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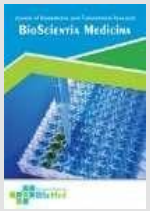

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From Nasal Vestibulitis to Maxillofacial Abscess: Reconstruction of Extensive MRSA-Induced Defects in a Diabetic Patient

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A B S T R A C T

Background: Nasal vestibulitis is frequently regarded as a benign localized infection. However, in immunocompromised hosts, specifically those with uncontrolled diabetes mellitus, it can rapidly escalate into a life-threatening maxillofacial abscess involving the danger triangle of the face. The synergistic destructive potential of *Methicillin-resistant Staphylococcus aureus* (MRSA) and hyperglycemia poses a formidable challenge for reconstructive surgery due to extensive tissue necrosis and compromised microvasculature. This study evaluates the efficacy of a dual-flap approach—combining Rotation and V-Y Advancement flaps—for restoring extensive midfacial defects. **Case presentation:** A 51-year-old male with uncontrolled Type 2 Diabetes presented with a massive, ruptured maxillofacial abscess originating from neglected nasal vestibulitis exacerbated by rhinotillexomania. The infection resulted in extensive necrosis spanning the nasal dorsum, infraorbital regions, and forehead. Microbiological analysis confirmed MRSA. Laboratory markers indicated severe sepsis with leukocytosis of 34,840 /mm^3 and hyperglycemia of 328 mg/dL. Following acute stabilization and surgical debridement, the patient sustained a complex soft-tissue defect crossing multiple aesthetic subunits. A staged reconstruction was performed three weeks post-debridement. A V-Y advancement flap was utilized for the infraorbital and medial cheek defects to minimize ectropion risk, while a rotation flap was designed for the glabella and nasal dorsum to recruit forehead tissue. **Conclusion:** The combination of V-Y advancement and rotation flaps provides a robust, anatomically adaptable, and aesthetically superior solution for complex midfacial defects where skin laxity is compromised. This approach allows for tension-free closure in the aesthetic subunits of the face, even in patients with compromised perfusion due to diabetes. Early recognition of MRSA in diabetic vestibulitis is critical to preventing catastrophic tissue loss.

1. Introduction

The human midface constitutes a region of profound anatomical intricacy and aesthetic significance, representing the intersection of critical functional orifices—the nasal and oral cavities—with the primary centers of non-verbal communication and identity. Structurally, the midfacial region is a mosaic of distinct aesthetic subunits, including the nasal dorsum, the infraorbital rim, the malar eminence, and the upper lip, all of which must function in concert to maintain facial harmony.[1] However, beyond its cosmetic importance, this region harbors a complex and potentially perilous vascular architecture. Infections in this area, particularly those originating in the nasal vestibule, are frequently dismissed as benign, self-limiting conditions. Yet, the anatomical zone colloquially known as the danger triangle of the face—defined by the apex at the glabella and the base at the corners of the mouth—possesses a unique venous drainage system that predisposes patients to catastrophic intracranial complications.[2]

The venous system of the midface, primarily comprising the facial, angular, and ophthalmic veins, is notably valveless. In a healthy physiological state, gravity and hydrostatic pressure facilitate drainage.[3] However, in the presence of fulminant infection and inflammation, this valveless architecture permits the bidirectional flow of blood. Consequently, septic thrombi originating from a superficial facial abscess can propagate in a retrograde fashion, bypassing the systemic circulation to enter the cavernous sinus directly. This anatomical vulnerability facilitates the spread of pathogens from the skin surface to the central nervous system, presenting a tangible risk of life-threatening sequelae such as cavernous sinus thrombosis, meningitis, and cerebral abscess.[4] Therefore, what begins as a superficial dermatological infection can rapidly escalate into a medical emergency requiring aggressive neuro-surgical and infectious disease intervention.

The etiology of such severe facial infections often traces back to nasal vestibulitis, a localized infection of the hair-bearing skin within the nasal vestibule.[5] The nasal vestibule, lined with squamous epithelium and vibrissae (protective hairs), serves as the first line of defense for the respiratory tract. However, it is also a reservoir for *Staphylococcus aureus*, which colonizes the anterior nares in approximately 20–30% of the healthy population. Under normal circumstances, the epithelial barrier prevents invasion. Yet, the integrity of this barrier is easily compromised by minor trauma. Habits such as rhinotillexomania (nose picking), hair plucking, or excessive nose blowing create micro-abrasions that serve as portals of entry for commensal bacteria. Environmental factors, such as low humidity, and iatrogenic causes, like the use of nasal splints, further predispose the epithelium to excoriation and subsequent infection. While typically manifesting as a minor furuncle or crusting, neglected vestibulitis can breach the dermis, allowing bacteria to invade the extensive subcutaneous network of the face.

