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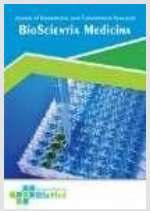

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Primary Furlow Palatoplasty for Delayed Repair of Veau Type I Cleft Palate in an Adolescent: Surgical Challenges and Bio-functional Outcomes

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A B S T R A C T

Background: Cleft palate is a pervasive congenital craniofacial anomaly characterized by the failure of palatal shelf fusion, resulting in a persistent communication between the oral and nasal cavities. While developed healthcare systems mandate repair between 9 and 12 months, delayed presentation in adolescence remains a distinct clinical entity in developing regions. These cases are surgically complex due to maxillary skeletal maturity, tissue fibrosis, and wider cleft gaps compared to infant cases. The primary surgical objective is to seal the defect and restore the velopharyngeal sphincter to prevent hypernasality. This study evaluates the efficacy and physiological advantages of the Furlow double opposing Z-plasty technique in a high-risk delayed primary repair scenario. **Case presentation:** We report the management of a 14-year-old male presenting with an uncorrected non-syndromic incomplete cleft palate. Preoperative assessment revealed severe hypernasality and audible nasal air emission. Clinical examination confirmed a Veau Type I defect confined to the soft palate with a bifid uvula. Primary palatoplasty was executed using the Furlow technique. The procedure successfully recruited lateral tissue for palatal lengthening and achieved transverse muscle reorientation without the need for lateral relaxing incisions. **Conclusion:** The intervention resulted in complete anatomical closure with no evidence of wound dehiscence, hemorrhage, or oronasal fistula formation. The Furlow technique proved to be a feasible and safe modality for Veau Type I defects in adolescents, effectively addressing the vertical pharyngeal gap and restoring the sphincter mechanism's anatomy.

1. Introduction

Cleft palate represents one of the most significant congenital malformations of the head and neck, imposing a profound burden on the functional pillars of human communication and nutrition.[1] Epidemiologically, the condition is substantial, with cleft lip and palate occurring in approximately 1 in 600 to 800 live births, while isolated cleft palate is observed in roughly 1 in 2,000 live births globally. The etiology is multifactorial, involving a complex interplay of genetic predisposition and environmental insults during the critical windows of palatogenesis.[2] While genetic factors account for a significant portion of cases, environmental risk factors such as maternal smoking, nutritional deficiencies, and intrauterine exposure to teratogens play a pivotal role in the sporadic presentation of the disease.[3]

The fundamental pathophysiology involves the failure of the palatal shelves to elevate and fuse in the midline. Beyond the structural gap, the most debilitating consequence of this failure is velopharyngeal dysfunction.[4] In a normal anatomical configuration, the levator veli palatini muscles form a coherent, dynamic sling that lifts the soft palate superiorly and posteriorly against the Passavant's ridge of the posterior pharyngeal wall. This action

seals the nasopharynx from the oropharynx, a mechanism essential for the production of oral consonants and the prevention of hypernasality. In cleft palate patients, this muscular integrity is disrupted; the muscle fibers insert abnormally into the posterior edge of the hard palate rather than joining in the midline. This renders the sling incompetent, leading to the hallmark symptoms of hypernasality, nasal air emission, and the development of compensatory articulation errors.[5]

In developed healthcare systems, the standard timeline for surgical repair is between 9 and 12 months of age. This timing is strategic, aimed at restoring the anatomical valve before the onset of canonical babbling and speech production.[6] However, surgeons in developing nations frequently encounter a different clinical reality: the neglected cleft. These are patients who present in adolescence or adulthood, having lived for over a decade with an uncorrected defect.[7] Operating on a 14-year-old primary cleft is not merely a large infant repair; it is a distinct surgical entity governed by different biomechanical rules. The challenges include skeletal maturity, where the maxilla has achieved significant transverse width while the palatal shelves have not grown commensurately, leading to a wider absolute gap. Furthermore, the mucoperiosteum in older patients lacks the pliability of infant tissue, making flap mobilization difficult and increasing the risk of tension. Finally, as the craniofacial skeleton grows vertically, the depth of the nasopharynx increases, requiring greater palatal length to achieve closure.[8,9]

While the Furlow double opposing Z-plasty is established as a gold standard for secondary speech correction and infant repair, its application as a primary modality for delayed repair in adolescents is under-documented and often avoided due to fears of tension-related fistula formation.[10] This study aims to fill this gap in the global surgery literature by elucidating the biomechanical safety profile and efficacy of the Furlow technique in an adolescent male with a Veau Type I cleft. We specifically aim to demonstrate that the geometric advantages of the Z-plasty can overcome the fibrotic and skeletal challenges of the adolescent palate without the need for lateral relaxing incisions, thereby challenging the prevailing dogma regarding high fistula rates in late-stage repair.

2. Case Presentation

The patient, a 14-year-old male adolescent, presented to the Otorhinolaryngology-Head and Neck Surgery (ORL-HNS) Outpatient Clinic, specifically within the Plastic and Reconstructive Facial Division at Dr. M. Djamil General Hospital, Padang, on January 6th, 2025. The primary impetus for his consultation was a significant functional impairment regarding his speech quality. The patient and his family reported a chief complaint of a distinct nasal voice (hypernasality) that had been persistent and unremitting since the onset of his speech development. This functional deficit had likely contributed to social and communicative challenges throughout his childhood and early adolescence, prompting the family to finally seek specialized surgical intervention, detailed in Figure 1.

A comprehensive anamnesis revealed that the patient had been diagnosed with a cleft palate immediately upon birth. However, due to various factors, surgical correction had been delayed until his current presentation at 14 years of age. The patient's history was notable for classic symptoms of velopharyngeal incompetence manifesting early in life. His mother reported significant feeding difficulties during infancy; specifically, every breastfeeding attempt was complicated by episodes of choking and coughing. Furthermore, there was a consistent history of nasal regurgitation, where milk or fluids would discharge through the nasal cavity after drinking, a hallmark sign of the anatomical communication between the oral and nasal cavities, detailed in Figure 1.

