

New Insight about Management lines of Atrophic Rhinitis

Mai Abdel Baseer Abdel Qader, Tarek Abdel Zaher Emara, Ahmed Mohamed El Hady Anany, Ahmed Nagy Hadhoud

Otorhinolaryngology Department, Faculty of Medicine - Zagazig University, Egypt Email: <u>Maibaseerro@gmail.com</u>

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Abstract

Background: Atrophic rhinitis (AR) is a chronic progressive condition characterized by formation of thick dry crusts in a roomy nasal cavity, which has resulted from progressive atrophy of the nasal mucosa and absorption of underlying bone. The various symptoms which result from the primary nasal pathology and its sequelae may comprise foul smelling thick black or greenish crust, nasal obstruction, epistaxis, and anosmia. Atrophic rhinitis can be classed as primary or, where it is a consequence of another condition, secondary. The use of synthetic implants has been studied in ENS reconstructive surgery. These implant types include silicone perforated sheeting or porous polyethylene (MedPor, Stryker Corporation, Kalamazoo, MI, USA). These materials do not react and are designed to allow tissue growth to penetrate the material and improve long-term integration. Synthetic implants are generally inexpensive and easy to obtain in most practices. Since synthetic implants are foreign bodies by nature, infection, and extrusion are possible complications. After tissue ingrowth, implant removal due to scarring is challenging if such extraction is warranted.

Keywords: Management lines, Atrophic Rhinitis

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Atrophic rhinitis (AR) is a chronic progressive condition characterized by formation of thick dry crusts in a roomy nasal cavity, which has resulted from progressive atrophy of the nasal mucosa and absorption of underlying bone. The various symptoms which result from the primary nasal pathology and its sequelae may comprise foul smelling thick black or greenish crust, nasal obstruction, epistaxis, and anosmia. Atrophic rhinitis can be classed as primary or, where it is a consequence of another condition, secondary (1).

Primary atrophic rhinitis

Primary atrophic rhinitis is a progressive chronic nasal disease characterized by sclerotic transformation of the mucous membrane and increased patency of the nasal passages due to atrophic changes in the underlying bones and mucosa. (2).

Primary atrophic rhinitis usually affects young- and middle-aged adults, especially females (F:M = 5.6:1) (3). Etiology of primary atrophic rhinitis

Etiology of primary atrophic rhinitis is still a debatable topic, and it has kept various stakeholders like otorhinolaryngologists, microbiologists, epidemiologists, etc., interested for more than 100 years. Factors involved in its genesis include autoimmunity, chronic sinus infection, hormonal imbalance, poor nutritional status, heredity, and iron deficiency anemia. Chronic bacterial infection is considered one of the major causes of primary atrophic rhinitis, especially in highly densely populated regions of the world like countries of Southeast Asia, because sinonasal bacterial infections are contagious (3).

1. Bacteriology

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Klebsiella Ozaenae is considered the most common causative agent. Other infectious agents which have been implicated include Coccobacillus foetidus-ozaenae, Bacillus mucosus, Diphtheroids bacillus, Bacillus

pertussis, Haemophilus influenzae, Pseudomonas aeruginosa, Proteus mirabilis, and *Staphylococcus aureus.* According to the experience of physicians who treat upper respiratory tract infection, one cannot be sure whether these bacteria cause the disease or are merely secondary invaders. Few researchers have also isolated *Escherichia coli* from cases with primary atrophic rhinitis. Nutritional deficiency especially iron, fatsoluble vitamins like A, D, E, and K, and proteins leads to an increased susceptibility to recurrent upper airway infections (4).

2. Mycology

Concomitant fungal infection like *aspergillus* species have been isolated from patients diagnosed with atrophic rhinitis. Though no multicenter studies confirm the role of fungal infections in atrophic rhinitis, others clinical experience suggests that it is mainly due to contamination or nosocomial acquired. The only patients where saw *aspergillus* species as primary agents were cases of invasive fungal sinusitis causing atrophy.

(2).

Clinical presentation

3. Signs

On endoscopic exam, patients universally have thick, adherent crusts that are yellow-green to gray-black. On removal of the crusts, marked atrophy of the turbinates, especially the inferior ones, can be seen. The turbinate atrophy creates an excessively patent nasal passage through which the posterior nasopharynx and upper portions of the soft palate can be seen. The nasal mucosa is markedly thin, pale, shiny, and bleeds easily. Less frequently encountered clinical signs of atrophic rhinitis include septal perforation, columellar necrosis, and a depressed nasal bridge (2).

