90% on ENoG in the first 2 weeks after onset of FFP without recovery showed better outcome after surgical decompression.[20] After 2 weeks of paralysis, however, ENoG is not indicated because patients who fail to reach degeneration threshold of more than >90% in the first 2 weeks have a good prognosis for recovery.

Facial nerve EMG evaluates motor activity by measuring electrical action potentials generated by spontaneous and voluntary muscle contraction. This is measured by needle electrodes placed in orbicularis oculi and oris muscles and asking the patient to make forceful contractions. Positive waves and fibrillation potentials are signs of denervation, while polyphasic motor unit potentials indicate active reinnervation. Electromyography is mainly helpful in more longstanding FP, the appearance of polyphasic potentials on EMG indicates nerve regeneration and often precedes functional recovery. In acute FP setting, EMG is mainly helpful in patients with complete palsy where ENoG demonstrates >90% degeneration of the facial nerve. If a subsequent EMG investigation shows voluntary action potentials, the prognosis of facial nerve recovery is nevertheless excellent, and there is no indication for surgical decompression. This is caused by the asynchronous discharge of regenerating nerve fibers that fail to produce measurable potential on ENoG and is called "early de-blocking phenomenon."[21]

Surgical Decompression

The following criteria for surgical decompression were validated by Gantz et al[22]:

- Idiopathic and posttraumatic complete FP;
- ENoG: > 90% degeneration;
- EMG: no voluntary motor action potentials;
- Decompression must occur within 14 days of onset of complete FP.

Intra-operative electrical testing suggests that surgical decompression in BP should involve the entire labyrinthine segment and the bony canal proximal to the geniculate ganglion.[23] Recovery of facial nerve function should not be expected in the first weeks after surgery.[24] A systematic review by Casazza et al reported significantly better facial nerve outcomes for middle cranial fossa decompression (MFD) performed less than 14 days versus more than 14 days after onset of symptoms. Regarding transmastoid decompression (TMD), there was no significant better outcome regarding facial nerve function when compared to medical controls.[25] To be concise, there is evidence for performing MFD in Bell's palsy patients within 14 days of symptom onset, if ENoG shows degeneration greater than 90% and EMG shows no voluntary potentials.[26] There is no robust evidence to support TMD at any stage.

Conclusion

In conclusion, facial nerve palsy is a relatively rare condition, and most practitioners outside specialized centers will only see a few cases per year. While most patients will ultimately show spontaneous and full recovery of facial nerve function, the importance of accurate diagnosis and early treatment cannot be underestimated. Incorrect diagnosis can be harmful to patients, resulting in worse facial function outcomes as a result of delay in diagnosis of occult neoplasms and failure to treat patients within a timely fashion. Management of FP is complex and requires a multidisciplinary board with specific focus (facial plastic reconstructive surgery, opthalmology, neurotology, neurology, neurosurgery, and speech language therapy).

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Surgical Approach to Inferior Concha Bullosa: A Case Report and Literature Review

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ABSTRACT

Inferior concha bullosa is a rare anatomic variation in the nasal cavity and is usually asymptomatic. There is no specific algorithm for surgical approaches to be used for treatment in symptomatic cases. In this case report, we suggest that surgical planning should be done considering the anatomical features of inferior concha bullosa. Here, we present a case of inferior concha bullosa connected with the maxillary sinus. On the basis of the current literature, this patient was treated with a different surgical approach, including partial inferior turbinectomy preserving mucosa on the medial side and repairing the window with a mucosal flap. The anatomical relationship between the inferior turbinate and maxillary sinus is important in determining the surgical approach to inferior concha bullosa. In the surgical treatment of inferior concha bullosa connected with the maxillary sinus, repairing the window between the maxillary sinus and nasal cavity is important to prevent maxillary recirculation. Preoperative radiological evaluation is essential to understand the anatomical relationship more precisely.

Keywords: Endoscopic sinus surgery, Endoscopy, inferior concha bullosa, inferior turbinate

Introduction

Inferior concha bullosa (ICB) is an anatomical variation of the nasal cavity in which an air-filled cavity is located within the inferior turbinate (IT). There are 4 turbinates in the lateral wall of the nasal cavity: inferior, middle, superior, and supreme (not always present). Concha bullosa is most common in the middle turbinate, whereas ICB is very rare.[1]

Management of IT hypertrophy (ITH) includes medical treatment with topical nasal steroids and interventional treatment (radiofrequency ablation, extramucosal or submucosal electrocautery, laser-assisted resection or ablation, cryosurgery, submucosal resection, turbinoplasty, or turbinectomy) to reduce the size of the IT.[2] If radiological imaging is not performed, it is very difficult to diagnose ICB that is refractory to medical treatment.[3] Patients with ICB generally do not benefit from even prolonged medical treatment.