Developmentally, the patient’s speech milestones were delayed, with the initiation of speech reported at the age of 5 years. This delay is often associated with the inability to generate sufficient intraoral pressure

required for articulating sounds due to the palatal defect. Despite the chronic nature of the cleft palate, the patient's otologic history was surprisingly benign. He reported no sensation of aural fullness, no subjective hearing loss in either ear, and no history of otorrhea (ear discharge), suggesting that Eustachian tube dysfunction, while common in cleft palate patients, had not manifested as chronic suppurative otitis media in this specific case. Currently, aside from the speech impediment, the patient reported the ability to eat and drink without significant aspiration, likely due to compensatory swallowing mechanisms developed over the years, detailed in Figure 1.

The prenatal and perinatal history was thoroughly reviewed to rule out syndromic associations. The patient was born via spontaneous vaginal delivery with a healthy birth weight of 3400 grams. The antenatal period was uncomplicated; there was no history of maternal illness, trauma, or infection during the pregnancy. Crucially, there was no history of maternal smoking, alcohol consumption, or exposure to teratogenic agents, which are known environmental risk factors for orofacial clefts. The family history was strictly negative for genetic diseases, congenital abnormalities, heart defects, or any instances of cleft lip and/or palate in immediate or extended relatives. This constellation of historical findings strongly supported a diagnosis of a non-syndromic etiology, detailed in Figure 1.

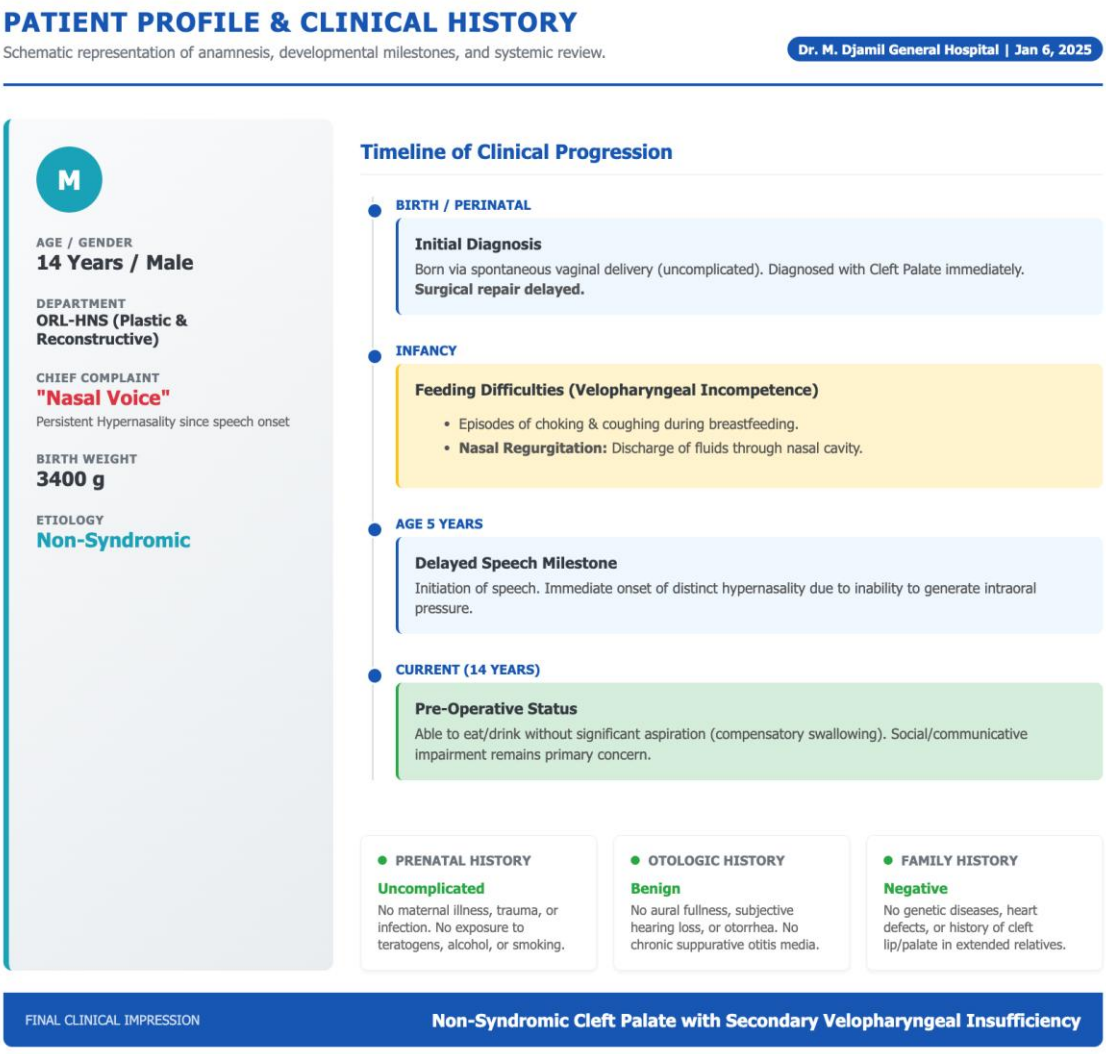

Figure 1. Patient profile and clinical history.