4. Symptoms

Patients typically complain of excessive nasal crusting and "paradoxical" nasal obstruction despite the fact that the nasal cavities are actually enlarged, and foul smell emanating from the nasal cavity that is a source of embarrassment and occasional social rejection. The sense of obstruction may be the result of crusting or disrupted airflow. Other associated symptoms include facial pain, headache, mucosal dryness, dyspnea, epistaxis, sleep disturbance, and occasional mucopurulent rhinorrhea and anosmia. This last symptom most likely occurs as a result of atrophy of the olfactory epithelium in the nasal roof (2).

The feeling of "not getting air" is not alleviated with mouth breathing and often has a negative impact on psychological wellbeing, which manifests as anxiety, depression, anger, frustration, irritability, and fatigue. A unique symptom is *aprosexia nasalis*, where the patient becomes extremely preoccupied with the attempt to maintain a sensation of breathing such that they experience chronically decreased concentration (5). **Diagnosis**

The diagnosis of atrophic rhinitis is made clinically and confirmed by carefully obtained endoscopically guided middle meatal cultures. Nasal biopsy specimens can show loss of the normal pseudostratified columnar epithelium and atrophy of the mucus glands. A nasal culture identifying *K. ozaenae* strongly suggests the diagnosis and the isolation of other associated organisms is also helpful. Multiple microorganisms are frequently cultured, including *Proteus*, *Escherichia coli*, *Staphylococcus aureus*, *pneumococci*, *Perez–Hofer bacillus*, and an atoxic form of *Corynebacterium diphtheriae*. *K. ozaenae* is an encapsulated gram-negative rod that is most often associated with and isolated in this disease. *K. ozaenae* displays a ciliostatic effect by creating intraciliary adherence that leads to poor mucociliary clearance (6).

The use of the endoscope is critical to obtain culture material and to avoid contaminated cultures. Characteristic nasal features include enlarged nasal cavities, resorption of the turbinates, mucosal atrophy, thick crusts, and ozena. The foul odor of atrophic rhinitis appears to be the most distressing symptom (6).

Because of the high incidence of concurrent sinusitis, CT is frequently included in the diagnostic evaluation of atrophic rhinitis. list characteristic changes identified by CT as the following:

(1) Mucosal thickening of the paranasal sinuses.

- (2) Loss of definition of the osteomeatal complex secondary to resorption of the ethmoid bulla and uncinate process.
- (3) Hypoplasia of the maxillary sinuses.
- (4) Enlargement of the nasal cavities with erosion and bowing of the lateral nasal wall.
- (5) Bony resorption and mucosal atrophy of the inferior and middle turbinates. (7)

Management

5. Medical management

The mainstay of the management of AR is conservative medical management, locally or systemically, which includes:

(1) Nasal douches

A solution made of 28.4 g of sodium bicarbonate, 28.4 g of sodium diborate, which acts as an antiseptic and buffers the bicarbonate in the solution, 56.7 g of sodium chloride to make the solution isotonic, and 280 mL of warm water. Irrigation for both nostrils was performed with a 10 to 20 cc syringe or any other nasal irrigation device 3 to 4 times daily. During irrigation, the patient was advised to say "k k k" to close the velopharyngeal sphincter, thereby preventing aspiration (8).

Sumaily et al., (8) compared homemade saline solutions used for nasal irrigation and their effect on mucociliary clearance (MCC). They found that saline with iodized table salt improved MCC in healthy people, similar to saline without iodized salt, with a significant decrease in saccharine clearance time (SCT), which was more pronounced in iodized saline solution. They evaluated the effect of diluted baby shampoo saline irrigation (1% concentration) on MCC in healthy subjects and found an increase in SCT after using the irrigation compared to SCT before using.

(2) Nasal drops

Glucose glycerin drops consisting of 25% glucose were found to inhibit bacterial growth by fermentation, which produced local lactic acidosis and prevented bacterial growth. Glycerin is a lubricant that increases mucosal moisturization by absorbing atmospheric water. It is recommended to use it after nasal douches. Liquid paraffin nose drops are effective lubricants, but they are not recommended due to the risk of inhalant lipoid pneumonia (9).

Estradiol in Arachis oil drops can be used based on histopathological findings, and it was found to be beneficial in Type 1 but not recommended in Type 2 AR, as it may lead to a worsening of the condition.