Although ICB does not cause any problems in asymptomatic patients, it requires a surgical approach in patients with symptoms. There is no consensus in the literature regarding the stage of the surgical treatment algorithm. As it is a rare anatomical variation, case reports have presented merely outcomes of their surgical approach. In this study, a patient with ICB connected with the maxillary sinus was presented, who was treated with an approach different from current surgical approaches, and the recent surgical approaches have been discussed according to the anatomical features of ICB.

Case Presentation

A 43-year-old male patient was admitted to our clinic with complaints of nasal obstruction and headache. The patient had these complaints for a long time and was given nasal steroid therapy for ITH and did not benefit. There was no asthma in his medical history. On physical examination, the nasal septum deviated to the right, and there was bilateral ITH, of which the left one was larger.

Paranasal computerized tomography (CT) showed that the nasal septum had deviated to the right. There were some anatomical variations in the left nasal cavity, such as paradoxical middle turbinate, ICB connected with maxillary sinus, uncinate bullosa attached to the lamina papyracea, and supraorbital ethmoid cell (Figures 1 a-c). Sinusitis was not found in the paranasal sinuses. The surgery was planned to correct the septal deviation and to reduce the volume of the bilateral IT.

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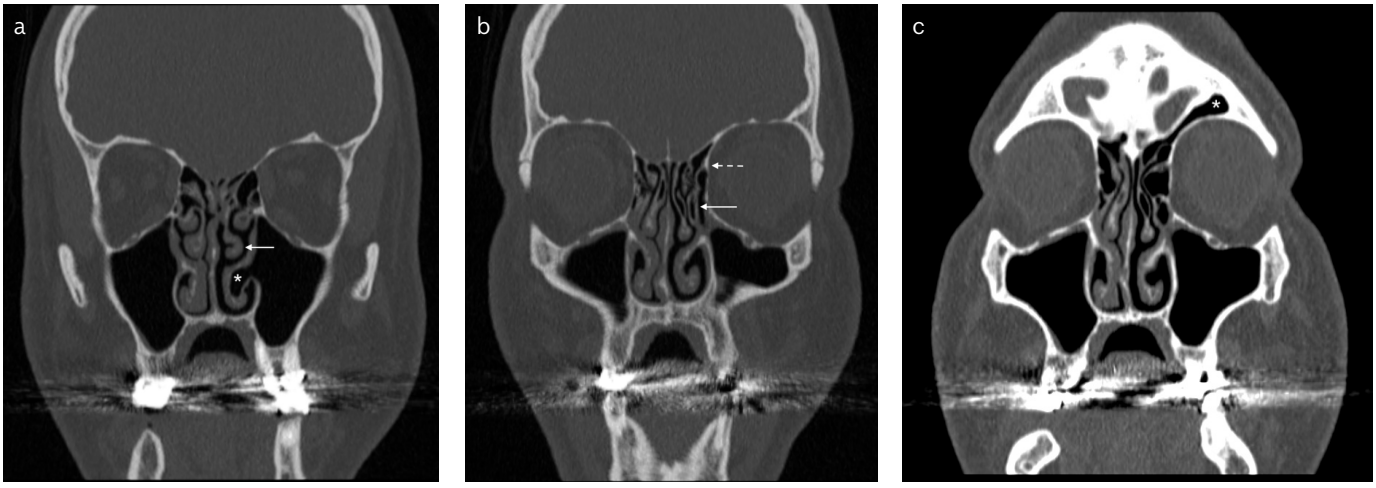

Figure 1. a-c. Coronal computed tomography images of the bone window display the anatomical variations of the left nasal cavity. (a) Left paradoxical middle concha (white arrow) and left inferior concha bullosa connected with left maxillary sinus (white asterisk). (b) Left uncinate bullosa (white arrow) and its attachment to the lamina papyracea (dashed white arrow). (c) Supraorbital ethmoid cell (white asterisk).