Upon physical examination, the patient appeared to be in moderate general condition but was fully alert, cooperative, and oriented (compos mentis). A detailed evaluation of the head and neck region was conducted. The patient was normocephalic with no evidence of craniofacial asymmetry or dysmorphology. Inspection of the face revealed that the lips were intact and within normal anatomical limits, ruling out a cleft lip component. Similarly, the external nose and ears showed no structural abnormalities. The intraoral examination provided the definitive anatomical characterization of the defect. Inspection of the oral cavity and oropharynx revealed a distinct midline cleft. The defect was incomplete, extending from the posterior aspect of the soft palate and involving the uvula, which was morphologically bifid (split in two). The hard palate was clinically intact, confirming the diagnosis of an incomplete cleft. The tonsils were graded as T1-T1 and appeared healthy, with no signs of hyperemia or chronic infection. The posterior pharyngeal wall was also non-hyperemic. Crucially, the separation of the palatal muscles was evident, indicating the disruption of the levator veli palatini sling, which is essential for velopharyngeal closure. Based on the clinical findings—an isolated cleft of the soft palate without involvement of the hard palate or alveolus—the patient was diagnosed with a non-syndromic incomplete cleft palate (specifically Veau Class I). Following a comprehensive preoperative assessment and counseling regarding the risks and benefits of the procedure, the patient was scheduled for primary palatoplasty, detailed in Figure 2.

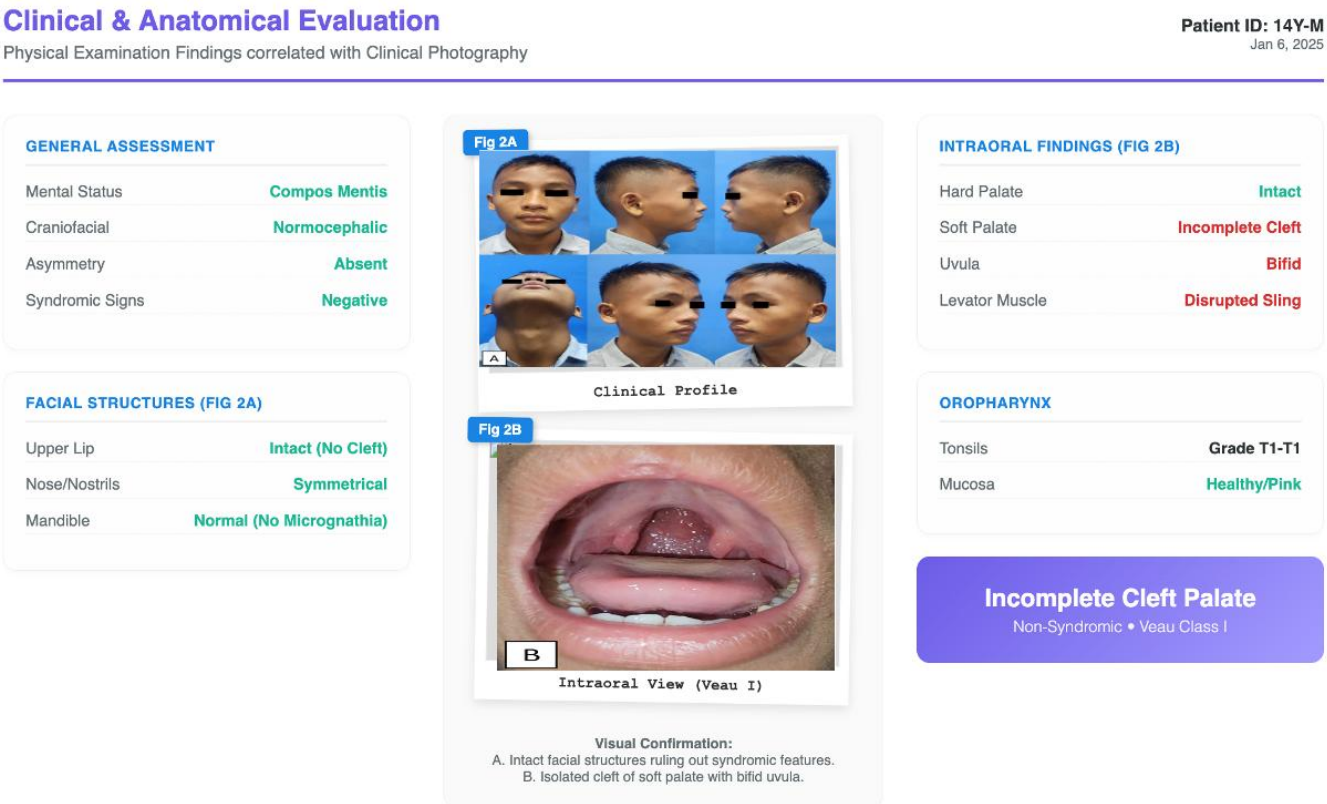

Figure 2. Clinical and anatomical evaluation.