Topical antibiotic solutions, such as 80 mg of gentamicin sulfate dissolved in 1 L of normal saline solution, should be reversed in patients with purulent discharge and target the causative organism, mainly Klebsiella. In one study, patients who received streptomycin and vasodilator medication orally or subcutaneously had satisfactory results (**10**).

Nasal spray of pure α -tocopherol acetate, 2 puffs in each nostril, 3 times a day for 6 months, and found an improvement in nasal symptoms and perception of nasal airflow but no increase in nasal resistance. (10).

Mitomycin C has an antiproliferative effect, inhibiting fibroblast activity and preventing nasal mucosa fibrosis. (11)

(3) Systemic medications

It is usually necessary from time to time and should be directed according to the nasal culture of purulent discharge. Tetracycline, aminoglycosides, and, more recently, ciprofloxacin (250-500 mg twice daily for 4 weeks) have been successful. Supplements are recommended for iron, zinc, protein, and vitamin (A and D) supplements for malnutrition patients with mineral deficiencies. A previous study found satisfactory results from iron therapy in 50% of patients, as they noticed improvements subjectively (12).

(4) Nasal submucosal injections

Many materials have been used for nasal submucosal injection to treat patients with AR. A study using autologous fat and platelet-rich plasma injected into the inferior and middle turbinate, septum, and nose floor showed a significant improvement in subjective and objective aspects (13).

(5) Vaccine

In swine, AR is mainly caused by *Bordetella bronchiseptica* and *Pasteurella multocida*. Vaccines consisting mainly of toxoids against these organisms showed control of the situation, which must be evaluated in managing AR in humans (13).

6. Surgical management

Surgical approaches have been used to manage AR. They aim to decrease the size of the nasal cavities to increase nasal resistance. Therefore, the drying effect of air turbulence decreases. Second, regeneration of the normal nasal mucosa is promoted by temporary partial or complete closure of the nostril; third, increasing lubrication of the dry nasal mucosa by increasing the activity of the nasal mucosal glands or bringing secretions from other places. Furthermore, it improves the vascularity of the nasal cavities mainly by blocking the sympathetic system (1).

(1) Decreasing the size of the nasal cavities

Many approaches have been used to decrease the nasal cavity, from Caldwell-Luc up to the endoscopic approach. Many materials have been used, including homologous autologous endings with synthetic materials and a Dermo-fat implant, harvested from the anterior thigh and implanted on the nasal floor but has the risk of resorption (8).

The graft is implanted in the lateral nasal wall just below the inferior turbinate after the elevation of the mucoperiosteum starting at the level of the pyriform aperture. (3).

(2) Promoting regeneration of the normal nasal mucosa

Young operation: suggested complete closure of the unilateral nostrils or both to promote regeneration of healthy mucosa. This technique involved a circumferential incision at the mucocutaneous junction, and the skin was sutured together. (8).

They believed that the closure of the nostril would stop air turbulence and increase CO_2 and pH, which would help in mucosal regeneration. In addition, they thought that closure would create negative pressure, which would promote revascularization. However, patients may develop scars and vestibular stenosis.⁴ Some modifications were applied to Young's approach, targeting improving the results or preventing its disadvantages (14).

- Sinha modification: Instead of complete nostril closure, he did partial nostril closure with the same technique as the Young by leaving a small opening in the nostril. He achieved complete cure in cases with a 3 mm opening, while the percentage of cure dropped to 70% as the size increased to 5 mm (15).

- **Gadre modification:** He suggested a modification that involves elevating the mucosal flap posterior to the incision rather than reflecting the nasal vestibular skin. The mucosal flap was then sutured together, forming the obliterating membrane. The result was a three-layered membrane; the external epithelial layer continued with the skin externally, the internal epithelial layer continued with the mucous membrane internally, and fibrous tissue was found. The advantage is the simplicity of raising the flap and aesthetic improvement because the obliterating membrane will not be evident (16)

- **Ghosh's vestibuloplasty:** Ghosh hypothesized the etiopathogenesis of AR, namely that decalcification of the turbinate secondary to reflex sympathetic dystrophy, which makes them collapsible under the impact of the inspiratory stream, which hits them continuously, resulting in AR. His technique elevates the flap from the skin on the lateral wall from the ala 3 mm posterior to the nostril opening with its base to the vestibular lumen. Then fold it over itself with its base laterally and suture it together so the air stream hits it and is redirected to the septum. Thus, the lateral wall is kept without direct air turbulence. This technique will allow the lateral nasal wall to regenerate normal mucosa while the nasal cavity is opened and accessible for cleaning and the advantage of doing both nostrils at 1 stage (**8**).