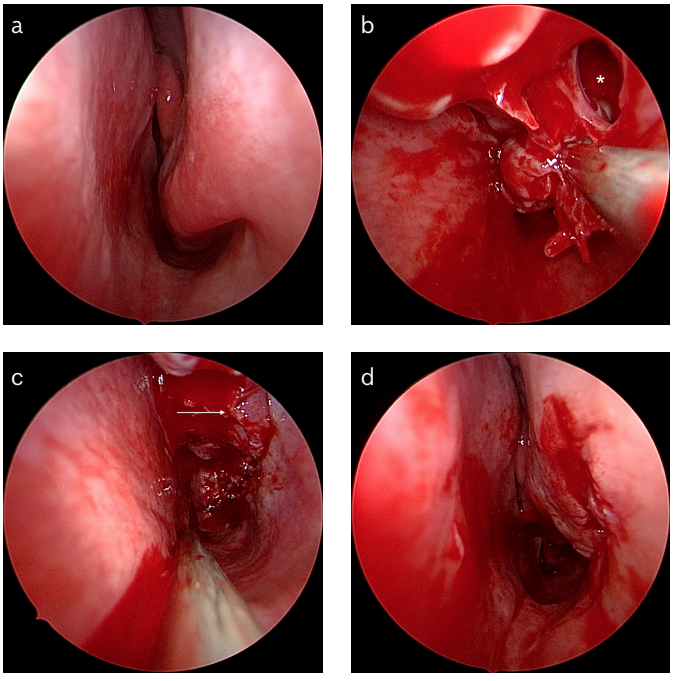

Figure 2. a-d. Intraoperative endoscopic views of the surgical approach. (a) Before the surgery, examination of the vasoconstricted left inferior turbinate. (b) The window created because of left partial inferior turbinectomy (white asterisk). (c) Repairing the window with the mucosal flap (white arrow). (d) Appearance of the left nasal cavity at the end of surgery.

Main Points:

- Inferior concha bullosa (ICB), a rare anatomic variation of inferior turbinate (IT), should be considered in the differential diagnosis of IT hypertrophy (ITH).
- A paranasal computed tomography should be performed before surgery to precisely evaluate the IT anatomy, especially in symptomatic ITH not responding to medical treatment.
- In cases of ICB associated with the maxillary sinus, partial inferior turbinectomy might be considered as a surgical option, provided that the second window of the maxillary sinus medial wall is repaired with a mucosal flap.

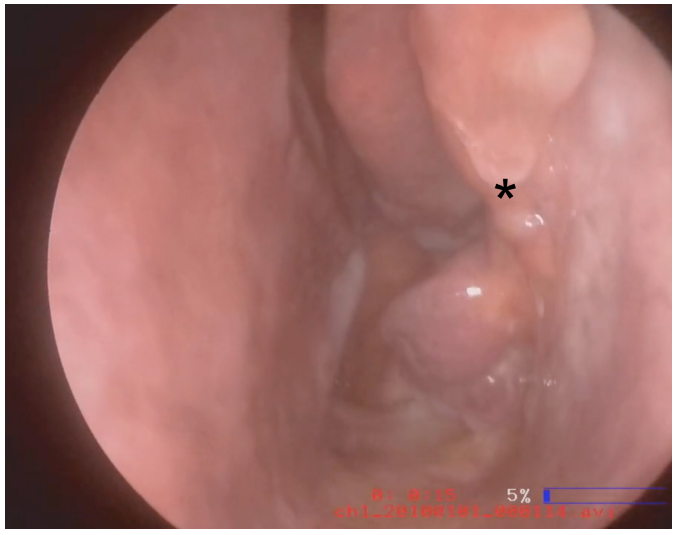

Figure 3. Nasal endoscopic examination 1 month after surgery. The window repaired with the mucosal flap (black asterisk).

During surgery, the deviated part of the nasal septum was removed by submucosal resection. Incisions were performed along the anteroposterior axis at the inferior margin of the ITs. The mucosal flaps were elevated to the medial surface of the ITs. The bones and lateral mucosal surface of the ITs were excised. The maxillary sinus window, formed during bone excision from the medial wall of the left maxillary sinus, was repaired with a medial mucosal flap (Figures 2a-d). Bilateral Merocel nasal packs (Medtronic XOMED, Jacksonville, FL, USA) coated with 0.2% nitrofurazone ointment were inserted inside the nasal cavity after the surgery. They were removed on the third postoperative day. After the removal of the nasal packs, the patient used topical decongestant into his nose for 5 days and rinsed his nasal cavity with saline solution for 3 weeks.

The symptoms of the patient had improved in the first month of follow-up. During endoscopic examination, it was observed that the nasal passage was open and the window between the left maxillary sinus and nasal cavity, which had been formed

