REVIEW ARTICLE

Sclerotherapy in Head and Neck Vascular Malformations: A Contemporary Review of Agents, Indications, and Outcomes

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Abstract

Vascular malformations of the head and neck represent a major subset of congenital vascular anomalies, with a marked predilection for the craniofacial region and frequent early-life presentation. These lesions, particularly venous and lymphatic types, exhibit progressive enlargement and may result in functional impairment, including compromised speech, mastication, swallowing, and airway patency, along with esthetic deformity. Their diffuse and infiltrative nature limits the predictability of surgical excision and increases the risk of recurrence and morbidity. Sclerotherapy has emerged as a minimally invasive modality that induces endothelial damage, thrombosis, and fibrosis, leading to a reduction in lesion volume. The aim of this review is to evaluate the role of sclerotherapy in the management of head and neck vascular malformations, focusing on epidemiology, lesion characteristics, sclerosant mechanisms, clinical outcomes, and complications. Evidence indicates that sclerotherapy is particularly effective in low-flow lesions, with improved functional and esthetic outcomes when applied with appropriate technique. In conclusion, sclerotherapy represents a reliable and effective modality in oral and maxillofacial practice, offering predictable outcomes with careful case selection and individualized treatment planning.

Keywords: Arteriovenous malformations, Bleomycin, Ethanol, Head and neck vascular malformations, Sclerotherapy, Vascular malformations.

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Introduction

Vascular anomalies of the head and neck represent heterogeneous congenital disorders arising from aberrant vascular morphogenesis, characterized by a persistent, noninvoluting course.[1] Nearly 50–60% occur within the craniofacial region, posing significant challenges in oral and maxillofacial practice.[2] Vascular malformations predominate, being present at birth and growing proportionally throughout life.[3] Venous malformations are most frequent (approximately 70–75%), with a prevalence of 1 in 5,000–10,000 and up to 40% localized to the head and neck.[4] Lymphatic malformations (LMs) demonstrate a strong predilection for this region (≈75%), typically diagnosed by 2 years of age, with an incidence of 1.2–2.8% and with 50%–60% evident at birth.[5] These lesions often involve the neck, tongue, and oral cavity, producing airway, swallowing, and speech compromise.[6] Arteriovenous malformations, though less common, represent high-flow lesions with aggressive clinical behavior, progressive enlargement, hemorrhage, and high recurrence following incomplete treatment.[4] Their chronic course, with <5% spontaneous regression, necessitates active intervention in most cases.[7] The ISSVA classification provides a standardized framework to distinguish malformations from vascular tumors and categorize lesions by flow dynamics.[1]

Low-flow malformations, including venous and lymphatic types, are most frequently encountered, presenting as compressible, nonpulsatile swellings with progressive enlargement and deformity.[6] Their natural history involves gradual progression with episodic exacerbations triggered by trauma, infection, or hormonal influences, leading to pain, bleeding, and airway compromise.[7] Historically, surgical excision was the primary modality, but its role in the head and neck is limited by incomplete resection, recurrence, and risk to adjacent neurovascular structures.[5] Minimally invasive techniques, particularly sclerotherapy, have gained prominence.[8]

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Sclerotherapy induces endothelial injury, thrombosis, and fibrosis, thereby reducing lesion volume and symptoms.[3] A wide spectrum of agents—including ethanol, bleomycin, sodium tetradecyl sulfate (STS), polidocanol, and ethanolamine oleate—has been employed, each with distinct pharmacodynamics, efficacy, and safety profiles.[6] Reported response rates range from 60% to >90%, with foam-based sclerotherapy exceeding 90% and improving intralesional distribution.[9]

Despite these advances, comparative studies have not identified a consistently superior sclerosant, with outcomes largely influenced by lesion-specific factors such as anatomical location, flow dynamics, and cystic architecture.[6] Meta-analytic data suggest that sclerotherapy, surgery, and combined approaches may yield comparable results, underscoring the importance of individualized, multidisciplinary management.[7] Combined strategies integrating surgical resection with adjunctive sclerotherapy have demonstrated improved functional and esthetic outcomes in complex cases.[10] Nevertheless, sclerotherapy is not devoid of complications, with major adverse events reported in 10%–15% of cases, including tissue necrosis, nerve injury, and airway compromise.[11] These findings

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highlight the necessity of careful patient selection and adherence to established protocols. The purpose of this review is to critically evaluate sclerotherapy in head and neck vascular malformations, exploring the pharmacological spectrum of sclerosants and their clinical application in oral and maxillofacial surgery, where minimally invasive yet effective modalities are essential for optimizing both functional and esthetic outcomes.