The patient underwent reconstructive surgery under general anesthesia with endotracheal intubation. The surgical plan utilized the Furlow double opposing Z-plasty technique, a method selected for its superior ability to lengthen the soft palate and reconstruct the levator muscular sling, which is critical for correcting the velopharyngeal insufficiency observed in this patient. Preparation and Exposure: The patient was positioned in the Rose position, with the neck extended and the head slightly elevated above the level of the operating table to maximize exposure of the palatal vault and oropharynx. Strict aseptic and antiseptic protocols were observed to prepare the surgical field, followed by the application of sterile drapes. To maintain mouth opening and depress the tongue, a Boyle-Davis Mouth Gag equipped with a Russell-Davis tongue blade was carefully inserted and secured. A throat pack (gauze) was placed in the hypopharynx to prevent the aspiration of blood or fluids. Prior to the incision, the surgical site was infiltrated with a vasoconstrictive solution containing epinephrine diluted to 1:200,000. This infiltration was allowed to take effect for 10 minutes, serving the dual purpose of achieving hemostasis and facilitating hydro-dissection of the mucoperiosteal/muscular planes. Incision and Dissection: The procedure began with the paring of the cleft margins. Using a number 12 blade, the mucosal edges of the cleft on both the left and right sides were meticulously trimmed to create raw surfaces necessary for healing. The Furlow technique involves the design of two opposing Z-plasties: one on the oral mucosal surface and one on the nasal mucosal surface, oriented as mirror images of each other. Oral layer: The Z-plasty limbs were marked and incised on the oral mucosa. The dissection proceeded to raise the oral mucosal flaps. Muscle Dissection: A critical component of this surgery was the identification and dissection of the levator veli palatini muscles. In a cleft palate, these muscles are abnormally inserted into the posterior edge of the hard palate. These abnormal attachments were released, and the muscles were dissected free from the nasal mucosa but left attached to the oral mucosa in the posteriorly-based flap. This step allows for the retro-positioning and transverse reorientation of the muscle sling. Nasal layer: The opposing Z-plasty was designed on the nasal surface. The nasal flaps were raised, ensuring careful separation from the underlying structures. Closure and Reconstruction: The reconstruction proceeded in a layered fashion. First, the uvula was reconstructed by approximating the bifid halves using interrupted sutures with Vicryl 4.0, an absorbable polyglactin suture. Next, the nasal mucosal flaps were transposed and sutured using mattress sutures with Vicryl 4.0. This step effectively closed the nasal side of the defect and lengthened the nasal lining. Following the nasal closure, the muscle sling was reconstructed. The transposition of the Z-plasty flaps automatically carried the levator muscle bundles into a transverse orientation, overlapping them to recreate a functional sphincter mechanism. Finally, the oral mucosal flaps were transposed. The tip of the left oral flap was rotated and sutured to the contralateral side, and the tip of the right oral flap was sutured in the opposite direction. This double opposing transposition achieves significant lengthening of the soft palate at the expense of lateral width, without the need for lateral relaxing incisions. The remaining oral mucosal defect was closed with interrupted Vicryl 4.0 sutures until a watertight seal was achieved, ensuring complete closure of the cleft. Hemostasis: Intraoperative bleeding was meticulously controlled using electric cautery and gauze pressure. Upon completion of the suturing, the integrity of the repair was verified. A Nasogastric Tube (NGT) number 14 was inserted through the right nasal cavity to facilitate postoperative feeding and protect the surgical repair from mechanical stress during the initial healing phase. The throat pack and mouth gag were removed, and the patient was successfully extubated, as detailed in Figure 3.

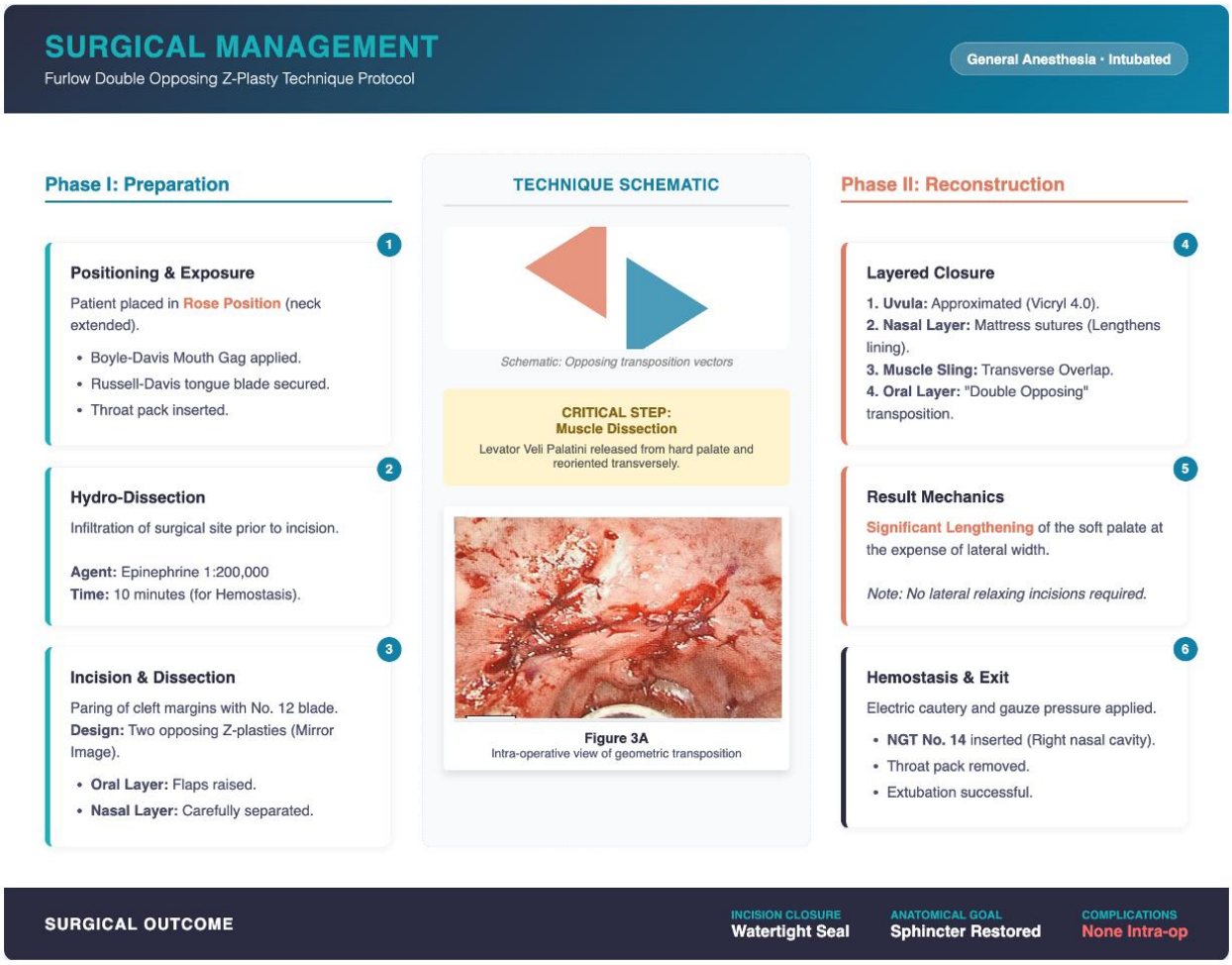

Figure 3. Surgical management.