The progression from simple nasal vestibulitis to extensive maxillofacial necrosis is not solely a function of anatomical vulnerability but is a distinct clinical entity driven by a dysregulated host-pathogen interaction. This progression is rarely seen in immunocompetent individuals but becomes a formidable threat in the presence of comorbidities, most notably diabetes mellitus (DM).[6] Diabetes creates a physiological environment that is exquisitely conducive to rapid bacterial proliferation and tissue destruction. The pathophysiology of this susceptibility is multifactorial. Hyperglycemia induces a state of immunoparalysis, characterized by defects in granulocyte function; specifically, neutrophil chemotaxis, phagocytosis, and intracellular bactericidal activity are significantly impaired in the presence of elevated blood glucose. Furthermore, chronic hyperglycemia promotes the formation of advanced glycation end-products, which further compromise the vascular endothelium. Diabetic microangiopathy, a hallmark of the disease, results in poor tissue perfusion and hypoxia, which not only hinders the immune response but also impedes the delivery of systemic antibiotics to the infected nidus. Consequently, soft tissue infections in diabetic patients are prone to rapid expansion, often progressing to necrotizing cellulitis or deep space abscesses within 48 hours of onset.

Compounding the risk posed by host immunocompromise is the evolving microbiology of head and neck infections.[7] The emergence of community-acquired *Methicillin-resistant Staphylococcus aureus* (MRSA) has fundamentally altered the clinical landscape. Historically confined to healthcare settings, MRSA has become a predominant pathogen in community-onset abscesses. The virulence of MRSA extends beyond its resistance to beta-lactam antibiotics. Many community-acquired strains produce specific exotoxins, such as the Panton-Valentine leukocidin (PVL), which targets and lyses leukocytes. The release of these cytotoxins accelerates tissue necrosis and promotes the formation of large, destructive abscesses that are

disproportionate to the initial lesion size. In the midface, this necrotizing process can obliterate skin, subcutaneous fat, and muscle, leaving behind profound defects that expose the underlying cartilaginous and bony skeleton. The synergistic destructive potential of MRSA virulence factors and diabetic immunopathy creates a perfect storm, necessitating urgent surgical debridement that often results in significant soft tissue loss.[8]

The reconstruction of the resultant defects presents a significant surgical challenge that requires a sophisticated understanding of facial biomechanics and aesthetics. The primary objective of reconstruction extends beyond simple wound coverage; it aims to restore the aesthetic units of the face to their pre-morbid state. The Principle of Subunits, a cornerstone of facial plastic surgery, dictates that the face is divided into distinct topographical regions—lips, cheeks, nose, eyelids, and forehead—based on skin texture, color, thickness, and solar orientation. A defect that crosses the boundaries of these subunits, such as one involving the nasal dorsum and the infraorbital cheek, cannot be treated as a single entity without compromising facial symmetry.

Conventional reconstructive options, such as full-thickness skin grafts, often prove inadequate for deep midfacial defects. While grafts offer a simple solution for superficial coverage, they frequently result in poor color matches, contour depression due to the lack of subcutaneous bulk, and a patch-like aesthetic outcome that disrupts the visual continuity of the face. Furthermore, in the infraorbital region, the secondary contraction of a skin graft poses a severe functional risk: the vertical pull of the contracting scar can drag the lower eyelid inferiorly, causing ectropion, epiphora, and exposure keratitis. Therefore, the recruitment of adjacent tissue via local flaps is generally preferred.[9]

Local flaps, such as rotation and advancement flaps, offer superior texture and color matches because they utilize skin from the same aesthetic region or an immediately adjacent one. However, the design of these flaps in the midface requires meticulous planning to avoid the distortion of mobile free margins, such as the nasal ala and the eyelid. The challenge lies in selecting a donor site that possesses sufficient laxity to close the defect without creating excessive tension on these critical structures. For defects bridging the nose and the cheek, a single flap is often insufficient to address the disparate requirements of the distinct subunits. The nasal dorsum requires thin, non-hair-bearing skin that conforms to the rigid skeletal framework, while the cheek requires thicker, more mobile tissue that can blend into the nasolabial fold.

This dichotomy necessitates a combinatorial approach. The Rotation Flap is a pivotal flap ideal for triangular defects, particularly those on the nasal dorsum or glabella, as its curvilinear design allows for the recruitment of forehead skin while hiding the incision along the brow line or nasofacial sulcus.[10] Conversely, the V-Y Advancement Flap operates on a different biomechanical principle. Instead of rotating, the V-Y flap advances by recoil or pushing tissue into the defect. This mechanism is particularly advantageous in the infraorbital region because it avoids the vertical traction associated with other closure methods, thereby protecting the lower eyelid position. By combining these two techniques, the surgeon can address multi-unit defects by recruiting tissue from different vectors, respecting the boundaries of the aesthetic subunits, and minimizing tension on free margins.

In this context, we present a case of a massive midfacial defect caused by an MRSA-induced abscess in a diabetic patient, originating from neglected nasal vestibulitis. This case serves as a paradigm for the catastrophic potential of common nasal infections in immunocompromised hosts and illustrates the complexity of subsequent reconstruction. Therefore, this study aims to demonstrate the utility and geometric rationale of a dual-flap technique—specifically the simultaneous use of Rotation and V-Y Advancement flaps—to reconstruct extensive, multi-unit facial defects. This report highlights the distinct

pathophysiology of rapid MRSA progression in the diabetic danger triangle and validates a reproducible, anatomically based surgical algorithm for high-risk midfacial reconstruction, offering a novel perspective on salvaging aesthetic outcomes in the face of severe infectious necrosis.