Materials and Methods

Study Design

This study is a narrative review designed to provide a structured and clinically relevant synthesis of existing literature on sclerotherapy for vascular malformations of the head and neck, with emphasis on comparative evaluation of sclerosants and their practical clinical applicability.

This narrative review was undertaken to provide a structured and clinically relevant synthesis of the current evidence on sclerotherapy in the management of vascular malformations of the head and neck. A narrative design was selected due to the heterogeneity in lesion characteristics, variability in treatment protocols, and predominance of noncomparative clinical studies, which limit the feasibility of quantitative meta-analysis.

A comprehensive literature search was conducted using PubMed, Scopus, and Google Scholar databases for studies published between January 2000 and December 2024. Retrieved articles were screened based on relevance to the topic, followed by detailed evaluation of full-text manuscripts that addressed sclerotherapy in head and neck vascular malformations. Studies were included if they reported clinical outcomes, complication profiles, or comparative evaluation of sclerosants in the management of vascular malformations involving the craniofacial region. Exclusion criteria comprised experimental or laboratory-based studies, articles unrelated to head and neck lesions, isolated case reports with limited clinical applicability, and studies lacking adequate methodological detail. Study selection was performed through sequential assessment of titles, abstracts, and full texts. Priority was given to studies demonstrating methodological clarity, adequate sample size, and direct clinical relevance to oral and maxillofacial surgical practice. Given the narrative nature of this review, formal risk-of-bias assessment tools were not applied. However, methodological rigor was maintained by emphasizing higher levels of evidence and by cross-evaluating consistency of findings across the included literature. This structured approach enabled a focused synthesis of available evidence, facilitating critical appraisal of sclerosant agents and their clinical applicability in the management of vascular malformations of the head and neck.

Vascular Lesions of the Head and Neck

Classification, Pathobiology, and Surgical Relevance

Vascular lesions affecting the head and neck constitute a complex spectrum of developmental anomalies arising from disordered vasculogenesis and angiogenesis, with direct implications for oral and maxillofacial surgical planning.[1] These entities are broadly categorized into vascular tumors and vascular malformations, a distinction grounded in endothelial kinetics, histopathological architecture, and clinical evolution.[2] The ISSVA framework provides a contemporary, biologically oriented classification that separates proliferative lesions, such as hemangiomas, from structural anomalies characterized by dysplastic vascular channels.[1]

Vascular malformations are further subclassified according to the dominant vascular component—capillary, venous, lymphatic, arteriovenous, or combined forms—and stratified into low-flow and high-flow lesions based on hemodynamic behavior.[5] This hemodynamic distinction is critical in maxillofacial surgery, as low-flow lesions typically demonstrate localized expansion with compressibility, whereas high-flow malformations exhibit rapid shunting with significant intraoperative hemorrhagic risk.[4]

From a biological perspective, these anomalies originate from embryological errors in vascular channel differentiation, leading to structurally abnormal yet mature endothelial-lined spaces that persist throughout life.[3] Unlike vascular tumors, which undergo proliferative and involutional phases, malformations exhibit continuous growth proportional to somatic development, often exacerbated by hormonal or inflammatory stimuli.[7] Molecular insights have identified dysregulation in signaling pathways governing endothelial stability and vascular remodeling, further explaining the persistent and progressive nature of these lesions.[1]

Clinically, this classification is not merely descriptive but directly influences operative decision-making, choice of intervention, and risk stratification in the maxillofacial region, where vital neurovascular structures and airway considerations are paramount.[8]

Epidemiology, Anatomical Predilection, and Disease Burden

Vascular lesions demonstrate a marked epidemiological concentration within the head and neck, accounting for approximately 50–60% of all vascular anomalies, reflecting the complexity of craniofacial vascular development.[2] Venous malformations represent the most prevalent subtype, constituting nearly three-quarters of cases and frequently involving intraoral and perioral structures such as the tongue, buccal mucosa, lips, and submandibular regions. The estimated prevalence ranges from 1 in 5,000 to 1 in 10,000 individuals, with a significant proportion identified during childhood or early adolescence.[4]