Table 1. Demographic and Radiologic Findings of the ICBs and Surgical Approach

Study	Age (years), Sex	Complaint	CT Findings		Surgery of ICB
			ICB Type	Communication with Maxillary Sinus	
Doğru, 1999[4]	35, Female	Nasal obstruction and frontal headaches	Bulbous	No	Excision of the lateral portion
Cankaya, 2001[5]	35, Male	Headaches and nasal obstruction	Bulbous	No	Partial inferior turbinectomy
Özcan, 2002[6]	35, Female	Nasal obstruction and facial headache	Bulbous	No	Outfracture
Uzun, 2004[3]	25, Male	Nasal obstruction and intermittent headache	Bulbous	No	Excision of the lateral portion
Kıroğlu, 2006[7]	14, Female	Nasal blockage, nasal pain, and purulent discharge	Bulbous	No	Excision of the lateral portion
Pittore, 2011[8]	24, Female	Rhinorrhea, nasal obstruction, snoring, and frequent squeezing	Bulbous	No	Removing the free edge of the inferior turbinate
Fidan, 2012[9]	17, Female	Rhinorrhea and nasal obstruction	Bulbous	No	Removing the free edge of the inferior turbinate
Toplu, 2013[12]	37, Female	Nasal obstruction, persistent headache, postnasal discharge, and occasional epistaxis	Extensive	Yes	Outfracture, crushing, and radiofrequency thermocoagulation
Özturan, 2013[10]	23, Male	Nasal congestion, nasal drip, and intermittent facial pain	Bulbous	No	Crushing, radiofrequency ablation, and outfracture
Sapmaz, 2014[13]	23, Female	Continuous headache and breathlessness	Extensive	Yes	Radiofrequency ablation and lateralization
Erdur, 2017[11]	13, Female	Nasal obstruction, intermittent facial pain, and postnasal drip	Bulbous	No	Submucosal bone resection and lateralization
Koo, 2018[1]	14, Male	Nasal obstruction, chronic headache, and purulent nasal discharge	Extensive	Related to maxillary sinus ostium	Middle meatal antrostomy, inferior turbinectomy, and outfracture
This study	43, Male	Nasal obstruction and headache	Extensive	Yes	Partial inferior turbinectomy and repairing the window with a mucosal flap

CT, computed tomography; ICB, inferior concha bullosa.

intraoperatively, was closed (Figure 3). A written informed consent was taken from the patient.

Discussion

Inferior concha bullosa is one of the rare anatomic variations of the nasal cavity, which should be considered as a differential diagnosis of ITH.[1] Whereas asymptomatic patients can be followed up without surgery, a surgical approach is required in symptomatic patients. The cases requiring surgical treatment is rare in the literature. The characteristics of these cases are presented in Table 1.

The relationship between ICB and maxillary sinus is important in the evaluation of the surgical approach. In most patients who required surgery, ICB was not associated with the maxillary sinus.[3-11] Hence, excisional surgical approaches of the IT were used in these patients, such as excision of the lateral portion, partial inferior turbinectomy, removing the free edge of the IT, and submucosal bone resection. In addition, less invasive approaches such as radiofrequency ablation, lateralization, and outfracture were also preferred. In these patients, as there was no risk of opening a new window on the medial wall of the maxillary sinus, all options for reducing the volume of IT could be used readily. Preoperative CT evaluation is, therefore, the most determinant examination in the surgical approach.

When it was observed that ICB was associated with the maxillary sinus in the radiological evaluation, excisional options of the IT were not preferred.[12,13] In these patients, maxillary sinus recirculation was observed owing to the second window. Therefore, less invasive approaches preserving the medial wall of the maxillary sinus were preferred, such as outfracture, crushing, radiofrequency thermocoagulation, and lateralization. Although less invasive methods have been sufficient in these cases for resolving complaints, increased volume reduction in

IT may be needed. Because of the extensive ICB surrounded by low submucosal soft tissue, an excisional treatment approach that provides an effective volume reduction without opening a second maxillary window was preferred in our patient. There is, however, a need for studies with large case series evaluating the effect of less invasive techniques (outfracture, crushing, radiofrequency thermocoagulation, and lateralization) and excisional techniques on nasal obstruction according to the type and size of the concha bullosa and amount of submucosal soft tissue surrounding the concha bullosa.

A rare condition in ICB is also its association with the maxillary sinus ostium. In a case report, because the ICB was associated with the maxillary sinus ostium, a wide drainage pathway was provided from the maxillary sinus ostium by performing a maxillary antrostomy. Inferior turbinectomy and outfracture were performed for the second ICB not connected with the maxillary sinus.[1]

According to the anatomical relationship between ICB and maxillary sinus, 3 anatomic forms appear as isolated ICB, ICB associated with the maxillary sinus ostium, and ICB associated with the maxillary sinus.[1,11,12] In isolated ICBs, a broad spectrum of surgical approaches, which can be used in ITH, could be preferred to provide sufficient airway. ICBs associated with the maxillary sinus ostium can also be treated like isolated ICBs. Providing a wide drainage pathway for the maxillary sinus ostium by opening the ICB widely is a critical issue to be considered in these patients. In ICBs associated with the maxillary sinus, the maxillary sinus should be kept separate from the nasal cavity by a bony barrier or repaired with a mucosal flap. In our surgical approach, this isolation was provided with a mucosal flap obtained from ICB itself.