2. Case Presentation

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images. The patient was fully briefed on the nature of the publication, the anonymity of the data presentation, and the educational purpose of the report.

A 51-year-old male was referred to the Emergency Department of Dr. M. Djamil General Hospital Padang, a tertiary academic medical center, presenting with a chief complaint of a progressively enlarging, painful facial mass. The patient reported that the onset of the disease trajectory began approximately 15 days prior to admission. The initial lesion manifested as a solitary, circumscribed furuncle (boil) located within the nasal vestibule. The patient admitted to a history of rhinotillexomania (habitual nose picking) and frequent digital manipulation of acneiform lesions, specifically noting that he had squeezed the vestibular furuncle in an attempt to drain it.

Following this mechanical trauma to the nasal triangle, the infection failed to resolve and instead exhibited rapid local progression. Ten days prior to his hospital admission, the patient developed significant nasal airway obstruction accompanied by the discharge of purulent material. The inflammatory process, initially confined to the vestibule, transgressed anatomical boundaries, extending superiorly to the nasal dorsum and laterally to involve the bilateral infraorbital regions, cheeks, and the frontal region. The patient reported a sensation of heaviness in the cheek and forehead but denied visual disturbances such as diplopia or blurred vision, suggesting the orbit itself was spared despite the proximity of the inflammation.

The patient's medical history was significant for several risk factors contributing to poor wound healing and immune dysregulation. He was a chronic smoker with a 30-year history, consuming approximately one pack of cigarettes per day since the age of 20. Most critically, the patient had a known diagnosis of Type 2 Diabetes Mellitus; however, this condition was managed poorly, with the patient admitting to non-compliance with both pharmacological therapy and dietary restrictions. He denied any history of alcohol consumption or known malignancies in his immediate family.

Upon physical examination in the Emergency Department, the patient appeared moderately ill. His sensorium was intact, with a Glasgow Coma Scale (GCS) of 15, indicating he was fully alert and cooperative. Vital signs indicated a hyperdynamic and hypertensive state, likely secondary to pain and physiological stress, with a blood pressure of 169/80 mmHg and a heart rate of 84 beats per minute. He was afebrile at the time of examination (36.5°C) with a respiratory rate of 18 breaths per minute.

Examination of the face revealed a massive, diffuse, and fluctuant swelling indicative of a large abscess (Figure 1). The lesion involved multiple aesthetic subunits, spanning the nasal dorsum, the bilateral infraorbital and malar regions, and extending superiorly into the forehead. The skin overlying the abscess was erythematous and tense, with multiple areas of focal necrosis and crusting. Spontaneous drainage of purulent exudate was noted from the nasal region. The clinical appearance was consistent with the Rudolph Sign, a hallmark of severe nasal vestibulitis characterized by intense erythema and edema of the nasal tip and dorsum.

Anterior rhinoscopy demonstrated severe pathology within the nasal cavity. The nasal vestibule was markedly edematous, hyperemic, and obstructed by crusts and purulent discharge. Clotting was observed within the nasal passage. The inflammation had narrowed the internal nasal valve, significantly compromising the airway.

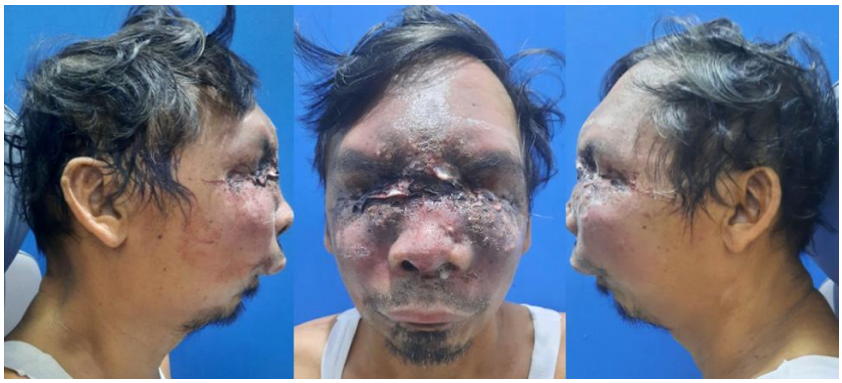

Figure 1. Clinical appearance of the patient on admission.

While the inferior turbinates appeared eutrophic, visualization of the middle meatus was obscured by the extensive edema, preventing a complete assessment of the osteomeatal complex. Posterior rhinoscopy and oropharyngeal examination were within normal limits. To delineate the deep extent of the infection, a Computed Tomography (CT) scan of the paranasal sinuses was performed. The imaging confirmed a large, multiloculated abscess involving the maxillofacial soft tissues, extending from the nasal vestibule to the frontal region. Crucially, the scan ruled out orbital subperiosteal abscess formation and confirmed that the infection had not breached the posterior table of the frontal sinus or the cribriform plate, thereby excluding intracranial extension.