- **El Kholy modification:** To prevent scar and vestibular stenosis, he modified by making a hemi transfixion incision in the contralateral nasal cavity, elevating the flap, and then making a cartilage incision to access the ipsilateral mucoperichondrial flap. Additionally, an incision was made at the posterior end of the elevated ipsilateral flap, which was then reflected and sutured with another flap raised on the lateral vestibular skin on the ipsilateral side (**17**).

(3) Increasing lubrication of the dry nasal mucosa

Wittmaack's operation was reimplanting the Stenson's duct to the maxillary antrum. This technique will help moisturize the dry nasal cavity through parotid secretions. other operations using the Caldwell-Luc approach, in which the maxillary sinus mucosa was elevated from the sinus wall and then reflected through the antrum to the nasal cavity, filling the roomy nose and increasing secretion through the normal sinus mucosa. (18).

(4) Improving vascularity of the nasal cavities

Stellate ganglion injections: A local anesthetic agent (10–15 cc of 1% xylocaine) was injected into the stellate ganglion of the cervical sympathetic chain to block the sympathetic action that causes vasodilation. The effect of injection was confirmed by Horner syndrome and nasal mucosa congestion. The daily injection is recommended in unilateral cases, while in bilateral cases, injected separately on alternative days to avoid bilateral recurrent laryngeal nerve palsy. However, it was found that the effect is temporary, and patients relapse within 4 to 8 days. Cervical sympatheteomies have the same concept as stellate ganglion injection but with permanent results (8).

Secondary AR

Etiology

The secondary type of AR is attributed to a known underlying cause. Causes can be traumatic or iatrogenic. Other causes could be secondary to granulomatous diseases such as tuberculosis, leprosy, granulomatosis with polyangiitis, or sarcoidosis. (19).

Clinical presentation

SECONDARY ATROPHIC RHINITIS patients typically present with excessive nasal crusting, malodor emanating, and paradoxical nasal obstruction in the nasal cavity. Other associated symptoms include headache, facial pain, anosmia, mucosal dryness, dyspnea, sleep disturbances, epistaxis, and rarely mucopurulent rhinorrhea. (20).

Pathophysiology

Numerous studies have emphasized that the sensation of nasal obstruction is not correlated with anatomical nasal obstruction; therefore, these studies suggest that the perception of nasal patency is a neurosensory rather than an anatomical mechanism. Consequently, when a large amount of inspired airstream has less contact with the surface area of the mucosal wall due to the lack of turbulence, this airflow pattern produces insufficient mucosal cooling, leading to a lesser perception of nasal patency (**21**).

In individuals with SECONDARY ATROPHIC RHINITIS, there were 3 consistent changes following inferior turbinate excision. The airflow patterns observed are laminar as opposed to turbulent. The velocity of the airflow vectors is increased rather than medium velocity. The compromised airflow to the inferior portion of the nasal cavity causes a shift in the airflow upward toward the middle meatus and nasopharynx region (22).

Another theory suggested that poor trigeminal nerve regeneration or damage after nasal surgeries may lead to poor input into the air-inspired perception pathway. Patients with SECONDARY ATROPHIC RHINITIS are strongly associated with conditions that affect mental health, such as depression and anxiety. This psychogenic effect affects nasal perception of airflow (23).

Diagnosis

1. History

patients with SECONDARY ATROPHIC RHINITIS generally have an underlying cause of symptoms such as trauma, radiation therapy, and nasal surgery.

2. clinical symptoms

as several methods have been described to objectively assess nasal patency: anterior rhinomanometry, acoustic rhinometry, and peak nasal inspiratory flow, but they are not correlated with the severe symptoms of the patients (21).

3. CT scans

Patients with SECONDARY ATROPHIC RHINITIS can reveal thickening of the mucosa in the septum and sinuses, enlargement of the sinuses of the nasal cavity, destruction of the lateral nasal wall, and partial or complete resection of the middle/inferior turbinate.

4. ENS 6Q

contains 6 questions with a score of 0 to 5 is used to investigate common presenting symptoms of SECONDARY ATROPHIC RHINITIS. A score equal to 10.5 and more confirms the diagnosis of SECONDARY ATROPHIC RHINITIS (24).

