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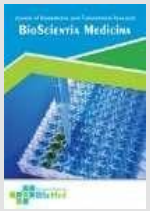

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Maternal Hyperthyroidism and Delayed Diagnosis of Bilateral Choanal Atresia in a 4-Month-Old Infant: A Case Report on Stentless Endoscopic Reconstruction

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A B S T R A C T

Background: Bilateral choanal atresia (BCA) is a life-threatening congenital anomaly typically presenting as a neonatal respiratory emergency. Survival beyond the neonatal period without surgical intervention is exceptionally rare. While the etiology is multifactorial, emerging evidence implicates maternal thyroid dysregulation in craniofacial malformations. This study reports a rare case of BCA diagnosed in a 4-month-old infant and evaluates the efficacy of stentless endoscopic repair using laterally-based mucoperiosteal flaps. **Case presentation:** A 4-month-old female infant presented with failure to thrive (weight 5.2 kg, less than the 3rd percentile) and cyclical respiratory distress. Perinatal history revealed the mother had Graves' disease and discontinued methimazole at 6 weeks gestation. Retrospective analysis of maternal serum indicated uncontrolled thyrotoxicosis during the critical organogenesis window (TSH less than 0.01 mIU/L; fT4 2.8 ng/dL at 7 weeks). Diagnostic imaging confirmed mixed bony-membranous atresia. The patient underwent transnasal endoscopic choanoplasty using a laterally-based mucosal preservation technique. A 10-Fr silicone feeding tube was placed transnasally but did not function as a structural stent. **Conclusion:** The intervention resulted in immediate airway patency. Quantitative outcomes showed an increase in oxygen saturation from 96% to 99% on room air and significant weight gain from 5.2 kg to 6.7 kg over two months. Follow-up at six months showed no restenosis. This case suggests a potential dual-hit teratogenic mechanism involving early methimazole exposure and subsequent uncontrolled maternal hyperthyroidism. Furthermore, it supports the efficacy of stentless repair in minimizing granulation tissue formation.

1. Introduction

Choanal atresia represents one of the most clinically significant congenital anomalies of the upper airway, characterized by the complete obliteration of the posterior nasal aperture. This disruption creates a physical barrier between the nasal cavity and the nasopharynx, fundamentally altering the respiratory physiology of the newborn.[1] The genesis of this pathology is rooted in the intricate embryological events of the first trimester. Specifically, it results from a failure in the canalization of the choanae, a process that normally concludes between the fourth and eleventh weeks of fetal development. Two primary theories govern this dysregulation: the persistence of the nasobuccal membrane of Hochstetter due to a failure to rupture, or an abnormal mesodermal flow that creates adhesion rather than cavitation.[2]

Epidemiologically, the condition is relatively rare but impactful, with an estimated incidence ranging from 1 in 5,000 to 1 in 7,000 live births.[3] Demographic analyses consistently demonstrate a sexual dimorphism, with a female-to-male ratio of approximately 2:1. Anatomically, the obstruction has historically been classified based on the nature of the

atretic plate. While early literature suggested a predominance of purely bony (29%) or membranous (71%) types, the advent of high-resolution Computed Tomography (CT) has refined this understanding. Modern imaging confirms that the vast majority of cases present with a mixed pathology—a bony-membranous complex—characterized by the thickening of the medial pterygoid plates and the widening of the posterior vomer, often reinforced by a membranous diaphragm.[4]

Bilateral choanal atresia (BCA) is classically regarded as an immediate, life-threatening neonatal emergency.[5] The urgency of this condition is dictated by the unique physiology of the human neonate, who functions as an obligate nasal breather for the first 4 to 6 weeks of life. This dependency is not merely habit but an anatomical necessity; the high position of the neonatal larynx allows the epiglottis to interlock with the soft palate, creating a velopharyngeal seal. This configuration facilitates simultaneous suckling and breathing, a survival mechanism essential for nursing.