The laboratory profile obtained at admission painted a picture of severe sepsis complicated by metabolic dysregulation. A profound leukocytosis was present, with a White Blood Cell (WBC) count of 34,840 /mm^3, markedly higher than the upper limit of normal, indicating a massive systemic immune response to the bacterial load. A neutrophil predominance was noted. The patient exhibited severe hyperglycemia with a random blood glucose level of 328 mg/dL. Further evaluation of his metabolic control revealed a Glycated Hemoglobin (HbA1c) level of 11.2%, confirming a state of chronic, uncontrolled hyperglycemia that likely impaired neutrophil chemotaxis and facilitated the rapid spread of infection. Acute phase reactants were significantly elevated. C-Reactive Protein (CRP) was 145 mg/L, and Procalcitonin was 2.1 ng/mL, values that strongly correlated with the clinical diagnosis of bacterial sepsis and extensive tissue necrosis. Pus specimens collected from the nasal discharge were sent for culture and sensitivity analysis. The cultures yielded *Methicillin-resistant Staphylococcus aureus* (MRSA). Antibiotic susceptibility testing revealed that the isolate was resistant to beta-lactams but retained sensitivity to Gentamicin, Clindamycin, and Tetracycline. This microbiological finding was pivotal, necessitating a shift from standard empiric therapy to targeted anti-MRSA agents.

Upon admission to the high-dependency unit, the immediate therapeutic priority was the stabilization of the patient's systemic inflammatory response and the control of metabolic dysregulation (Table 2). Given the severity of the infection within the danger triangle of the face, a broad-spectrum empirical antibiotic regimen was instituted without delay. The patient commenced intravenous Ampicillin-Sulbactam (1.5 g administered every 6 hours) combined with Metronidazole to cover potential anaerobic co-pathogens often present in polymicrobial facial abscesses. Simultaneously, an aggressive insulin sliding scale was implemented to counteract the patient’s profound hyperglycemia (random blood glucose >300 mg/dL), as optimizing glycemic control is a critical prerequisite for effective wound healing and leukocyte function.

Table 1. Summary of clinical findings on admission.

CATEGORY	FINDING / VALUE	CLINICAL INTERPRETATION
1. PATIENT PROFILE & ANAMNESIS		
Chief Complaint	Rapidly expanding, painful facial mass (15-day duration) initiated by nasal vestibular furuncle.	*Neglected infection with traumatic exacerbation (Rhinotillexomania).*
Risk Factors	Type 2 Diabetes (Non-compliant), Chronic Smoker (30 pack-years).	*Immunocompromised state; poor microvascular perfusion.*
2. VITAL SIGNS & HEMODYNAMICS		
Blood Pressure	**169/80 mmHg**	*Hypertensive response to pain/stress.*
Other Vitals	HR: 84 bpm | RR: 18/min | Temp: 36.5°C	*Afebrile at presentation despite severe local pathology.*
3. LOCAL STATUS (HEAD & NECK)		
Facial Inspection	Fluctuant swelling: Nasal dorsum, Bilateral Infraorbital, Forehead. Erythema and necrotic crusts present.	*"Rudolph Sign" indicating severe vestibulitis with abscess extension.*
Rhinoscopy	Severe vestibular edema, purulent discharge, clotting. Internal nasal valve narrowing.	*Significant airway obstruction.*
4. LABORATORY INVESTIGATIONS		
Leukocytes (WBC)	**34,840 /mm³**	*Severe Leukocytosis (Neutrophilia).*
Glycemic Profile	Random Glucose: **328 mg/dL** HbA1c: **11.2%**	*Uncontrolled Hyperglycemia.*
Inflammatory Markers	CRP: **145 mg/L** Procalcitonin: **2.1 ng/mL**	*Systemic Bacterial Sepsis.*
5. MICROBIOLOGY & IMAGING		
Pus Culture	**MRSA** (Methicillin-Resistant *S. aureus*) Sensitive: **Gentamicin, Clindamycin**	*Multi-drug resistant pathogen requiring targeted therapy.*
CT Scan Findings	Multiloculated abscess (Nasal vestibule to frontal region).	*No orbital subperiosteal abscess or intracranial extension.*

Antihypertensive therapy comprising Candesartan (16 mg) was also initiated to manage the patient's hypertensive urgency. On the third day of hospitalization, microbiological culture results from the purulent exudate confirmed the presence of *Methicillin-resistant Staphylococcus aureus* (MRSA). This finding necessitated a prompt de-escalation of the empirical regimen to targeted therapy. The antibiotics were adjusted to Intravenous Gentamicin (80 mg every 12 hours) and Clindamycin to ensure adequate tissue penetration and bactericidal activity against the resistant strain.