5. Office-based cotton test

This is an office test to evaluate the effect of the placement of cotton moisturized with normal saline in the nose area with deficient tissue. Cotton was left in place for 20 to 30 minutes, then a reassessment of patient symptoms was performed using EMS6Q to confirm diagnosis and results in the surgical management of those patients (**25**).

Histopathology

Histopathology is based on the underlying causes. These include granulomatous sinonasal diseases such as sarcoidosis, tuberculosis, leprosy, and GPA. In the case of sarcoidosis, the histopathology shows noncaseating granulomata associated with progressive fibrosis due to a granulomatous response, and no vasculitis or cholesterol crystals can be detected (**26**).

In tuberculosis and leprosy, histopathology shows that caseating granulomas have submucosal fibrosis and decrease the number of submucosal glands. A biopsy study showed epithelioid cell granulomas associated with T lymphocytes and vasculitis in granulomatosis with polyangiitis (**26**).

Management

1. Prevention

Prevention of the disease is mandatory because the management options become restricted once the SECONDARY ATROPHIC RHINITIS manifests. Carefully examining the need for surgical turbinectomy and other strategies, such as medical management, in allergic patients to reduce tissue loss and allow fast restoration of nasal physiologic mechanisms is critical. Other surgical approaches for the management of hypertrophied turbinate have been used to minimize the incidence of SECONDARY ATROPHIC RHINITIS, including electrical cauterization, laser surgery, submucosal turbinoplasty using microdebrider, partial turbinectomy, and submucosal resection using special techniques, including radiofrequency ablation or quantum molecular resonance (QMR) techniques (24).

SECONDARY ATROPHIC RHINITIS treatment can be classified into medical and surgical options. In some patients, a multidisciplinary approach may be needed that involves ear, nose, and throat surgeons, allergists, and psychiatrists to manage this challenging disease; surgical choices are usually attempted only after an adequate trial of medical management, counseling, and education has not been able to improve for a period of 6 to 12 months, taking into account the potential neurologic mechanisms of SECONDARY ATROPHIC RHINITIS development (27).

Medical treatment

Medical treatment includes hydration with saline or oil-based lubricants, mucosal moisturization, increased fluid intake, intermittent closure of the nostrils (to return humidity), and an aggressive regimen of nasal saline sprays. For patients with psychological symptoms, cognitive behavioral therapy might help, and the careful use of newer-generation antidepressants might complement therapy (**25**).

2. Surgical management

Recently, surgical therapy for SECONDARY ATROPHIC RHINITIS has received excellent acceptance because evidence of beneficial outcomes in both nasal symptoms and the comorbid mental health sectors have been reported. (28)

The general concept of surgical intervention is to restore nasal physiologic function. Submucosal filler injections are ideal short-lived bulking agents to increase tissue deficit sites, and this procedure can be performed in the clinic. This measure provides time for total healing of the operated tissues, which may subside the patient's initial complaints. Young's approach to temporary nasal closure (either through fabricated plugs or tissue flaps) has been described as beneficial for AR disease. (8).

Most surgical treatments for SECONDARY ATROPHIC RHINITIS involve reshaping nasal cavities to act as previously resected nasal tissue. These procedures are considered turbinate reconstruction procedures. Fortunately, reconstruction of the lower turbinate may be accomplished at the lateral nasal wall at the level of the inferior meatus. Several materials have been used for submucosal grafting (8):

- 1. Temporary fillers.
- 2. Autologous cartilage.
- 3. Acellular dermal allografts.
- 4. Synthetic implants.

(1) Temporary Fillers

The submucosal filling of cross-linked hyaluronic acid has been described as a minimally invasive option in patients with SECONDARY ATROPHIC RHINITIS. In this description, the commercial product Juvéderm (Allergan, Dublin, Ireland) was used, but a variety of similar products are available; these alternatives are likely to offer similar usefulness (**29**).

(2) Autologous cadaver cartilage and the donor

The ease of harvest, biocompatibility, and reduced material cost of autologous cartilage make this an attractive option for SECONDARY ATROPHIC RHINITIS surgery. The objectives of cartilage grafting are similar to those of allograft placement, and the cartilage placement techniques are analogous to allograft placement, techniques. However, cartilage has the upper hand of a semirigid consistency and thus can be formed and sized to achieve the desired 3D shape. Autologous cartilage harvested from the nasal septum, ear, or rib can be carved and built to form a cylindrical shape before placing it in a submucosal pocket to simulate the lost turbinate tissue and narrow the nasal airway. Because many patients with SECONDARY ATROPHIC RHINITIS do not have adequate septal cartilage due to previous septoplasty procedures, costal or auricular cartilage may be required if the surgeon wants to use an autologous cartilage graft (**30**).