However, in the presence of BCA, this anatomical advantage becomes a fatal liability. Complete nasal obstruction presents immediately after birth with a pathognomonic clinical sign known as cyclic cyanosis. The infant experiences asphyxia and cyanosis while at rest or feeding (when the mouth is closed), which is paradoxically relieved by crying (when the mouth is forced open), only to return once the infant settles and the oral airway closes again. Survival beyond the neonatal period without surgical intervention is exceptionally rare. It implies that the infant has defied physiological norms by developing a maladaptive oral breathing strategy—a learned behavior that usually requires months of neuromuscular maturation—thereby creating a survival paradox that challenges standard clinical paradigms.[6]

The etiology of choanal atresia remains a subject of intense investigation, residing at the intersection of genetic predisposition and environmental teratogenicity.[7] While approximately 50% of cases are associated with well-defined syndromic complexes—most notably CHARGE syndrome (Coloboma, Heart defect, Atresia choanae, Retarded growth, Genital hypoplasia, Ear anomalies)—the remaining isolated cases often present a diagnostic enigma. Recent literature has increasingly scrutinized the role of maternal thyroid homeostasis in craniofacial morphogenesis. Thyroid hormones (TH) are not merely metabolic regulators but are potent transcription factors essential for the migration and differentiation of neural crest cells, which form the skeletal framework of the face. Both ends of the thyroid spectrum have been implicated in midline defects. Maternal hyperthyroidism, characterized by uncontrolled thyrotoxicosis, can disrupt fibroblast growth factor (FGF) signaling pathways crucial for facial development. Conversely, the treatment of this condition using thioamides, specifically methimazole, carries its own risks. Methimazole embryopathy is a recognized teratogenic phenotype characterized by choanal atresia, aplasia cutis, and gut malrotations. Clinically, distinguishing whether the malformation is a result of the teratogenic drug (methimazole) or the teratogenic disease state (uncontrolled hyperthyroidism) is challenging, particularly when medication is altered during the critical organogenesis window.[8]

The management of choanal atresia has undergone a profound evolution. Historical approaches, such as transpalatal and transseptal techniques, were often associated with significant morbidity, including palatal dysfunction and disturbance of midfacial growth. These have largely been superseded by transnasal endoscopic approaches, which offer superior visualization, precise instrumentation, and reduced operative trauma.[9]

Despite these advancements, a significant area of debate persists in current otolaryngology practice: the use of postoperative stents. Traditionally, stents were considered mandatory to splint the newly created choanae and prevent collapse. However, a growing body of evidence suggests that stents may be counterproductive. Acting as foreign bodies, stents

can promote biofilm formation (often harboring *Staphylococcus aureus* or *Pseudomonas aeruginosa*) and induce circumferential inflammation. This inflammatory response triggers the formation of granulation tissue, which acts as a scaffold for restenosis—the very complication stents are intended to prevent. Consequently, modern rhinology is witnessing a paradigm shift toward stentless techniques. This approach relies on the meticulous preservation of mucoperiosteal flaps to cover raw bony surfaces (biological dressing), thereby facilitating primary intention healing and minimizing the fibroblastic activity that leads to scarring.[10]

Current literature is replete with reports on neonatal BCA repair; however, documentation of uncorrected survival into infancy is exceedingly sparse. This study aims to report the rare survival of a 4-month-old infant with undiagnosed bilateral choanal atresia, presenting a unique opportunity to analyze the physiological adaptations of delayed diagnosis. The novelty of this study is twofold: First, we reconstruct a precise teratogenic timeline to propose a double-hit hypothesis involving both early methimazole exposure and subsequent uncontrolled maternal hyperthyroidism, offering new insights into the environmental etiology of the disease. Second, we demonstrate the efficacy of a stentless endoscopic approach utilizing laterally-based mucoperiosteal flaps in an older infant, providing evidence that this technique yields superior functional patency and minimizes morbidity even outside the neonatal period.

2. Case Presentation

Written informed consent was obtained from the patient's parents for the publication of this case report and any accompanying images. The parents were fully briefed on the surgical procedure, potential risks, and the use of their clinical data for academic purposes, ensuring compliance with ethical standards for clinical research and the Declaration of Helsinki.

A 4-month-old female infant was referred to the Department of Otorhinolaryngology-Head and Neck Surgery at Dr. M. Djamil General Hospital, Padang, with a chief complaint of chronic difficulty breathing and inability to breastfeed continuously since birth. The patient presented with significant failure to thrive, weighing only 5.2 kg at the time of admission, which is less than the 3rd percentile for age (Z-score -2.1 SD). Perinatal history revealed the infant was born at 40 weeks gestation via cesarean section due to premature rupture of membranes. Birth weight was 3.1 kg. Immediately following delivery, the neonate exhibited a weak cry and respiratory distress, necessitating admission to the Neonatal Intensive Care Unit (NICU) for one month. During this period, nutrition was administered via an orogastric tube. Despite repeated desaturations during feeding attempts, the diagnosis of choanal atresia was missed, and the patient was discharged with a diagnosis of laryngomalacia and instructions for upright feeding (Table 1).