Lymphatic malformations exhibit a similarly high craniofacial distribution, with approximately 75% localized to the head and neck and up to 90% diagnosed within the first two years of life.[5] These lesions commonly involve cervical spaces, floor of the mouth, tongue, and parotid region, often extending across anatomical compartments due to their infiltrative nature.[12] The reported incidence ranges between 1.2% and 2.8%, with a substantial proportion presenting at birth and progressing during early childhood.[5]

Arteriovenous malformations, although less frequent, represent a clinically aggressive subgroup due to their high-flow characteristics and capacity for rapid expansion.[4] These lesions are associated with significant morbidity, including recurrent hemorrhage, tissue destruction, and high recurrence rates following incomplete treatment.

The disease burden associated with vascular malformations is considerable, given their chronic progression and minimal likelihood of spontaneous regression, which is reported in less than 5% of cases.[7] Functional impairment is particularly pronounced in maxillofacial involvement, where lesions may compromise airway patency, mastication, phonation, and deglutition.[6] In addition to physiological consequences, the esthetic disfigurement associated with facial lesions contributes significantly to psychosocial morbidity.[2]

Clinical Behavior, Maxillofacial Implications, and Treatment Challenges

The clinical presentation of vascular lesions in the head and neck is determined by lesion type, vascular dynamics, and anatomical distribution, with distinct features observed between low-flow and high-flow anomalies.[1] Low-flow malformations typically manifest as compressible, nonpulsatile masses with bluish discoloration, often demonstrating progressive enlargement and episodic thrombosis.[3] These lesions may infiltrate surrounding soft tissues, resulting in diffuse involvement of oral and cervicofacial compartments.[12]

High-flow malformations, including arteriovenous lesions, present with pulsatility, bruit, and localized warmth due to increased vascular flow, and may exhibit rapid progression with a propensity for hemorrhage and tissue destruction.[4] Such lesions are associated with a higher risk of intraoperative bleeding, necessitating meticulous preoperative planning and often multidisciplinary management.[8]

The natural course of vascular malformations is characterized by gradual enlargement, interspersed with episodes of acute exacerbation triggered by trauma, infection, or hormonal influences.[7] Intraoral and cervical lesions may result in airway obstruction, dysphagia, speech impairment, and recurrent bleeding, posing significant challenges in both elective and emergency settings.[6]

From an oral and maxillofacial surgical perspective, these lesions are particularly challenging due to their ill-defined margins, infiltrative growth pattern, and close proximity to critical structures, including facial nerve branches, major vessels, and airway structures. Complete surgical excision is often not feasible, especially in diffuse or microcystic lesions, and is associated with a high risk of recurrence and postoperative morbidity.[5]

Consequently, contemporary management strategies emphasize minimally invasive modalities and multimodal approaches, integrating sclerotherapy, surgical debulking, and adjunctive therapies to optimize functional and esthetic outcomes.[10] Despite advances in treatment, complication rates, including tissue necrosis, nerve injury, and airway compromise, remain clinically significant, underscoring the need for precise diagnosis and individualized treatment planning.[11]

Principles of Sclerotherapy

Sclerotherapy is a minimally invasive technique that achieves vascular collapse through chemical-mediated endothelial disruption, ultimately leading to fibrosis and regression of abnormal vascular networks.[9] In the maxillofacial region, it offers effective lesion control without extensive surgery, thereby reducing morbidity and preserving function.[13] The process begins with cytotoxic injury to endothelial cells, exposing the extracellular matrix and initiating platelet adhesion, coagulation, fibrin deposition, and thrombus formation, which subsequently undergo fibroblastic transformation to achieve permanent obliteration.[3,6,12]