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – O.E., A.Y.; Design – O.E., A.Y.; Supervision – O.E., A.Y.; Resources – O.E., A.Y.; Materials – O.E., A.Y.; Data Collection and/or Processing – O.E., A.Y.; Analysis and/or Interpretation – O.E.; Literature Search – O.E., A.Y.; Writing Manuscript – O.E., A.Y.; Critical Review – O.E., A.Y.;

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CASE REPORT
PEDIATRIC OTOLARYNGOLOGY
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Laryngeal Foreign Body: A Rare Case Report

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ABSTRACT

Foreign body aspiration is a condition commonly encountered in childhood that can lead to serious complications. The most common symptoms in foreign body aspiration are coughing and hoarseness, and these symptoms may mimic the symptoms of common diseases such as asthma, recurrent pneumonia, and upper respiratory tract infections. These common symptoms can cause delays in the diagnosis. Neck and chest radiography are the basic methods for evaluating a patient suspected of foreign body aspiration. The absence of foreign bodies on imaging does not rule out the diagnosis. An 11-month-old baby with cough, hoarseness, and wheezing that persisted for 3 months was consulted in our clinic. The patient was reported to have presented to an outpatient center 3 months ago with the suspicion of foreign body aspiration and no foreign body was detected. Inspiratory stridor was observed on the physical examination, and the cry was hoarse in quality. A flexible laryngoscopic examination was performed, and rima glottis foreign body impaction was observed. The foreign body was removed using forceps with the help of a videolaryngoscope under sedation. In patients with a sudden onset of respiratory tract symptoms, the presence of foreign bodies should be ruled out. Endoscopic laryngeal examination is essential in these patients. It is very important to suspect the presence of a foreign body in diagnosis.

Keywords: Aspiration, foreign body, larynx, treatment, videolaryngoscope

Introduction

Foreign body aspiration (FBA) is a common childhood pathology and may cause various symptoms depending on its size and location.[1] The aspirated objects can cause life-threatening airway obstruction at any age.[2] In patients with FBA, the foreign body is mostly (80%-90%) lodged in the bronchial tree, and laryngeal localization is rare. This could be because most foreign bodies are small enough to pass through the larynx and trachea and get lodged distally.[2,3] In addition to laryngeal foreign bodies posing diagnostic difficulties for family physicians, emergency room physicians, and ENT physicians; incomplete obstruction at the laryngeal level can lead to unclear and often overlooked symptoms.[4] In this study, an 11-month-old patient with 3-month history of cough, hoarseness, stridor, and wheezing complaints was detected with a laryngeal foreign body, and our approach to this diagnosis is presented in light of the literature.

Case Presentation

An 11-month-old baby with cough, hoarseness, and wheezing that persisted for 3 months was consulted in our clinic. The patient was reported to have presented to an outpatient center 3 months ago with the suspicion of FBA, and no foreign body was detected on posteroanterior (PA) chest radiography. It was also reported that the patient whose complaints did not regress, was admitted to several different hospitals during this period, and there was no response to the treatments provided for bronchiolitis (salbutamol nebulization). Inspiratory stridor was observed on the physical examination of the patient, and the cry was hoarse in quality; however, the patient's vital signs were normal. Oxygen saturation was measured at 95 mmHg by a finger pulse oximeter. No finding related to a foreign body was detected on PA chest radiography (Figure 1). In addition, no signs of hyperinflation or atelectasis were observed. On the flexible laryngoscopic examination performed to rule out the presence of a foreign body in the airway, rima glottis foreign body impaction was observed. Both vocal cords and the aryepiglottic plicas were edematous. Vocal cord movements were normal. A linear, hyperdense foreign body, approximately 6 × 2 mm in size, was observed in the anterior of the laryngeal air column on a neck computed tomography (CT) scan, which was taken to determine the relationship of the foreign body with soft tissues and to rule out deep neck infection (Figure 2). Arrangements for intubation and tracheotomy was undertaken,