A detailed retrospective anamnesis was conducted to establish a precise teratogenic timeline regarding the patient's prenatal exposure. The 32-year-old mother had a documented history of Graves' disease diagnosed one year prior to conception and was maintained on a daily regimen of Methimazole 10 mg. She continued this pharmacological therapy through the first six weeks of gestation; however, upon sonographic confirmation of pregnancy at gestational week 6, she abruptly discontinued the medication without medical consultation due to fear of potential teratogenicity. Subsequently, between gestational weeks 7 and 12, the mother reported a resurgence of thyrotoxic symptoms, including palpitations, heat intolerance, and tremors. Biochemical analysis of archived maternal records from week 7 corroborated this clinical flare, revealing a suppressed TSH of less than 0.01 mIU/L (Reference: 0.1-2.5 mIU/L) and an elevated Free T4 of 2.8 ng/dL (Reference: 0.8-1.5 ng/dL), confirming a state of uncontrolled thyrotoxicosis during the critical window of palatogenesis.

Table 1. Summary of Clinical Findings on Admission

Category	Clinical Findings	Interpretation / Significance
Patient Profile	**Age:** 4 Months **Sex:** Female **Chief Complaint:** Chronic difficulty breathing, inability to breastfeed continuously.	*Delayed diagnosis (typically neonatal presentation).*
Anthropometry	**Weight:** 5.2 kg **Percentile:** < 3rd Percentile **Z-Score:** -2.1 SD	***Failure to Thrive*** *Indicates high metabolic cost of respiratory effort.*
Vital Signs	**Heart Rate:** 138 bpm **Resp. Rate:** 48 bpm (Tachypnea) **SpO2 (Rest):** 94-96% (Room Air) **SpO2 (Activity):** Drops to **88%** during crying/feeding	*Cyclic cyanosis / Paradoxical cyanosis relieved by crying (mouth breathing).*
Airway Examination	• Audible stertor • Mild intercostal retractions • Mucoid pooling in nasal cavities	*Signs of upper airway obstruction and increased work of breathing.*
Functional Assessment	**Catheter Test:** 6 Fr suction catheter failed to pass >3cm bilaterally. **Mirror Mist Test:** Negative (No condensation).	***Confirmed Obstruction*** *Physical confirmation of bilateral choanal atresia.*
Maternal History (Teratogenic Risk)	**Diagnosis:** Graves' Disease **Medication:** Methimazole (Weeks 0-6) **Biochemistry (Week 7):** TSH <0.01 mIU/L; fT4 2.8 ng/dL	***Double-Hit Hypothesis*** *Exposure to both drug (Methimazole) and uncontrolled thyrotoxicosis during organogenesis.*
Family Social History	**Paternal Status:** Active smoker (1 pack/day) **Exposure:** Indoor household smoking	***Confounding Factor*** *Potential for paternal DNA fragmentation and environmental pollutant exposure.*

Furthermore, the family history identified a significant confounding variable: the father was an active smoker consuming one pack per day within the household, thereby introducing potential risks associated with paternal DNA fragmentation and environmental smoke exposure. Physical examination upon admission revealed an infant exhibiting audible stertor and mild intercostal retractions. Vital signs recorded a heart rate of 138 beats per minute and a respiratory rate of 48 breaths per minute, with oxygen saturation (SpO_2) fluctuating between 94% and 96% on room air at rest but desaturating to 88% during episodes of crying or feeding. Anterior rhinoscopy demonstrated mucoid pooling within the nasal cavities (Figure 1), while functional assessment confirmed complete obstruction; a 6 Fr suction catheter failed to

pass more than 3 cm through either nostril, and a mirror mist test was bilaterally negative. No external dysmorphic features indicative of syndromic associations, such as CHARGE, Treacher Collins, or Apert syndromes, were observed. Diagnosis was definitively confirmed via non-contrast computed tomography (CT) of the paranasal sinuses, where axial and sagittal views demonstrated thickening of the posterior vomer and medial pterygoid plates consistent with bilateral mixed bony-membranous choanal atresia (Figure 2). Quantitative analysis revealed the right atretic plate measured 3.2 mm in thickness and the left 2.8 mm, with the membranous component accounting for approximately 30% of the obstruction. Secondary radiological findings included fluid accumulation consistent with inspissated mucus (20 to 32 Hounsfield Units) in the nasal cavity, alongside maxillary sinus hypoplasia resulting from the absence of nasal airflow pneumatization.