By the eighth day of hospitalization, the medical optimization protocol had successfully reduced the

patient's blood glucose levels to below 200 mg/dL, rendering him a suitable candidate for surgical intervention. Under general anesthesia, the patient underwent definitive source control via incision and drainage. The surgical exploration revealed extensive tissue destruction, necessitating aggressive debridement of necrotic skin, subcutaneous adipose tissue, and the superficial musculoaponeurotic system (SMAS) involving the nasal dorsum, infraorbital regions, and forehead. The abscess cavity was evacuated of purulent collections and copiously irrigated to reduce the bacterial bioburden. Histopathological analysis of the debrided tissue specimens revealed a landscape of chronic inflammation superimposed with acute exacerbation. The microscopic architecture demonstrated dense infiltration by lymphoplasmacytic cells and neutrophils, alongside significant necrotic debris and capillary hyperemia, confirming the diagnosis of a ruptured abscess with severe localized tissue reaction. Following the procedure, the patient was managed with daily dressing changes to facilitate secondary intention healing and granulation tissue formation.

Three weeks following the initial debridement, the patient was re-evaluated for reconstruction. The wound bed appeared healthy, with robust granulation tissue and no clinical evidence of residual infection or active purulence. The resultant defect was substantial and geometrically complex, violating three distinct aesthetic subunits: the bilateral infraorbital regions, the nasal dorsum, and the paramedian forehead. The reconstructive challenge lay in the defect's location within the midfacial region, where the scarcity of mobile skin and the proximity to the lower eyelid margin posed a high risk of functional distortion. A primary closure or skin graft was deemed inappropriate due to the potential for secondary contracture, which could precipitate ectropion (eversion of the lower eyelid). Consequently, a decision was made to employ a staged local flap approach to import vascularized, color-matched tissue with similar textural qualities to the lost integument.

Intraoperatively, the patient was positioned supine and induced under general anesthesia. The facial field was prepared and draped in a sterile fashion. The irregular margins of the granulation tissue were freshened to expose healthy, bleeding wound edges, ensuring a viable interface for flap inset. To facilitate hemostasis and hydro-dissection of tissue planes, the operative field was infiltrated with a solution of 2% Lidocaine with Epinephrine (1:100,000). The reconstruction of the infraorbital and medial cheek defect required a technique that would recruit tissue without exerting vertical traction on the eyelid. A V-Y advancement flap was designed adjacent to the defect, incorporating the nasolabial fold (melolabial crease). The flap was incised in a V-configuration and undermined extensively in the subcutaneous plane to release retaining ligaments and mobilize the pedicle. Unlike rotational flaps that pivot, this island flap was advanced medially into the defect via a recoil or pushing mechanism. The donor site was closed in a Y-configuration, strategically placing the vertical limb of the closure within the relaxed skin tension lines (RSTLs) to camouflage the eventual scar. This vector of movement ensured tension-free closure at the eyelid-cheek junction.

To address the defect spanning the nasal dorsum and glabellar region, a rotation flap was designed utilizing the forehead as the donor site. The forehead skin provides an ideal match for the nose in terms of thickness, color, and sebaceous quality. A large, curvilinear incision was marked extending from the superior margin of the defect superiorly into the paramedian forehead. The flap was raised and rotated inferiorly to cover the exposed nasal dorsum. To facilitate this rotation without creating a dog-ear or standing cutaneous cone at the pivot point, a Burow's triangle was excised. This geometric modification allowed the flap to lie flat against the convex contour of the nasal bridge without tension or vascular compromise. Prior to final closure, a 16 Fr Foley catheter was temporarily inserted into the nasal cavity. The balloon was inflated to stent the internal structures and provide counter-pressure, ensuring

the patency of the nasal airway during the layering of the flaps. The flaps were inset using a layered closure technique. The subcutaneous and deep dermal layers were approximated with absorbable Vicryl 4.0 sutures to obliterate dead space and reduce tension on the skin edges. The epidermis was then closed with non-absorbable Prolene 5.0 sutures in a continuous locking fashion to ensure precise alignment of the wound edges and optimal aesthetic scarring.

The post-operative recovery was uncomplicated. Both the rotation and advancement flaps exhibited immediate viability. On the first post-operative day, mild edema was observed, which is expected following extensive facial dissection; however, capillary refill was brisk (<2 seconds) in both flaps, indicating robust arterial perfusion and adequate venous drainage. The Foley catheter was removed, and the patient reported subjective improvement in nasal airflow. By the seventh post-operative day, the wounds were clean, dry, and free of erythema or discharge. All skin sutures were removed, revealing well-apposed wound edges. The patient was discharged with prophylactic oral antibiotics (Clindamycin) and analgesics. At the three-week follow-up appointment, the aesthetic and functional outcomes were evaluated. The flaps had integrated well with the surrounding tissues, and the scars were beginning to settle within the natural skin creases. Importantly, there was no evidence of lower eyelid retraction (ectropion), nasal valve collapse, or recurrence of the MRSA infection. The patient expressed high satisfaction with the cosmetic restoration and restored nasal patency. Education regarding strict glycemic control and facial hygiene was reinforced to prevent recurrence.