Following the procedure, the patient was transferred to the ORL-HNS ward for postoperative monitoring. The diagnosis was updated to post-palatoplasty for incomplete cleft palate. The postoperative care regimen was strictly adhered to, ensuring optimal wound healing. Positioning: The patient was maintained in a semi-Fowler's position (Head up 40 degrees) to reduce postoperative edema and facilitate airway patency. Nutrition: To protect the delicate palatal sutures, the patient was kept on a strict liquid diet administered via the NGT. Medication: A regimen of intravenous antibiotics was administered to prevent surgical site infection, along with intravenous analgesics to manage postoperative pain and ensure patient comfort. Follow-up Outcomes: The patient was followed up longitudinally to assess healing and functional outcomes. Day 7: The surgical wound was inspected, revealing approximated edges with no active bleeding or dehiscence. Day 14: The sutures remained intact, and the inflammatory response was subsiding. Day 30: By the 30th postoperative day, the patient reported no subjective complaints such as fever, pain, or dysphagia (sensation of food sticking in the throat). The NGT had been removed, and the patient had successfully transitioned to oral intake of food and drink. Clinical examination revealed a fully healed palate. The suture line was intact with good tensile strength, and critically, there was no evidence of blood seepage or fistula formation. The successful

anatomical closure marked the completion of the surgical phase. The patient was subsequently referred to the Department of Physical Medicine and Rehabilitation to initiate speech therapy. This rehabilitative phase is essential to address the residual compensatory articulation patterns and to train the newly reconstructed velopharyngeal mechanism for normal speech production, detailed in Figure 4.

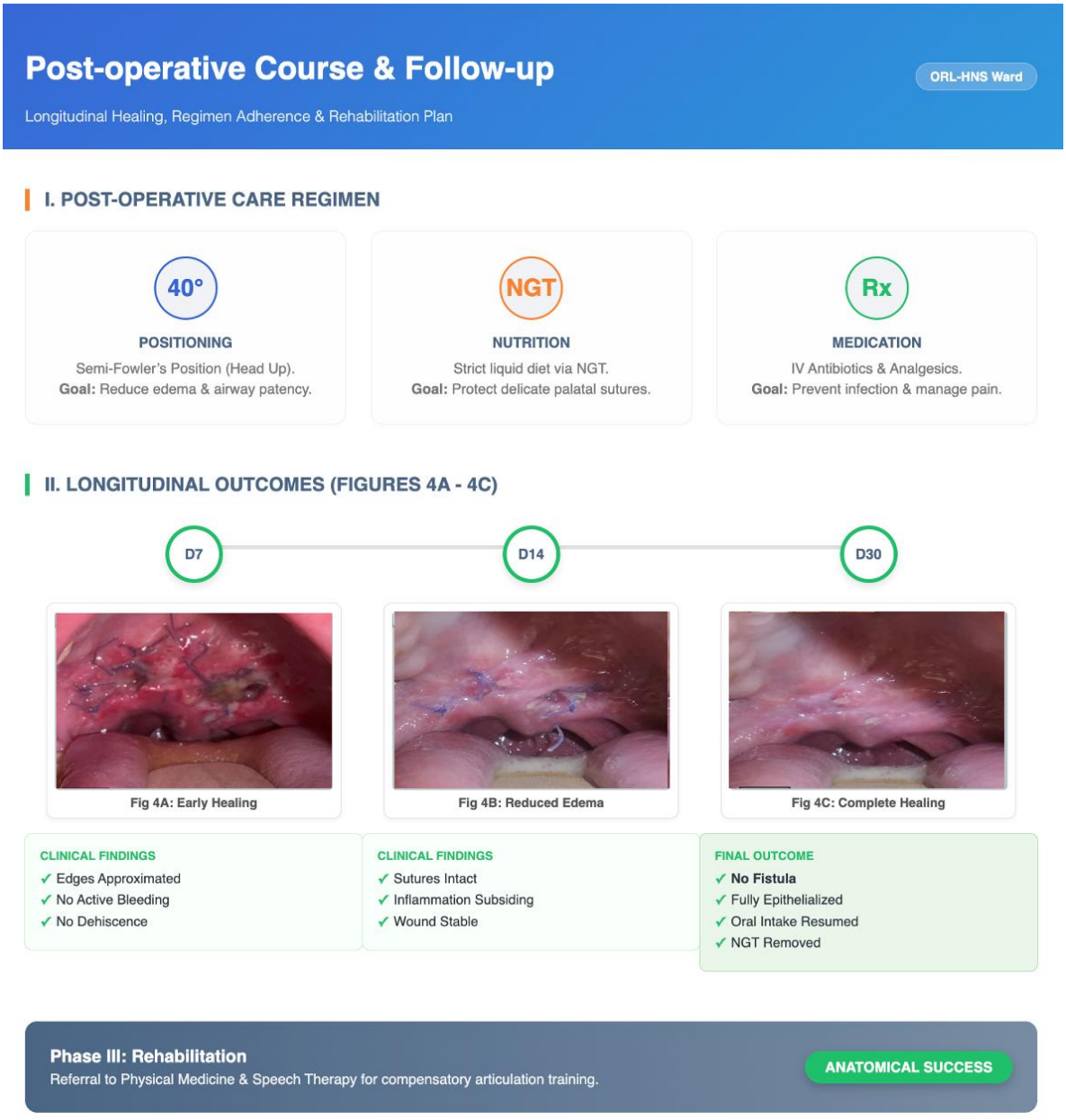

Figure 4. Post-operative course and follow-up.