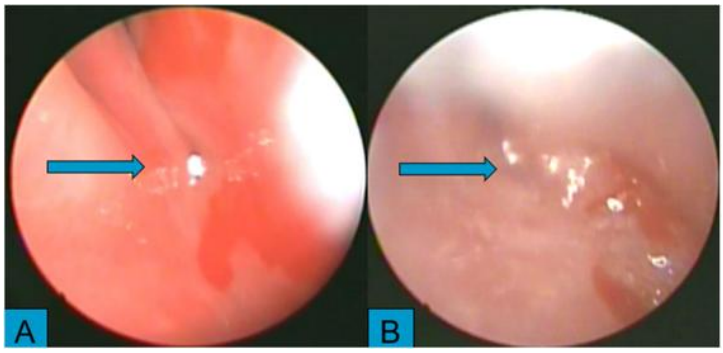

Figure 1. Appearance of bilateral choanal atresia (blue arrow). (A) right nasal cavity; (B) left nasal cavity.

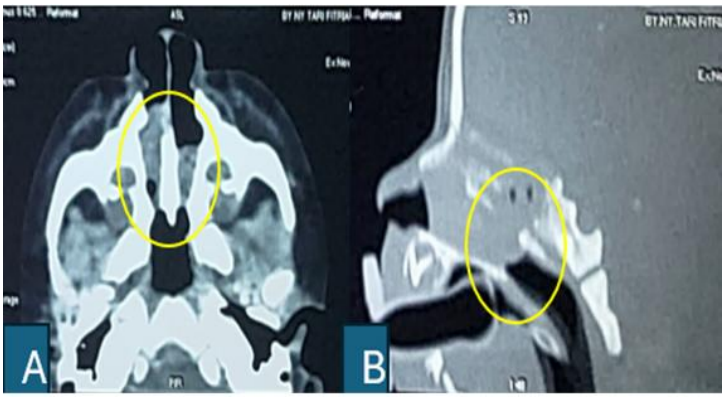

Figure 2. Coronal and sagittal CT scan images of the paranasal sinuses showing bilateral nasal cavity opacification, predominantly on the right side, and bilateral choanal atresia (yellow circles).

The definitive management of the patient's bilateral choanal atresia was achieved through a transnasal endoscopic reconstruction performed under general anesthesia. The surgical strategy prioritized a stentless philosophy, focusing on mucosal preservation to minimize the risk of postoperative restenosis. Initial preparation involved meticulous topical decongestion using 1:100,000 epinephrine pledgets to ensure optimal hemostasis and visualization within the narrow pediatric nasal corridor (Table 2). Visualization was maintained using a 0-degree 2.7 mm pediatric endoscope, which allowed

for precise instrumentation.

The cornerstone of the reconstruction was the design and elevation of a laterally-based mucoperiosteal flap. This delicate procedure involved making a vertical incision medially near the vomer and a horizontal incision superiorly. The flap was then carefully elevated toward the lateral wall, a trajectory specifically chosen to preserve the critical vascular supply derived from branches of the sphenopalatine artery. Following soft tissue elevation, the bony atretic plate was perforated and reduced utilizing a powered diamond burr. To create a unified and spacious posterior nasopharyngeal airway—termed the neochoana—a posterior vomerectomy was performed, and the aperture was widened to an approximate diameter of 16 French (Fr).

Crucially, the preserved mucoperiosteal flaps were then rotated to cover the raw bony edges of the lateral nasal wall and the remnant vomer. This step serves as a biological barrier, preventing the exposure of raw bone that typically triggers osteoneogenesis and circumferential scarring. Regarding the controversy of stenting, a nuanced approach was employed. A silicone 10 Fr feeding tube was passed transnasally, but its function was distinct from that of a traditional stent. Because the tube (10 Fr) was significantly smaller in caliber than the newly created choanae (16 Fr), it did not exert mechanical pressure on the mucosal walls. This floating configuration effectively avoided the pressure-induced ischemia and necrosis associated with structural stenting, with the tube serving solely as a nutritional conduit and a central anatomical guide during the immediate healing phase.

The immediate postoperative course validated the efficacy of this physiological approach. The patient was successfully extubated in the operating room, exhibiting an immediate and marked improvement in respiratory mechanics. The recovery was uncomplicated; the patient was discharged on the third postoperative day, and the nasogastric tube was removed on day five following the successful establishment of oral breastfeeding. Subsequent follow-up examinations provided objective evidence of surgical success. At three weeks, nasoendoscopy revealed patent choanae bilaterally with complete mucosalization (greater than 95%) and a notable absence of granulation tissue, confirming the utility of the mucosal flap technique. By the two-month interval, the restoration of a patent airway had translated into systemic physiological benefits, evidenced by a weight increase to 6.7 kg (representing a Z-score improvement to -0.8 SD) and stable oxygen saturation of 99% on room air. Long-term stability was confirmed at the six-month mark via flexible laryngoscopy, which demonstrated widely patent choanae with no evidence of restenosis or scar contracture.