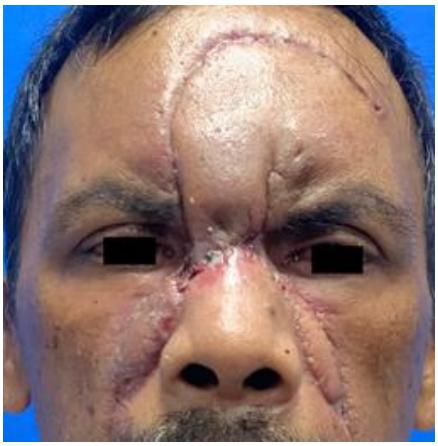

Figure 2. Clinical appearance at 3 weeks follow-up post-operative.

3. Discussion

This case provides a stark illustration of the rapid and potentially catastrophic progression of *Staphylococcus aureus* from a benign commensal organism to a highly invasive and destructive pathogen. It is well-established that approximately 30% of the healthy adult population carries *S. aureus* within the moist, squamous epithelium of the nasal vestibule without clinical consequence.[11] However, in this patient, the transition from colonization to fulminant invasive disease was not random; rather, it was driven by a distinct double-hit phenomenon comprising mechanical trauma and a compromised host immune system. The initiating event—rhinotillexomania, or chronic nose picking—created micro-abrasions in the vestibular lining, breaching the primary epithelial defense. In an immunocompetent host, such a breach would trigger a localized inflammatory response sufficient to contain the bacteria.

Table 2. Diagnosis, treatment, follow-up, and outcome.

CLINICAL PHASE	INTERVENTION / FINDINGS	CLINICAL RATIONALE / NOTES
1. DIAGNOSIS & ETIOLOGY		
Primary Diagnosis	Maxillofacial Abscess (Nasolabial, Infraorbital, Frontal) secondary to Neglected Nasal Vestibulitis.	*Triggered by rhinotillexomania (nose picking).*
Pathogen	**MRSA** (Methicillin-Resistant *Staphylococcus aureus*).	*Resistant to Beta-lactams.*
2. ACUTE MEDICAL MANAGEMENT (STABILIZATION)		
Antibiotic Therapy	1. Empirical: **Ampicillin-Sulbactam** + **Metronidazole**. 2. Targeted (Post-Culture): **Gentamicin** + **Clindamycin**.	*De-escalation to anti-MRSA agents on Day 3.*
Metabolic Control	Aggressive **Insulin Sliding Scale** & **Candesartan**.	*Target Glucose < 200 mg/dL for surgery.*
3. SURGICAL MANAGEMENT (STAGED APPROACH)		
Stage 1: Source Control (Day 8)	**Incision & Drainage (I&D)** + Radical Debridement.	*Removal of necrotic skin, subcutis, and SMAS.*
Stage 2: Reconstruction (Week 3 Post-Debridement)	**Defect Repair:** 1. Infraorbital/Cheek: **V-Y Advancement Flap**. 2. Nasal Dorsum/Glabella: **Rotation Flap** (Forehead Donor).	*Strategy: Prevent ectropion (V-Y) & Match skin texture (Rotation).*
4. FOLLOW-UP & OUTCOME		
Post-Op Recovery	• **Day 1:** Mild edema, Capillary refill < 2s. • **Day 7:** Suture removal, wounds clean/dry.	*Viable flaps, no venous congestion.*
Final Outcome (Week 3)	✔ **Aesthetic Satisfaction**: Scars settling in skin creases. ✔ **Functional Success**: Patent airway, No ectropion. ✔ **No Recurrence** of infection.	*Successful salvage of Danger Triangle defects.*

However, this patient's uncontrolled Type 2 Diabetes Mellitus created a physiological microenvironment uniquely favorable to bacterial proliferation and tissue necrosis. Chronic hyperglycemia induces the non-enzymatic glycosylation of tissue proteins and disrupts the cellular components of the immune system. Specifically, it impairs the chemotaxis, phagocytosis, and intracellular bactericidal activity of neutrophils, leading to a state of functional immunoparalysis. This failure of the innate immune response facilitates unchecked bacterial overgrowth. Furthermore, diabetic microangiopathy—characterized by thickening of the capillary basement membrane—impedes adequate tissue perfusion and reduces the delivery of systemic antibiotics to the infected nidus, effectively sequestering the pathogen.[12]

The pathogen itself, Community-Acquired Methicillin-Resistant *Staphylococcus aureus* (CA-MRSA), likely played a pivotal role in the extent of the necrosis. Unlike hospital-acquired strains, CA-MRSA frequently carries the gene for the Panton-Valentine 1143eucocidin (PVL) cytotoxin. This toxin creates pores in the membranes of host leukocytes, leading to

their destruction and the release of inflammatory mediators that accelerate tissue necrosis.[13] The extensive soft tissue destruction observed in this case is consistent with MRSA-induced necrotizing fasciitis, a pathology distinct from and far more aggressive than simple cellulitis.