The biological response varies with the agent employed: detergent-based sclerosants disrupt phospholipid membranes for rapid collapse,[14] alcohol-based agents induce protein denaturation and coagulative necrosis with broader tissue injury,[4] while others elicit controlled inflammation and gradual fibrosis, providing safer outcomes in sensitive regions.[6] Pharmacological behavior dictates precision and risk—aggressive agents achieve rapid occlusion but risk collateral damage, whereas moderated agents allow controlled sclerosis with fewer complications.[3,11] Hemodynamics further influence efficacy: slow-flow lesions favor sclerosant retention and fibrosis,[12] while high-flow lesions reduce contact time, often requiring adjunctive strategies.[4] Imaging guidance enhances accuracy, and foam formulations improve endothelial contact and distribution.[8,9] Incremental dosing protocols are preferred to balance efficacy and safety.[8,11]

Lesion architecture also impacts response, with cystic spaces showing better outcomes than diffuse, complex lesions that often require multiple sessions.[6,12] Combined approaches integrating sclerotherapy with surgery improve results by reducing lesion bulk and facilitating restoration.[10] Safe application demands anatomical expertise, meticulous planning, and strict adherence to protocols, with operator experience being critical for success.[1,11,13] This review is presented to explore the pharmacological spectrum of sclerosants and highlight their clinical relevance in oral and maxillofacial surgery, where sclerotherapy has emerged as a cornerstone for managing vascular malformations with functional and esthetic preservation.

Sclerosing Agents Used in Head and Neck Lesions

Sclerotherapy achieves therapeutic results through the use of substances capable of producing selective injury to abnormal vascular channels. These substances, referred to as sclerosants, are introduced directly into the lesion where they initiate controlled endothelial disruption. The injury triggers a cascade of biological events including inflammation, thrombus formation, and progressive fibrosis. As this process evolves, the abnormal vessel gradually collapses and becomes permanently occluded. The choice of sclerosant depends on several factors such as the type of vascular lesion, its flow characteristics, anatomical location, and the safety profile of the agent.[15]

Ethanol

Ethanol is among the most potent sclerosants for vascular malformations, exerting its effect through rapid endothelial cell injury. When injected into abnormal vessels, ethanol extracts cellular water and denatures structural proteins, causing immediate destruction of the vessel lining, thrombosis, and subsequent fibrotic transformation of the channel.[15] Its strong sclerosing action can produce substantial lesion reduction, but this potency also raises the risk of complications. Local tissue necrosis, severe pain, nerve injury, and systemic toxicity may occur if ethanol extends beyond the target. Consequently, ethanol treatments are performed under imaging guidance with strict control of total dose.[16]

Bleomycin

Bleomycin, an antitumor antibiotic repurposed as a sclerosant, is widely used for lymphatic and venous malformations in the head and neck. Its mechanism involves interaction with DNA, forming complexes that generate free radicals and cause DNA strand breaks, leading to endothelial injury and a subsequent inflammatory response that promotes fibrosis and collapse of abnormal vascular spaces.[17] Bleomycin is typically given intralesionally after dilution, with dosing based on body weight and lesion size and often delivered across multiple sessions to limit systemic exposure. Compared with stronger agents like ethanol, bleomycin produces more controlled fibrosis with less tissue necrosis, making it preferable near vital structures. However, cumulative dosing must be monitored closely because pulmonary toxicity, including inflammatory lung injury and potential progression to pulmonary fibrosis, is the principal systemic risk. Clinical studies report encouraging reductions in lesion size and symptom improvement with bleomycin sclerotherapy, supporting its role in head and neck vascular lesion management.[18,19]