3. Discussion

The clinical trajectory of this 4-month-old infant, presenting with the rare survival paradox of untreated bilateral choanal atresia (BCA), serves as a focal point for examining three critical domains in modern otolaryngology: the intricate environmental regulation of craniofacial embryogenesis, the physiological plasticity of the infant airway, and the evolving surgical philosophy regarding stentless endoscopic reconstruction. By integrating the retrospective maternal history with the intraoperative findings, this case highlights the necessity of a multidisciplinary approach that spans from perinatology to pediatric rhinology.

The etiology of non-syndromic choanal atresia remains one of the most challenging areas of craniofacial research. While genetic mutations (CHD7 in CHARGE syndrome) are well-documented, isolated cases often lack a definitive genetic corollary, pointing instead to environmental or teratogenic disruptions during critical windows of organogenesis. The development of the nasal cavity relies on a precise sequence of events between the 4th and 11th weeks of gestation: the invagination of the nasal placodes, the formation of the nasal sacs, and, crucially, the thinning and subsequent rupture of the buccopharyngeal (nasobuccal) membrane.[11]

Table 2. Summary of Diagnosis, Treatment, Follow-up, and Outcomes

PHASE	DETAILED SPECIFICATIONS	CLINICAL RATIONALE / NOTES
Diagnostic Imaging	**Modality:** Non-contrast Computed Tomography (CT) of Paranasal Sinuses. **Classification:** Bilateral mixed bony-membranous choanal atresia. **Measurements:** • Right Atretic Plate: **3.2 mm** • Left Atretic Plate: **2.8 mm** • Membranous Component: ~30%	*Secondary findings included fluid accumulation (* ***20-32 HU*** *) and maxillary sinus hypoplasia due to lack of nasal airflow.*
Surgical Treatment	**Technique:** Transnasal Endoscopic Reconstruction (Stentless). **Key Steps:** • **Preparation**: 1:100,000 Epinephrine pledgets. • **Flap**: Laterally-based mucoperiosteal flap preservation. • **Resection**: Powered diamond burr & posterior vomerectomy. • **Neochoana**: Widened to ~16 Fr diameter.	*Mucosal preservation is critical to cover raw bony edges, preventing osteoneogenesis and reducing reliance on structural stenting.*
Intraoperative Adjuvants	**Device:** Silicone 10 Fr Feeding Tube. **Placement:** Transnasal (Left nostril). **Purpose:** Nutritional support and central guide.	***NOT A STENT*** *Tube caliber (10 Fr) was significantly smaller than the opening (16 Fr), avoiding pressure necrosis on lateral walls.*
Postoperative Timeline	• **Immediate:** Successful extubation; improved respiratory mechanics. • **Day 3:** Patient discharged. • **Day 5:** Feeding tube removed; oral breastfeeding established.	*Rapid recovery indicates minimal tissue trauma associated with the endoscopic approach.*
Long-term Surveillance	**3 Weeks (Nasoendoscopy):** Patent choanae, >95% mucosalization, no granulation. **6 Months (Flexible Laryngoscopy):** Widely patent choanae, no restenosis.	*Absence of granulation tissue at 3 weeks validates the stentless, mucosal-sparing philosophy.*
Final Clinical Outcomes	**Respiratory:** SpO2 stable at **99%** (Room Air). **Growth (2 Months):** • Weight: Increased from 5.2 kg → **6.7 kg** • Z-Score: Improved from -2.1 SD → **-0.8 SD**	*Correction of airway obstruction resolved the survival paradox, allowing simultaneous breathing and feeding, reversing failure to thrive.*