(3) Acellular Dermis Xenografts and Allografts

The submucosal implantation of acellular dermis allografts is well-described for the surgical management of SECONDARY ATROPHIC RHINITIS .² Human acellular dermal matrix (such as Alloderm, Allergan, Inc.) and porcine submucosal intestine submucosa are outlined to integrate into surrounding soft tissues over time. To place these grafts, submucosal pockets are made under GA in the target location (typically the lateral wall of the inferior meatus). The graft material can then be tightly rolled and packed inside the pocket to narrow the overly patent nasal passage. Multiple sites along the lateral wall of the nasal cavity can be targeted for external graft placement. The locations of obvious tissue loss guide the implant site. In general, the data show the great efficacy of this procedure with lasting benefits. Some users have noted resorption and failure rates of 10 to 30% for matrix materials. Fragmentation of the intestinal submucosal membrane with loss of neo-turbinate projection has been reported, making this xenograft unfavorable for this application (**31**).

(4) Synthetic implants

The use of synthetic implants has been studied in SECONDARY ATROPHIC RHINITIS reconstructive surgery. These implant types include silicone perforated sheeting or porous polyethylene (MedPor, Stryker Corporation, Kalamazoo, MI, USA). These materials do not react and are designed to allow tissue growth to penetrate the material and improve long-term integration. Synthetic implants are generally inexpensive and easy to obtain in most practices. Since synthetic implants are foreign bodies by nature, infection, and extrusion are possible complications. After tissue ingrowth, implant removal due to scarring is challenging if such extraction is warranted (**32**).

Medpor microplasty

The aims of the endonasal surgery in AR are to reduce nasal cavity volume, increase resistance to the airflow, reduce the airflow to increase air humidity, and deviate the airflow from the surgical site toward a healthy or a non-operated side. The creation of a neo turbinate is the most common surgical solution for SECONDARY

ATROPHIC RHINITIS. Techniques vary from team to team, but the results have been very encouraging. The principle consists of positioning an implant in a pocket in the septum, floor, or lateral wall of the nose (24). The location of the implant is based on the patient's history, examination, computed tomography scan findings, and the results of the cotton test in the office. Patients who gain no benefit from the cotton test are deemed poor candidates for implantation. The location of the implant should recreate the natural airflow patterns within the nose. To simulate the inferior turbinate, the implant is placed at the septum, floor, or the lateral nasal wall. As the head of the natural inferior turbinate enters the nasal valve region, the graft should be sufficiently anterior to replace the former inferior turbinate head (33).

A septal implant located anteriorly might function similarly. The lateral wall implant is tethered by the nasolacrimal duct and does not extend sufficiently to the anterior area, and thus may not provide adequate relief (34)

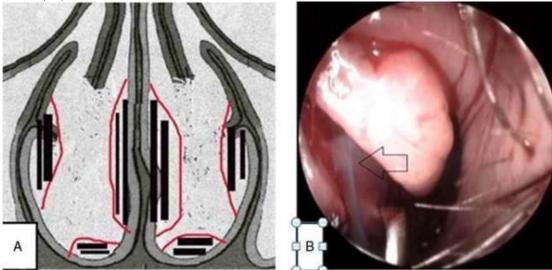


Figure 1: (a) Implants are inserted into pockets along the nasal septum, the floor of the nose, and along the lateral nasal wall on each side. (b) Intraoperative endoscopic view: sialastic implant (arrow) is introduced after raising the left mucoperichondrial flap (34)

The material used to reduce the nasal cavity volume should have cartilage-like elasticity, immunologic inertness, and combine minimal risk of extrusion, rejection, and infection with sufficient restoration of nasal cavity volume. Various materials are available including autologous (bone, cartilage, and fat) and exogenic materials (hydroxyapatite, goretex, teflon, plastipore). Although all of the synthetic implants may be effective, the use of autologous materials, such as cartilage, is considered ideal as it is cheap and available with a high level of biocompatibility. General evidence indicates long-term positive outcomes associated with the use of cartilage implants in rhinological surgeries. Septal cartilage is the most common material used in rhinological grafts; however, it is usually not enough in some patients, especially those who have undergone previous septal surgery. Conchal and costal cartilages can also be used in the procedure (**35**).