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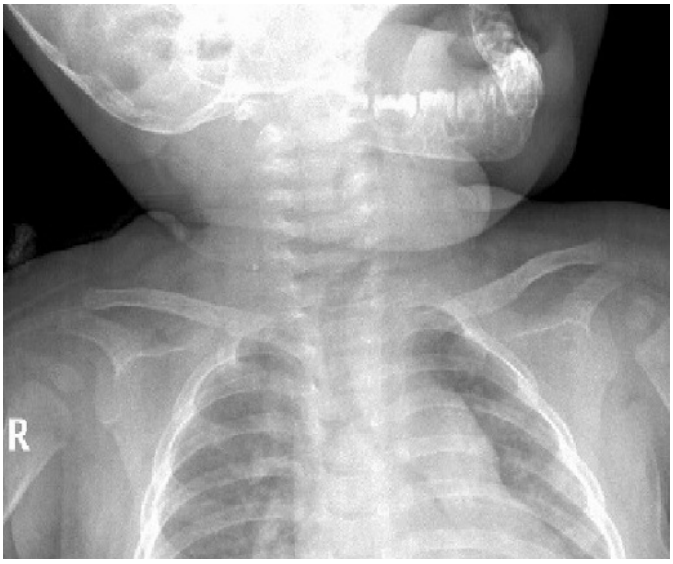

Figure 1. PAAC chest radiography does not show any foreign body, lung hyperinflation, or atelectasis findings.

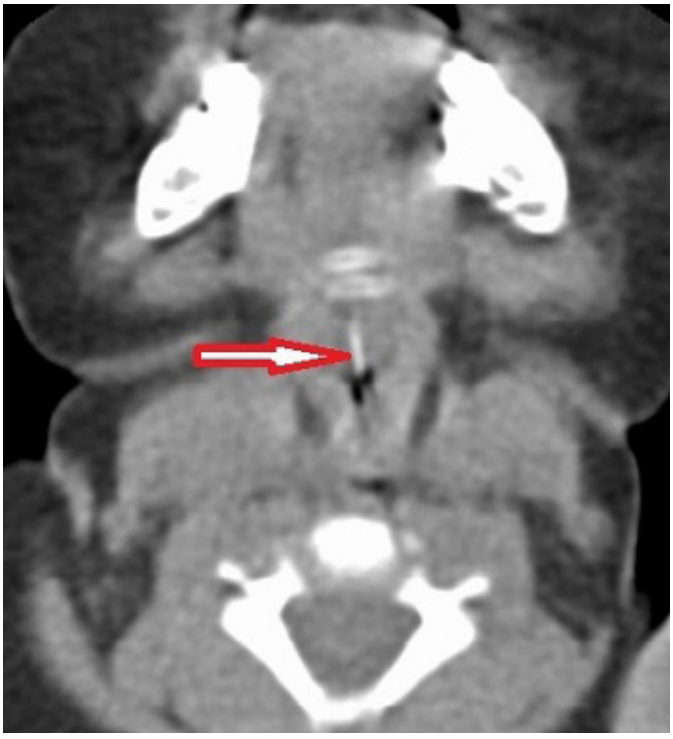

Figure 2. On computed tomography, a hyperdense foreign body is observed at the laryngeal level (the arrow indicates the foreign body).

Main Points:

- Foreign body aspiration (FBA) can cause life-threatening airway obstruction at any age.
- Symptoms caused by FBA may mimic the symptoms of common diseases such as asthma, recurrent pneumonia, and upper respiratory tract infection.
- It is very important to suspect FBA for a diagnosis.
- In patients with a sudden onset of respiratory tract symptoms, the presence of foreign bodies should be ruled out endoscopically.

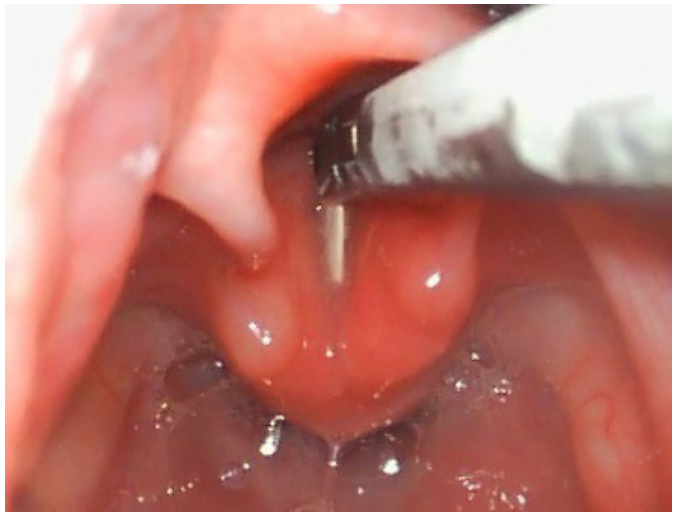

Figure 3. Rima glottis impacted plastic foreign body.