Failure of this membrane to rupture, or the abnormal persistence of mesodermal flow between the palatine and vomerine processes, results in atresia. In this case, we propose a double-hit Hypothesis to explain the failure of recanalization, postulating a synergistic toxicity arising from sequential exposure to a teratogenic pharmacologic agent and a teratogenic metabolic state. The first hit involves the maternal use of Methimazole during the earliest phase of gestation. Methimazole is a thioamide drug classified with known teratogenic potential. The phenotype of methimazole embryopathy is well-characterized, encompassing choanal atresia, aplasia cutis congenita, and esophageal dysmotility.[12] The mechanism is believed to involve the disruption of localized angiogenesis and the inhibition of neural crest cell migration. Although the mother in this case discontinued the medication at week 6, the pharmacokinetics of methimazole allow it to cross the placenta freely. Given the drug's half-life and the potential for tissue accumulation, it is biologically plausible that the embryo remained exposed to residual therapeutic levels during the initiation of the critical 4th-week window, potentially priming the nasal mesoderm for dysregulation. The second, and perhaps more devastating hit, was the subsequent onset of uncontrolled maternal thyrotoxicosis following medication cessation. Biochemical reconstruction places the mother in a state of profound hyperthyroidism (TSH <0.01 mIU/L) precisely during the window when the buccopharyngeal membrane should be rupturing (Weeks 7-11). Thyroid hormones (T3 and T4) are not merely metabolic regulators; they are potent transcription factors essential for craniofacial skeletal maturation. T3 binds to nuclear thyroid hormone receptors (TRα and TRβ) in osteoblasts and chondrocytes, regulating the genes responsible for midline fusion and remodeling.[13]

Under physiological conditions, the fetus is protected from maternal T3/T4 fluctuations by the placental and fetal expression of Type 3 Deiodinase (DIO3), an enzyme that inactivates thyroid hormones, effectively shielding developing tissues from thyrotoxicosis.[14] However, recent evidence by Martinez et al. suggests a saturation point for this protective mechanism. When maternal thyrotoxicosis is profound, the capacity of DIO3 is overwhelmed, leading to supraphysiological levels of T3 in fetal tissues. This excess T3 can disrupt the fibroblast growth factor (FGF) signaling pathways, which are critical for the apoptosis of the buccopharyngeal membrane. Therefore, the fetus was subjected to a sequential assault: first, an inhibition of angiogenesis by methimazole, followed immediately by a hyperthyroid state that accelerated osteoblastic activity, likely resulting in the thick, mixed bony-membranous plate observed on the CT scan.[15]

While the maternal thyroid axis presents a compelling biological mechanism, scientific rigor demands the evaluation of all potential confounders. The patient's father was identified as an active smoker within the household. Contemporary reproductive biology has increasingly focused on the paternal contribution to congenital anomalies, a field often overshadowed by maternal factors. Paternal smoking is a potent source of reactive oxygen species (ROS), which can lead to DNA fragmentation and oxidative stress in spermatozoa.[16] Recent meta-analyses indicate that paternal smoking is an independent, significant risk factor for congenital malformations, specifically cardiac and craniofacial defects. The mechanism is likely epigenetic; smoke exposure can alter DNA methylation patterns in sperm, which are then transmitted to the zygote, affecting gene expression during embryogenesis. In this patient, the paternal genotoxicity may have lowered the threshold for malformation, creating a susceptible substrate upon which the maternal thyroid insults acted. This underscores the multifactorial nature of non-syndromic choanal atresia and highlights the importance of comprehensive parental counseling regarding environmental exposures.

The survival of this infant to 4 months of age without surgical intervention presents a significant physiological paradox. Anatomically, human neonates are obligate nasal breathers due to the high position

of the larynx (C3-C4 level), where the epiglottis interlocks with the soft palate. This velopharyngeal lock allows for simultaneous suckling and breathing but makes oral breathing mechanically difficult, if not impossible, during the first weeks of life.[17] Complete bilateral obstruction typically results in severe asphyxia, cyclic cyanosis, and potential mortality if an oral airway is not immediately established. We hypothesize that this patient survived via a precarious, learned adaptation. Over the first weeks of life in the NICU, the infant likely developed a maladaptive oral breathing strategy, utilizing conscious effort to depress the mandible and break the velopharyngeal seal to gasp for air. However, this survival mechanism came at a severe metabolic cost, manifested clinically as failure to thrive (FTT).[18]

The energetics of respiration in this infant were fundamentally deranged. An infant with BCA must choose between two vital functions: breathing (which requires an open mouth) and feeding (which requires a closed mouth for suction). This creates a breath vs. eat conflict. Every feeding session induces hypoxia, forcing the infant to detach and gasp, leading to insufficient caloric intake. Furthermore, the work of breathing (WOB) utilized to maintain an open oral airway against pharyngeal muscle tone significantly increases basal metabolic rate. Thus, the weight of 5.2 kg (Z-score -2.1 SD) represents a state of catabolic collapse: the infant was consuming fewer calories due to feeding difficulty while simultaneously burning excess calories to mechanically maintain an airway. The rapid weight gain observed post-surgery (1.5 kg in 2 months) confirms that the FTT was purely obstructive and metabolic in origin, rather than malabsorptive.[19]