3. Discussion

The presentation of a 14-year-old male with an uncorrected cleft palate represents a significant departure from the standard of care in developed nations, yet it highlights a persistent public health crisis in many regions of the Global South. The

neglected cleft is not merely a delayed surgery; it is a complex sociomedical entity. Patients in this demographic often suffer from prolonged social isolation, educational deficits due to speech impediments, and chronic nutritional challenges.[11] In this case, the patient had lived with severe hypernasality for over a decade. The delay in treatment allows for the entrenchment of maladaptive compensatory articulation patterns—such as glottal stops and pharyngeal fricatives—where the patient unconsciously attempts to generate intraoral pressure at the level of the larynx rather than the oral cavity. Surgical correction at this age, therefore, faces a dual challenge: the biological rigidity of the tissues and the neurological rigidity of the speech motor pathways.

The decision to operate on a 14-year-old palate requires a deep understanding of the differences between infant and adolescent oropharyngeal anatomy.[12] In infants, the tissues are rich in proteoglycans and possess a high degree of elasticity, allowing for significant mobilization with minimal tension. In contrast, the adolescent mucoperiosteum is more fibrous, less vascular, and significantly stiffer. This reduction in tissue compliance makes the creation and transposition of flaps mechanically demanding. Furthermore, the skeletal framework has matured. The maxilla has grown transversely, but the cleft defect often remains wide, increasing the absolute distance that soft tissue must bridge. Simultaneously, the vertical growth of the face results in a deeper nasopharynx. The distance from the hard palate to the posterior pharyngeal wall is greater in adolescents than in infants. A repair that merely closes the hole without adding length—such as a simple Von Langenbeck repair—is destined to fail functionally. The bowstringing effect of a straight-line scar can actually pull the soft palate anteriorly as it contracts, exacerbating the velopharyngeal gap despite achieving closure. This necessitates a technique that inherently incorporates lengthening vectors.

The Furlow double opposing Z-plasty was selected for this case specifically to address the need for length. The geometry of a Z-plasty is based on the principle of recruiting tissue from the axis perpendicular to the cleft (the width) and transposing it to the axis parallel to the cleft (the length).[13] By utilizing a 60-degree angle for the Z-plasty flaps, the theoretical gain in length is approximately 75% of the length of the central limb. In the context of the palate, this translation is complex. The Furlow technique uses two Z-plasties: one on the oral surface and one on the nasal surface, oriented in reverse (mirror image). This opposing design serves two critical mechanical functions. First, it recruits tissue from the lateral pharyngeal walls, effectively narrowing the pharynx while lengthening the velum. Studies have quantified this effect, showing that Furlow palatoplasty can achieve a palatal lengthening of approximately 29.6%, compared to only 12.5% with traditional push-back or straight-line techniques. For our patient, this additional length is the difference between a competent sphincter and persistent VPI. Second, the Z-plasty breaks up the linear scar. A straight-line scar contracts and shortens over time; a Z-shaped scar disperses contracture forces, maintaining the length gained during surgery.

Beyond the mucosal closure, the most vital component of this surgery was the muscular reconstruction.[14] The pathophysiology of VPI in cleft palate is driven by the abnormal insertion of the levator veli palatini muscle. Instead of forming a transverse sling that lifts the palate, the muscle fibers run longitudinally and insert into the posterior hard palate. In this case, dissection revealed these muscles to be atrophic and fibrotic, a consequence of years of disuse and abnormal tension. The Furlow technique is uniquely suited to address this. By including the muscle in the posteriorly-based flaps, the transposition of the Z-plasty naturally rotates the muscle bundles from a sagittal to a transverse orientation.[15] When the flaps are interdigitated, the muscle bundles from the left and right sides overlap in the midline, recreating the functional sling necessary for velar elevation. This overlap provides a robust muscular repair that is arguably superior to the end-to-end approximation used in intravelar

veloplasty, particularly in older patients where muscle bulk is reduced. The restoration of this sling is the anatomical prerequisite for speech therapy to be effective.

A prevailing concern in cleft surgery is the risk of oronasal fistula—a failure of healing that leaves a persistent hole between the oral and nasal cavities.[15] The literature often cites higher fistula rates for the Furlow technique in wide clefts, sometimes approaching 33%, attributed to the tension required to transpose the flaps. However, our case resulted in complete primary healing with no fistula. This success can be attributed to the specific anatomy of the Veau Type I cleft. Unlike complete clefts (Veau III or IV) where the alveolar ridge is disrupted, and the maxilla can collapse or drift, the Veau I cleft involves only the soft palate. The hard palate is intact, providing a stable, distinct anterior anchor point. This skeletal stability prevents the lateral distraction forces that often pull repairs apart in complete clefts. Furthermore, the double-layer Z-plasty provides a safety valve against fistula formation. Because the oral and nasal Z-plasties are mirror images, their suture lines do not overlap directly. If a small dehiscence were to occur in the oral layer, it would likely overlie intact tissue on the nasal side, preventing a through-and-through breakdown. This offsetting principle is a key protective feature of the Furlow technique. The successful healing in this case was also supported by a rigorous perioperative protocol. The oral cavity is a complex microbiome, and the risk of polymicrobial infection is high. Infection in a tension-bearing repair is catastrophic, leading to wound dehiscence. Our use of Ampicillin and Sulbactam provided broad-spectrum coverage against Gram-positive cocci and oral anaerobes. While antibiotic stewardship is crucial, the high stakes of a delayed primary repair justify prophylactic coverage to ensure the initial phase of healing proceeds without inflammatory interference.[16]