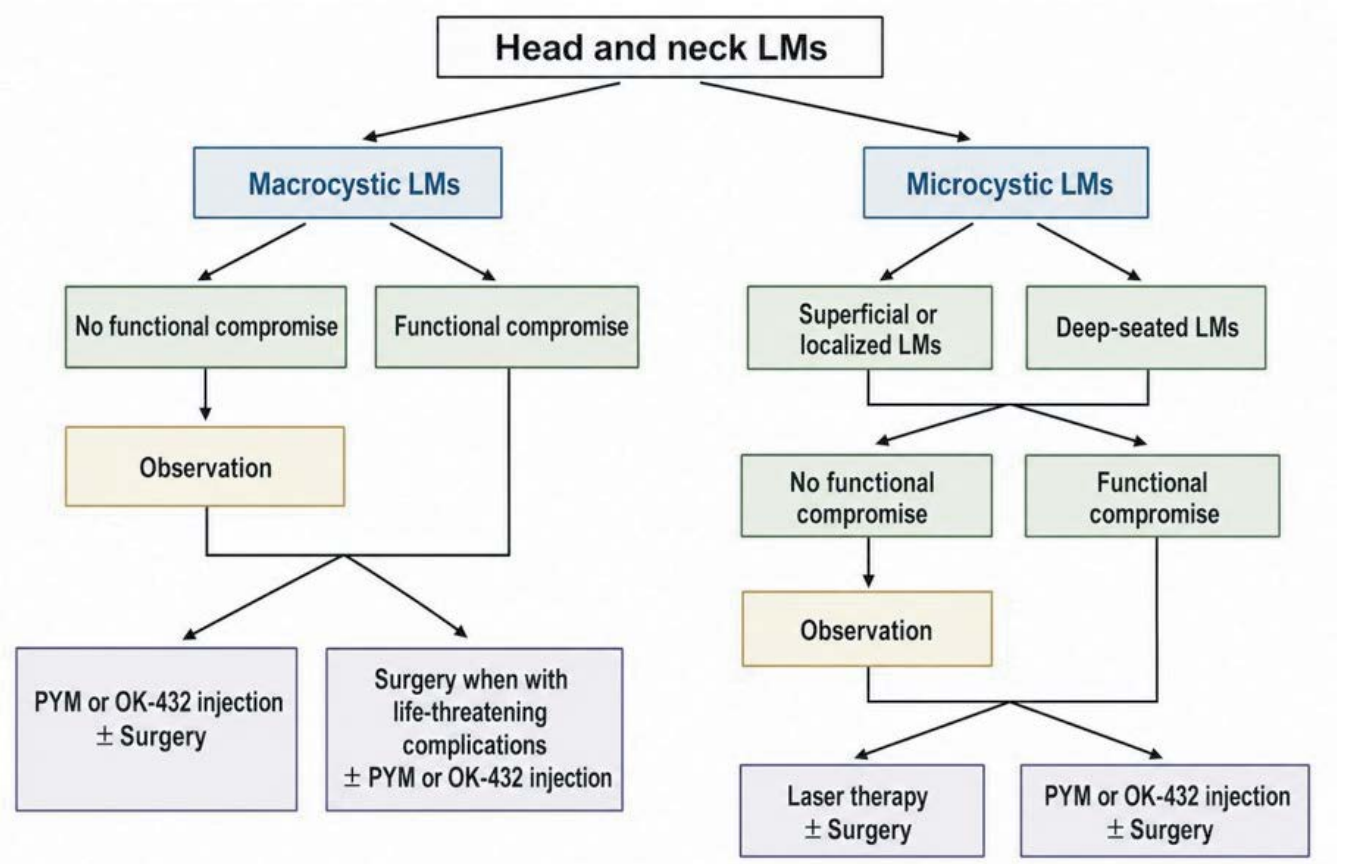


Fig. 1: Treatment decision-making algorithm for head and neck lymphatic malformations[5]

Table 1: Comparison of commonly used sclerosants in vascular malformations

Agent	*Mechanism*	*Indication*	*Advantages*	*Complications*
Ethanol	Protein denaturation	Venous malformation	Highly effective	Tissue necrosis
Bleomycin	DNA damage	Lymphatic malformation	Safer near vital structures	Pulmonary toxicity
STS	Detergent endothelial damage	Venous lesions	Widely used	Local swelling
Polidocanol	Membrane disruption	Small venous malformation	Less painful	Mild inflammation

Sodium Tetradecyl Sulfate

Sodium tetradecyl sulfate is a detergent sclerosant commonly used for venous malformations. STS disrupts lipid components of endothelial membranes, causing cell destruction, intraluminal clot formation, and subsequent fibrosis that permanently closes abnormal vessels.[15] It can be administered as a liquid or as foam; foam formulations enhance intralesional distribution and endothelial contact. STS generally yields reliable outcomes, with occasional mild local reactions such as pain or swelling.[16]

Polidocanol

Polidocanol, another detergent-type agent, similarly damages endothelial membranes and induces thrombosis followed by inflammation and fibrosis, resulting in vessel obliteration. It is often favored for causing less injection discomfort and for its favorable safety profile, though its moderate sclerosing strength may necessitate multiple treatment sessions to achieve satisfactory lesion regression.[16]