The surgical management of this case reflects a decisive shift in modern rhinology away from the historical dogma of stenting. For decades, the placement of hard stents (portex or endotracheal tubes) was considered mandatory to splint the neochoanae and prevent restenosis. However, this practice was based on mechanical theory rather than biological evidence. We advocate for a stentless endoscopic approach, grounded in the pathophysiology of wound healing. Previous studies have elucidated the cellular mechanisms of stent failure. Stents act as foreign bodies within the nasal ecosystem. They inevitably become colonized by pathogenic biofilms, predominantly *Staphylococcus aureus* and *Pseudomonas aeruginosa*, which induce a chronic inflammatory response. This inflammation triggers the proliferation of fibroblasts and the formation of circumferential granulation tissue. Clinically, this manifests as stent-induced stenosis, where the scar tissue forms around the stent, leading to re-obstruction once the stent is removed. Furthermore, the mechanical pressure of a stent against the lateral nasal wall can cause ischemic necrosis of the mucosa, exposing more raw bone and perpetuating the cycle of osteoneogenesis.[20]

Our technique prioritizes the concept of the biological dressing. By designing a laterally-based mucoperiosteal flap, we actively cover the raw bony edges of the resected vomer and pterygoid plates. In secondary intention healing (leaving raw bone exposed), the body rushes to cover the defect with granulation tissue, which contracts and scars. By rotating vascularized mucosa over the defect (primary intention healing), we inhibit the inflammatory cascade. The flap acts as a physiological barrier, preventing the exposure of the underlying osteoblasts to inflammatory cytokines, thereby halting osteoneogenesis. The outcome in this case—patent choanae with healthy mucosa at 3 weeks and no restenosis at 6 months—validates this biological approach.

A critical methodological clarification in this study is the definition of the device used. We utilized a 10 Fr feeding tube, but we argue this does not constitute a stent in the surgical sense. A therapeutic stent is sized to fit tightly against the canal walls to provide mechanical opposition to collapse. In contrast, the neochoana created was approximately 16 Fr in diameter. A 10 Fr tube in a 16 Fr canal leaves a circumferential gap of 2-3 mm. It floats within the airway. Therefore, it exerts zero mechanical pressure

on the mucosal walls, avoiding pressure necrosis. Its function was strictly distinct: to provide a route for enteral nutrition while the infant relearned nasal breathing and to act as a central anatomical guide for suctioning. This loose-fit strategy allows for drainage and airflow around the tube, maintaining a physiological environment that is hostile to anaerobic biofilm formation.[17,18]