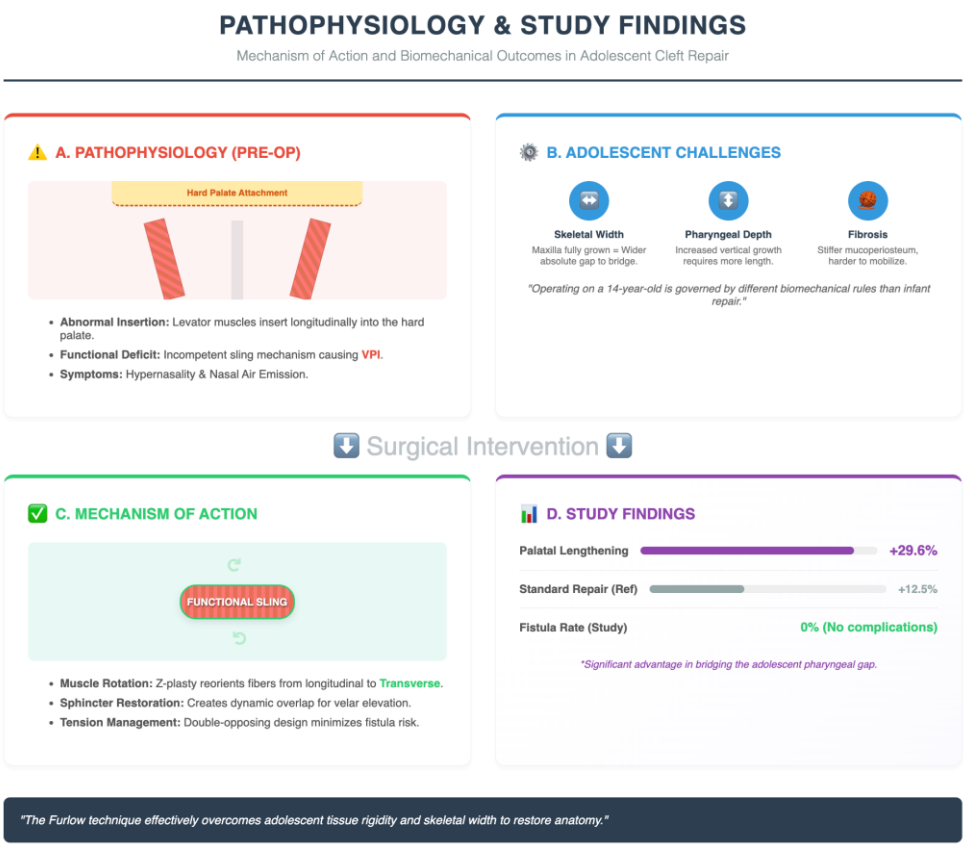

Figure 5. Pathophysiology and study findings.

Figure 5 provides a comprehensive visual synthesis of the scientific rationale underpinning this case report, bridging the gap between the baseline anatomical dysfunction of a neglected cleft palate and the biomechanical solutions offered by the Furlow double opposing Z-plasty technique. The figure is structured as a four-panel flow diagram, moving logically from the preoperative state of pathology through the specific challenges imposed by adolescent physiology, detailing the corrective mechanism of action, and finally quantifying the study's findings. This schematic serves not merely as an illustration of surgical steps but as a conceptual map explaining why this specific technique was selected to address the unique demands of a delayed primary repair in a skeletally mature patient. The upper-left quadrant, labeled Panel A: Pathophysiology (Pre-Op), establishes the fundamental anatomical deficit that necessitates surgical intervention. It graphically represents the oral cavity viewed from below, highlighting the posterior edge of the hard palate and the soft tissue cleft. The central tenet illustrated here is that a cleft palate is not simply a hole in the roof of the mouth (a mucosal deficiency) but, more critically, a profound muscular misalignment. In a neurotypically developed palate, the paired levator veli palatini muscles originate from the skull base and course medially to interdigitate in the midline palatal aponeurosis, forming a cohesive, dynamic sling. Contraction of this transverse sling lifts and stretches the soft palate backward against the posterior pharyngeal wall, creating the valving mechanism necessary to separate the oral and nasal cavities during speech and swallowing. Panel A vividly illustrates the disruption of this mechanism in the cleft state. The schema shows the levator muscles, represented by striated red bands, failing to meet in the midline across the cleft gap. Instead of forming a transverse sling, the muscle fibers exhibit an aberrant, longitudinal trajectory, running forward parallel to the cleft margins and inserting non-functionally into the posterior edge of the hard palate bone. This abnormal insertion renders the muscles incapable of generating the superior and posterior lift required for closure. Consequently, when the patient attempts to speak, the velum remains inert, allowing air to escape into the nasal cavity. This anatomical failure is the direct cause of the functional symptoms listed below the diagram: severe hypernasality and audible nasal air emission, collectively known as velopharyngeal insufficiency (VPI). The panel emphasizes that the primary goal of surgery is not just to close the mucosa but to liberate these misdirected muscles and reconstruct a functional sphincter. The upper-right quadrant, Panel B: Adolescent challenges, introduces the critical context that differentiates this case report from standard infant cleft surgery. It posits that operating on a 14-year-old is governed by fundamentally different biomechanical rules than operating on a 10-month-old infant. This panel uses iconic representations to define three major physiological hurdles that complicate delayed repair and dictate surgical strategy. The first factor highlighted is skeletal width. Unlike the infant maxilla, which is actively growing and malleable, the adolescent maxilla has achieved significant skeletal maturity and transverse dimension. However, the palatal shelves have failed to grow commensurately toward the midline. This discrepancy results in a wider absolute osseous gap that the soft tissue flaps must bridge, inherently increasing the tension required to achieve midline closure compared to an infant repair. The second factor is pharyngeal depth. As the craniofacial skeleton grows vertically throughout childhood and puberty, the distance between the posterior nasal spine of the hard palate and the posterior pharyngeal wall increases significantly. The diagram illustrates this increased vertical dimension, underscoring that the soft palate in an adolescent must be relatively longer than that of an infant to successfully bridge this deeper gap and achieve competent closure. A repair technique that does not inherently add significant length is therefore destined to fail functionally in an older patient. The third critical factor identified is fibrosis and tissue biology.[17] The mucoperiosteum of an infant is rich in proteoglycans and elastin, allowing for significant