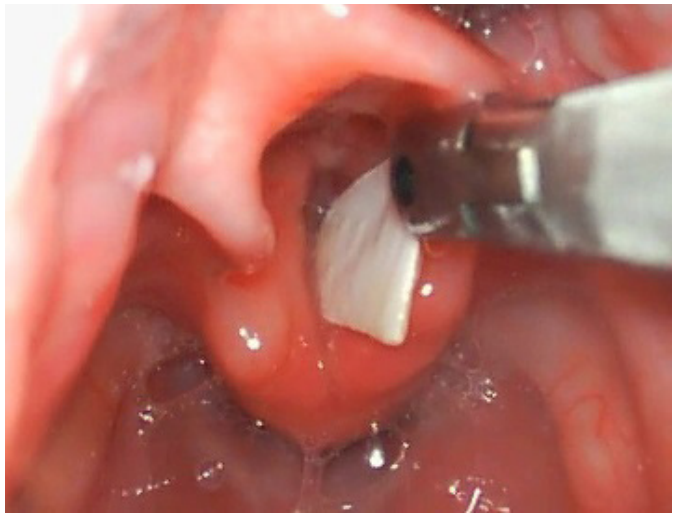

Figure 4. Foreign body was removaled with the help of a videolaryngoscope.

the patient was sedated (2.5 mg/kg propofol, 1 μg/kg remifentanil, and 1 mg/kg methylprednisolone), and intervened under operating room conditions. The foreign body was removed using forceps with the help of a videolaryngoscope (Figures 3-5). The foreign body removed was 10 × 8 mm in size, and the composition of the foreign body was plastic (Figure 6). The patient was discharged upon improving clinically after monitoring for 1 day after operation in the ward. During the postoperative second week and second month follow-up visits, it was observed that the symptoms of patient had disappeared, and a flexible endoscopic examination revealed unremarkable laryngeal structures and mobile vocal cords. The patient's parents gave informed consent for publication of this case report.

Discussion

Foreign body aspiration is a commonly encountered problem in pediatric patients, and deaths caused by asphyxia owing to FBA is a leading cause of accidental deaths in children under 4 years.[5] Foreign body aspiration is a life-threatening condition that requires prompt intervention. Its incidence peaks between the ages of 1 and 3 years and it is more common in boys.[1,6] The

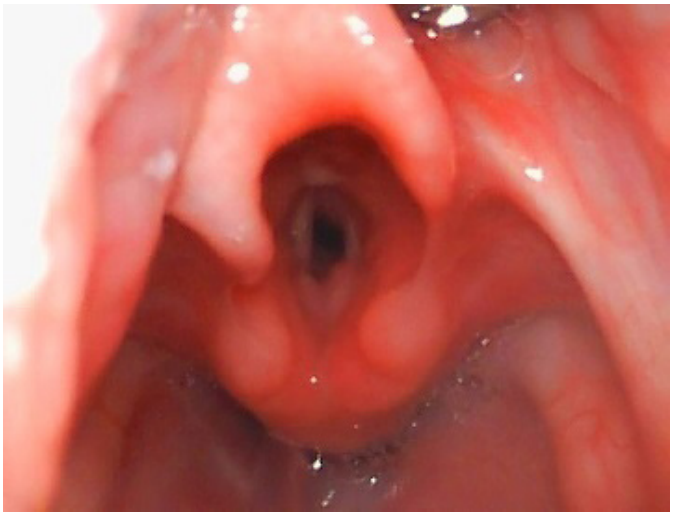

Figure 5. The appearance of the larynx after foreign body removal.

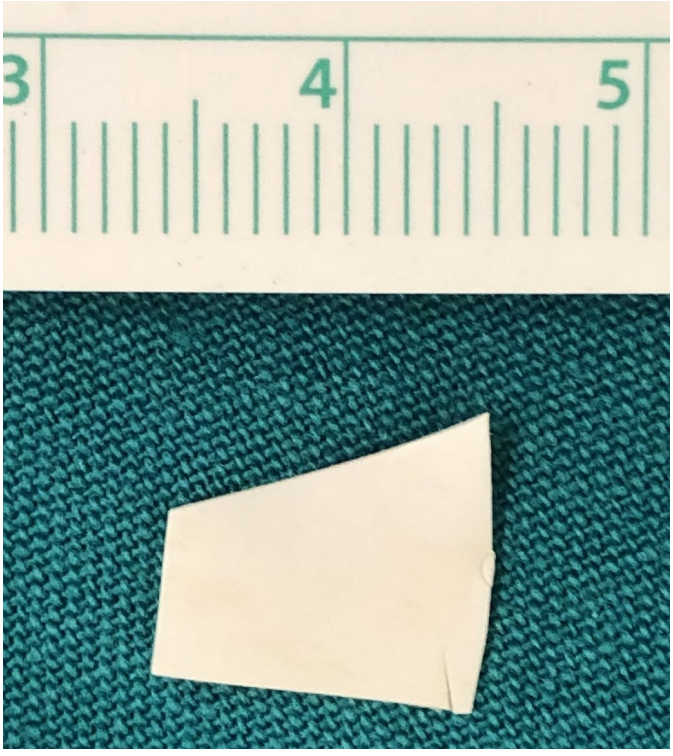

Figure 6. Image of plastic foreign body.