Physical properties

The Medpor implant is made of a medical-grade, high-density polyethylene that is sintered to create a somewhat flexible framework of interconnecting pores. The pore size ranges from 160 to 368 μ m and more than half of these pores are larger than 150 μ m in diameter. Since 1985, MEDPOR porous polyethylene implants have provided you with a range of off the shelf, biocompatible implant options for esthetic and reconstruction. Microthin, ultrathin, and sheet form is ready up to 0.25–3 mm. The vascular and fibrous ingrowth leads to excellent integration and stabilization of the implant and decreased rates of infection once the implant complex has formed. Mainly, thin Medpor has been used for the nose in Asia. Thin Medpor is used for the septal application, and thick Medpor is used for nasal dorsum (**36**).

Surgical technique

Implantation is performed with local anaesthesia (lidocaine, 1%, and epinephrine, 5 μ g/mL), which is administered at the submucosa of the inferior turbinate and/or lateral nasal wall and/or nasal septum opposite

the nasal concha. To create a submucosal pocket, an incision is made at the inferolateral side of the lateral nasal wall, just below the inferior turbinate remnant. For some seriously affected patients, the incision also extends to the mucosa of the nasal floor and/or nasal septum, opposite the site of the missing inferior turbinate. In these patients, the graft is placed at the septum or floor anteriorly. Care was taken not to obstruct the integrity of the sub-mucoperiosteal flap in order to ensure good vascular supply, which promotes the survival of the implants. After elevating the submucoperiosteal flap, a pocket is filled with the surgical Medpor implant to create a neoturbinate (**37**).

To simulate an inferior turbinate, the graft is also placed at the septum or floor anteriorly. The Medpor implants can be cut into different sized strips from a $13 \times 38 \times 3$ -mm piece depending on the volume of missing tissue to rebuild an appropriate inferior turbinate. On the basis of each individual's requirements, the implant is implanted in 1 to 4 pieces ($3-13 \times 20-38 \times 3$ mm) in each cavity. The implant is carefully positioned under endoscopic surveillance, and the pocket is closed with 1-0 silk braided sutures to keep it in position. Vaseline gauze packing is placed on it for 2 days, and the patients receives prophylactic antibiotics for 1 week following implantation (**37**).

After surgery, postoperative follow-up at the outpatient clinic depends on the status of mucosal recovery and is usually performed once a week during the first month and then every 3 months after surgery. Two weeks after surgery, AR patients are instructed to perform nasal douching with warm saline and to use intranasal corticosteroids (21).

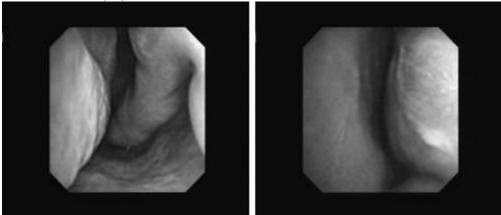


Figure 2: Endoscopic pictures of a preoperative nasal cavity (left) and a left inferior neo turbinate created with a Medpor implantation 12 months after surgery (right) (11)

Outcome measurements

The data of symptom scores, endoscopy, CT scan, mucociliary clearance (MCC), symptom scores of the SNOT-20, and acoustic rhinometry (Danish Rhinomanometer Rhino Scan 2000, Interacostics A/S, Assens, Denmark) are gathered before and 3, 6, and 12 months after surgery. SNOT-20 is a validated 20-item survey that examines general nasal symptoms and can be used for a severity comparison before and after some type of interventions; each item is scored from 0 (no symptoms) to 5 (severe symptoms) (**11**)

The mean value of nasal resistance (NAR), nasal volume (NV), and nasal minimum cross-sectional area (MCA) through an acoustic rhinometry examination are also evaluated. NAR is calculated according to Ohm's law: 1 / R = 1/Rr + 1/Rl (R = total nasal resistance, Rr = right nasal cavity resistance, Rl = left nasal cavity resistance). NV is calculated according to the following formula: NV = Vr + Vl (NV = total nasal volume, Vr = volume of the right nasal cavity, Vl = volume of the left nasal cavity). MCA is the average cross-sectional area of the bilateral nasal cavities. The assessment of MCC is determined with the saccharine method (**11**).

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