The anatomical location of this infection—the danger triangle of the face—adds a layer of critical urgency. This region, extending from the corners of the mouth to the glabella, possesses a unique venous drainage system. The facial vein communicates with the cavernous sinus via the ophthalmic veins and the pterygoid plexus. Crucially, these veins are valveless, allowing for bidirectional flow based on pressure gradients. In the setting of a high-pressure abscess, retrograde flow can carry septic thrombi directly into the intracranial cavity. This anatomical vulnerability places the patient at high risk for cavernous sinus thrombosis, meningitis, and cerebral abscess, complications that carry significant mortality rates.[14]

The reconstruction of the resultant defect was strictly dictated by the principle of subunits as originally described by Burget and Menick. This principle posits that the human eye perceives the face not as a continuous surface, but as a mosaic of distinct topographic units (nasal, orbital, oral, cheek) defined by ridges of reflected light and shadows.[15] A defect that crosses these boundaries—such as the one in this case, which violated the cheek, nose, and forehead—cannot be treated as a single entity without obliterating these visual landmarks and creating a patch-like deformity. Therefore, independent reconstruction of each subunit is required to restore facial harmony.

For the infraorbital and medial cheek defect, we utilized the V-Y advancement technique (Figure 3). The biomechanics of this flap are distinct from rotational flaps; the V-Y flap moves by recoil or pushing tissue into the defect rather than pulling it. This distinction is critical in the infraorbital region. The lower eyelid is a mobile, free margin that is highly susceptible to vertical traction. Any reconstructive technique that relies on superiorly directed tension vectors runs a high risk of dragging the lower eyelid inferiorly, resulting in ectropion (eyelid eversion) and lagophthalmos (inability to close the eye). By designing the V-Y flap along the melolabial crease and advancing the cheek tissue medially, we capitalized on the natural laxity of the aging face in the horizontal vector. The donor site closure, which forms the vertical limb of the Y, was strategically placed within the relaxed skin tension lines (RSTLs) of the nasolabial fold, effectively camouflaging the scar. This technique served a dual purpose: it recruited robust, color-matched cheek skin to cover the defect while simultaneously providing a superiorly directed support vector to the lower eyelid, thereby actively preventing ectropion.[16]

The nasal dorsum presents a different set of requirements. The skin here is thicker, more sebaceous, and relatively fixed to the underlying cartilaginous framework. The forehead skin is the ideal donor site for nasal reconstruction due to its similarity in texture, thickness, and solar exposure.[17] We selected a rotation flap over a transposition flap (such as a bilobed flap) or a skin graft. A rotation flap utilizes a curvilinear design that allows for the redistribution of tension over a much longer arc. This distribution of tension is crucial for preventing the trapdoor deformity, a common complication where a circular scar contracts and causes the flap tissue to bulge. Furthermore, the rotation flap is designed with a broad pedicle base. In a diabetic patient with potential microvascular compromise, the reliability of the vascular supply is paramount. A broad-based random pattern flap ensures a robust perfusion pressure from the subdermal plexus, which is generally safer than relying on a narrow pedicle or a specific axial vessel that might be affected by atherosclerotic calcification. This choice prioritized flap survival and reliable healing in a host with known compromised perfusion.[18]