patient who presented was an 11-month-old male baby. The reasons for the increased frequency of FBA in children of a playing age can be listed as high localization of the larynx, poor coordination of swallowing, children's desire to learn about the environment and their tendency for oral exploration, and their movements during the time of ingestion.[7] In a study by Chen et al,[3] the most common symptoms in patients with FBA were coughing and hoarseness, and these symptoms can mimic the symptoms of common diseases such as asthma, recurrent pneumonia, and upper respiratory tract infections. Although the classical physical examination findings of laryngotracheal foreign bodies are stridor and hoarseness, unilateral decreased respiratory sounds and wheezing are observed in bronchial foreign bodies.[8] If the foreign body causes partial obstruction of the larynx, it causes milder symptoms and can be difficult to distinguish from infections.[9]The delay in diagnosis in this patient can be explained by the penetration of a thin foreign body into the larynx and partial obstruction of the respiratory tract. As the diagnosis of FBA is delayed, the risk of complications and death rises.[1] In this case, the patient had received various treatments for wheezing and cough for 3 months. Delay of FBA diagnosis and intervention may cause asphyxia, atelectasis, chronic cough, granulation tissue, recurrent pneumonia, and growth retardation.[10]The existence of a prolonged cough in a child with one or both abnormal chest X-ray or auscultation findings should alert the clinician to the probability of FBA.[11] In the follow-up visits of the patient, the laryngeal structures were normal.

In evaluating a patient with suspected FBA, the presence of a radiopaque foreign body on the neck and chest X-ray may assist in the diagnosis; however, the absence of any finding in these methods does not rule out the diagnosis.[3] In a cadaver model study, plastic parts that were undetected on plain radiography were detected on CT scan.[12] In our patient as well, the foreign body that was not observed on direct radiography could easily be detected on CT. Because of the widespread availability of plastic toys and plastic parts in the surroundings where children play, clinicians should be suspicious of FBA in the presence of normal radiographic findings and vague symptoms.[13] Of the patients diagnosed with FBA, 45% are diagnosed within the first day, 83% in the first 1 month, and 17% after more than 30 days.[14] In the case presented here, the patient's complaints had been ongoing for 3 months, and the child had visited different health institutions several times. The patient had received treatment for lower respiratory tract infection and was receiving treatment for bronchiolitis. The underlying independent risk factors for misdiagnosis or overlooking foreign body in patients with laryngeal FBA can be listed as nonspecific symptoms, failure to witness the aspiration, late transfer of patient to the physician, and negative radiological findings.[3] If FBA is highly suspected on flexible endoscopic examination, direct laryngoscopy or rigid bronchoscopy, which are gold standards, is indicated.[3] In our patient's case, the foreign body was detected on endoscopic examination and was removed with the help of a videolaryngoscope under sedation. In a study conducted by Fidkowski et al,[5] they stated that an intervention performed with induction anesthesia and spontaneous ventilation minimizes the risk of progression of partial obstruction to complete obstruction. As intubation and positive pressure ventilation may cause the laryngeal foreign body to lodge distally and lead to complete obstruction, the foreign body in our patient was removed under sedation while preserving spontaneous respiration. In the presence of laryngeal FBA, apart from sudden death, subglottic stenosis and laryngeal granulation tissue may develop owing to longstanding airway foreign body.[3] Laryngeal structures were observed to be normal in the post-procedure follow-up visits of the patient.

In conclusion, laryngeal foreign body is a life-threatening emergency condition. In patients with a sudden onset of respiratory tract symptoms, the presence of foreign bodies should be ruled out endoscopically, or if necessary, bronchoscopically, even if imaging is negative in patients where symptoms do not regress and FBA is suspected. It is very important to suspect the presence of a foreign body in diagnosis and to take an accurate history.

Informed Consent: Written informed consent was obtained from the patients who agreed to take part in the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept– Ö.F.Ç, S.S.; Design – İ.K., M.G.Y.; Supervision – İ.O., C.Y.; Resources – İ.K.; Materials – Ö.F.Ç., C.Y.; Data Collection and/or Processing – İ.K., Ö.F.Ç.; Analysis and/or Interpretation – İ.K., M.G.Y.; Literature Search – İ.K., M.G.Y.; Writing Manuscript – İ.K.; Critical Review – İ.O, S.S , C.Y.

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