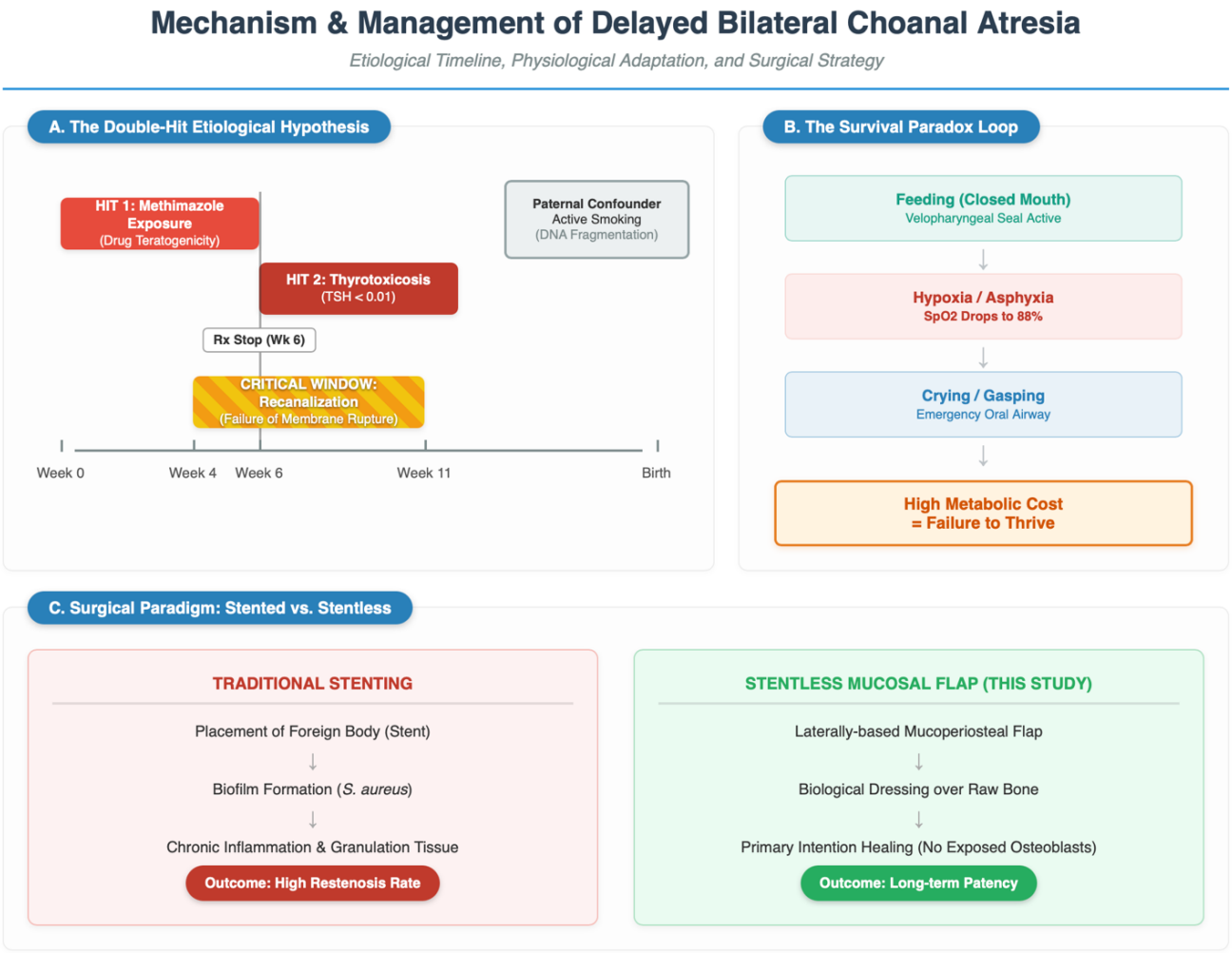

Figure 3. Mechanism and management of delayed bilateral choanal atresia.

While this study offers significant clinical and mechanistic insights, it is subject to the inherent limitations of a single case report (N=1). Case reports are hypothesis-generating rather than definitive; thus, the double-hit hypothesis, while biologically plausible, cannot be statistically validated without larger cohort studies comparing maternal thyroid profiles in BCA populations. Secondly, the absence of high-level genetic testing is a limitation. Although the patient exhibited no phenotypic features of syndromic disorders (such as colobomas or ear anomalies), sub-clinical mutations in genes such as CHD7, RBM10, or FGF8 cannot be definitively ruled out without whole exome sequencing (WES). It is possible that the teratogenic insults acted upon a background of genetic susceptibility. Finally, the follow-up period of 6 months, while sufficient to rule out immediate granulation-induced stenosis, does not cover the long-

term skeletal growth of the midface. Pediatric choanal atresia patients require surveillance until skeletal maturity to monitor for late stenosis due to relative hypoplasia of the nasopharynx as the child grows.[19,20]

4. Conclusion

The successful management of this 4-month-old infant with Bilateral Choanal Atresia serves as a potent testament to the resilience of pediatric physiology and the efficacy of biologically driven surgical techniques. This case report transcends the typical surgical technical note by integrating a rigorous retrospective analysis of maternal endocrinology, thereby illuminating a potentially preventable etiology for this devastating anomaly. Clinically, the survival paradox observed here—where an obligate nasal breather survives months of obstruction—teaches us that physiological adaptation is possible but comes at an immense metabolic cost, manifesting as severe failure to thrive. This underscores the need for a high index of suspicion for BCA in any infant presenting with feeding difficulties and failure to thrive, even in the absence of frank cyanosis.

Etiologically, we present the double-hit hypothesis as a cautionary framework for the management of thyroid disease in pregnancy. The intersection of early embryonic exposure to methimazole and subsequent exposure to uncontrolled thyrotoxicosis suggests a compounded teratogenic risk. This reinforces the necessity for strict, multidisciplinary management of maternal Graves' disease, with a goal of euthyroidism throughout the critical window of organogenesis. Surgically, this report adds to the growing body of evidence advocating for stentless endoscopic reconstruction as the gold standard of care. We have demonstrated that the meticulous use of laterally-based mucoperiosteal flaps allows for rapid epithelialization and prevents the granulation tissue formation notoriously associated with stenting. By shifting the focus from mechanical splinting to biological preservation, we can achieve superior functional outcomes with reduced morbidity. Future research should focus on longitudinal cohort studies to further validate the long-term patency rates of this technique and to explore the genetic-environmental interactions proposed in our etiological model.

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