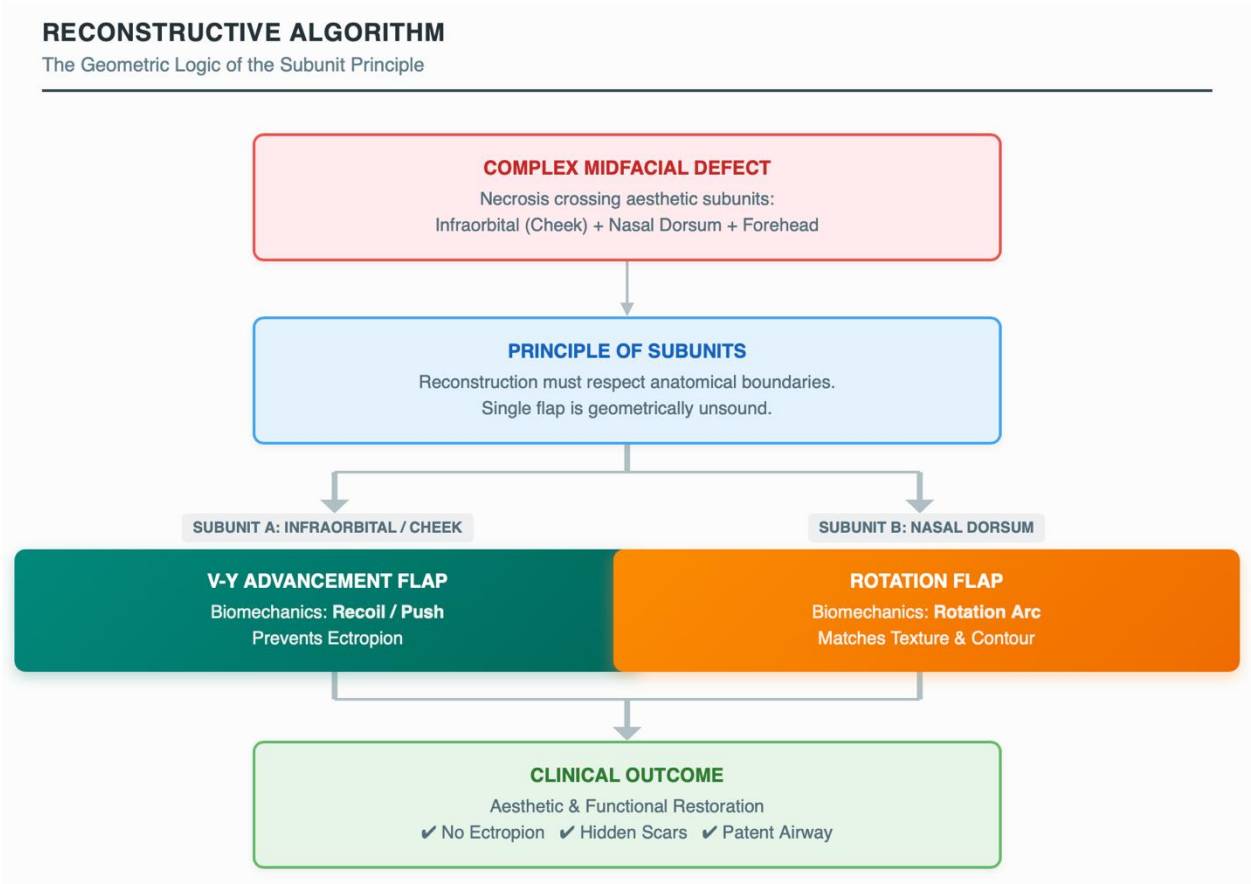

Figure 3. Reconstructive algorithm.

The decision-making process involved a careful rejection of alternative reconstructive options based on the specific constraints of the defect and the patient's physiology. Full-thickness skin grafts (FTSG) were considered but deemed inappropriate. Firstly, the depth of the debridement exposed deep subcutaneous structures and cartilage, which provide a poorly vascularized recipient bed for graft uptake, increasing the risk of graft failure. Secondly, all skin grafts undergo secondary contraction during the healing phase. In the infraorbital region, this contraction would almost certainly exert a vertical pull on the lower eyelid, leading to cicatricial ectropion. Thirdly, from an aesthetic standpoint, skin grafts rarely match the color or texture of the surrounding facial skin, often appearing as a depressed, hyperpigmented patch that is clinically inferior to the contour restoration provided by a flap.[19] A free flap, such as a radial forearm free flap, would have provided ample tissue but was considered excessive for a defect of this magnitude. Free flaps introduce significant donor site morbidity and require prolonged operative times under general anesthesia. Given the patient's ASA II status with uncontrolled diabetes and hypertension, minimizing the duration of anesthesia and surgical stress was a priority. The local flap combination allowed for a like-with-like reconstruction with significantly lower morbidity and a faster recovery trajectory.

This case highlights a novel and successful application of simultaneous local flaps in a high-risk infectious field. Historically, reconstructive surgeons have been hesitant to perform extensive flap surgery in the setting of recent MRSA infection due to the fear

of bacterial colonization leading to wound dehiscence or flap necrosis. Our results challenge this dogma, suggesting that complex local flaps can be safely employed even after severe infections, provided a strict protocol is followed. The staged approach was the linchpin of this successful outcome. By delaying the reconstruction for three weeks following the initial debridement, we allowed for the clearance of the bacterial load, the resolution of acute edema, and the formation of a healthy granulation bed. This interval also provided a window for medical optimization, specifically the stabilization of blood glucose levels. Performing reconstruction on a patient with active sepsis and hyperglycemia would have likely resulted in failure. This case validates that with adequate debridement, targeted antibiotic therapy based on culture sensitivity, and strict metabolic control, the infectious barrier to reconstruction can be overcome.[20]

4. Conclusion

The management of extensive maxillofacial abscesses in diabetic patients requires a paradigm shift from the traditional drain and manage approach to a more aggressive strategy of radical debridement followed by sophisticated, physiologically sound reconstruction. This case validates that nasal vestibulitis, often dismissed as a minor ailment, can rapidly evolve into devastating midfacial necrosis when complicated by MRSA virulence and diabetic immunopathy. The primary clinical takeaway from this study is the validation of the staged reconstructive algorithm. The stabilization of the patient—both microbiologically and metabolically—prior to tissue rearrangement is non-negotiable. Once the wound bed is optimized, the use of combined Rotation and V-Y Advancement flaps represents a safe, reliable, and aesthetically valid method for midfacial repair. This geometric approach respects the Principle of Subunits: the V-Y flap advances tissue to protect eyelid position, while the Rotation flap recruits forehead tissue to restore nasal contour. By selecting flaps with robust vascularity and minimal tension, surgeons can achieve optimal functional and aesthetic restoration, even in compromised hosts with significant comorbidities. This case serves as a testament to the importance of interdisciplinary care, combining infectious disease management, internal medicine, and reconstructive surgery to salvage the danger triangle of the face.

5. References

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