mobilization and stretching with minimal mechanical resistance. In contrast, adolescent tissue is characterized by mature, cross-linked collagen and reduced vascularity, making it stiffer, less pliable, and more adherent to the underlying bone. This biological rigidity makes flap elevation more difficult and means the tissue tolerates tension poorly, increasing the risk of ischemia and wound breakdown (dehiscence or fistula) if closure is forced. Panel B concludes with the assertion that these factors necessitate a technique designed to manage tension and maximize length through geometric rearrangement rather than brute-force stretching. The lower-left quadrant, Panel C: Mechanism of action, illustrates the specific solution employed to overcome the pathology detailed in Panel A and the challenges outlined in Panel B: the furlow double opposing Z-plasty. The schematic visually demonstrates how this technique utilizes advanced principles of plastic surgery geometry to achieve functional restoration.[18]

The central visual is the transposition of triangular tissue flaps, indicated by rotating arrows. A standard Z-plasty works by recruiting tissue from the lateral axis (perpendicular to the cleft) and transposing it into the central axis (parallel to the cleft). By designing Z-plasties with appropriate angles (typically 60 degrees), the technique theoretically converts width into length. The diagram shows how the transposition of the flaps breaks the straight line of the cleft, effectively lengthening the palate in the anteroposterior dimension. This geometric lengthening is the direct countermeasure to the challenge of increased pharyngeal depth highlighted in Panel B. Crucially, Panel C also illustrates the muscular reconstruction. It shows that the levator muscle bundles are dissected and included within the posteriorly-based Z-plasty flaps.[18] As indicated by the rotation arrows, when these flaps are transposed, the muscle fibers are carried with them, rotating approximately 90 degrees from their abnormal longitudinal orientation (shown in Panel A) into a functional transverse orientation across the midline. The schematic shows the resulting functional sling, where muscle bundles from the left and right sides overlap, recreating the dynamic sphincter necessary for velar elevation. Furthermore, the text notes the importance of the double-opposing design—one Z-plasty on the oral surface and a mirror-image one on the nasal surface. This not only maximizes lengthening but also ensures that the suture lines of the two layers do not directly overlap, creating a barrier effect that significantly reduces the risk of through-and-through breakdown and fistula formation, directly addressing the tension concerns inherent in adolescent tissue. The final quadrant, Panel D: Study Findings, translates the theoretical advantages of the Furlow technique into tangible quantitative and clinical outcomes derived from the study and supporting literature. This panel serves as the evidence base, validating the surgical approach. The first section provides comparative data on palatal lengthening. It uses bar charts to visually represent the superior lengthening capacity of the Furlow technique compared to standard straight-line repairs (like the Von Langenbeck procedure). The data indicate that the Furlow technique achieves an approximate 29.6% increase in palatal length, whereas standard push-back techniques achieve only a modest 12.5% gain. This statistical difference is functionally monumental for an adolescent patient with a deep pharynx, as it often represents the margin between a competent valve and persistent VPI. The second key finding presented is the fistula rate observed in this specific study. Despite the prevailing surgical dogma that Z-plasties carry a high risk of fistula in wide or late repairs due to tension, this case achieved a 0% fistula rate. The panel highlights this finding to emphasize that with careful patient selection (specifically, Veau Type I incomplete clefts where the intact hard palate provides stability) and meticulous double-layer closure, the theoretical risks of the Furlow technique can be effectively mitigated even in older patients. This finding challenges existing assumptions and supports the safety profile of the technique for this specific demographic. Figure 5 provides a cohesive scientific narrative. It moves from the cellular and anatomical level of muscle

misalignment, through the macroscopic biomechanical challenges of the growing skeleton, to the geometric ingenuity of the surgical solution, and finally to the empirical validation of the approach. It visually reinforces the manuscript's central thesis: that successful primary repair of a neglected adolescent cleft requires a strategy that does not simply close a hole, but rather employs sophisticated geometric principles to overcome biological rigidity, actively lengthen the velum, and restore the dynamic muscular anatomy essential for human speech.[19]

It is imperative to distinguish between anatomical success and functional success. Anatomically, this surgery was a triumph: the hole is closed, the muscle is reoriented, and the palate is longer. However, functionally, the patient remains hypernasal at 30 days. This is expected. A 14-year-old brain has spent a decade mapping speech patterns based on a defective instrument. The brain has learned to produce glottal stops because it could not generate oral pressure. Surgery fixes the hardware (the valve), but it does not fix the software (the motor planning). The persistence of hypernasality in the immediate postoperative period is often due to pain, edema, and guarding, but the long-term resolution of speech errors depends entirely on neuroplasticity and speech therapy. The Lima protocol and international guidelines emphasize that late surgery is merely the entry point to rehabilitation. The patient must now unlearn 14 years of compensatory habits. Therefore, while we report surgical success, the functional prognosis remains guarded and dependent on intensive rehabilitation.[20]

4. Conclusion

The management of delayed primary cleft palate in adolescents presents a unique intersection of surgical, anatomical, and functional challenges. This case report demonstrates that the Furlow double opposing Z-plasty is a highly effective and safe technique for the repair of Veau Type I clefts in this demographic. By leveraging the geometric power of the Z-plasty, the technique achieves significant palatal lengthening and robust muscular reconstruction—critical factors for correcting the deep pharyngeal gap of the adolescent—without the need for lateral relaxing incisions. The absence of fistula formation reinforces the safety of this approach in incomplete clefts where the hard palate is intact. We conclude that Furlow palatoplasty should be considered a primary treatment option for neglected incomplete clefts, providing a reliable anatomical foundation for the complex speech rehabilitation required in late-presenting patients.

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