Ethanolamine Oleate

Ethanolamine oleate produces endothelial injury and thrombus formation, provoking an inflammatory response that culminates in fibrosis and lumen closure. It has been used for certain venous lesions, including oral cavity abnormalities, but requires careful administration because extravasation can provoke local inflammation or hemolytic reactions.[15]

OK-432 and Other Agents

OK-432 (Picibanil), derived from inactivated *Streptococcus pyogenes*, acts biologically by eliciting a localized immune-mediated inflammatory response that gradually shrinks and fibroses LMs. Other explored agents include povidone-iodine and hypertonic saline, which induce localized inflammation, and normal saline used as a diluent or adjunct; these have milder sclerosing effects but may be useful in selected cases. A treatment decision-making algorithm for head and neck LMs is shown in Figure 1.

Overall, sclerotherapy efficacy depends on appropriate sclerosant selection, precise intralesional delivery, and careful dose monitoring. When these elements are optimized, sclerosing agents offer a valuable, minimally invasive option that preserves surrounding structures and reduces surgical morbidity.[16] Various sclerosants, including ethanol, bleomycin, polidocanol, and STS, differ in pharmacology, efficacy, and complication risk (Table 1).

Techniques of Sclerotherapy

The outcome of sclerotherapy depends greatly on the method used to prepare and deliver the sclerosant into the vascular lesion. In clinical practice, sclerosants may be injected either in liquid form or after conversion into foam, depending on the nature of the lesion and its vascular dynamics. Careful technique is especially important in the head and neck region because abnormal vessels often lie close to critical structures such as nerves, major vessels, and the airway. Accurate needle placement and controlled injection therefore play a key role in achieving effective treatment while minimizing complications.[15]

Liquid Sclerotherapy (Fluid Technique)

Liquid sclerotherapy represents the conventional approach for delivering sclerosants. Before the procedure, the lesion is evaluated clinically and radiologically to determine its extent and vascular characteristics. The treatment area is prepared under sterile conditions, and local anesthesia may be used when necessary. A fine needle or small catheter is then introduced directly into the vascular cavity. Correct positioning of the needle is confirmed either by aspiration of blood or through imaging guidance such as ultrasound or fluoroscopy.

Once the needle is properly positioned, the sclerosant solution is injected slowly into the lesion. Slow injection is important because it allows the agent to spread evenly through the abnormal vascular channels and reduces the risk of leakage into surrounding tissues. After injection, gentle pressure may be applied to the treated area to increase the contact between the sclerosant and the vessel wall, which helps improve the sclerosing effect.[20]

Foam Sclerotherapy

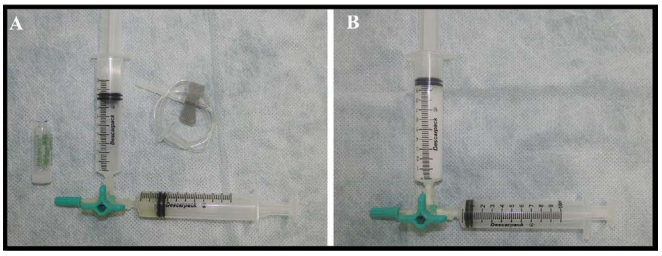
Foam sclerotherapy is a modified technique that improves the effectiveness of sclerosants by increasing their contact with the endothelial surface. When a sclerosant is converted into foam, the bubbles displace blood from the vessel lumen, allowing the chemical agent to remain in direct contact with the vessel wall for a longer period. This prolonged contact enhances endothelial damage and improves the overall therapeutic response.[16]

Tessari Technique for Foam Preparation

One of the most commonly used methods for producing sclerosant foam is the Tessari technique. This method involves the use of two sterile syringes connected by a three-way stopcock. One syringe contains the liquid sclerosant, while the second contains air. The typical proportion used is one part sclerosant to four parts air.

The contents of the syringes are pushed back and forth repeatedly through the stopcock several times. This rapid mixing creates a fine and uniform microfoam composed of numerous small bubbles. The foam should be used immediately after preparation to maintain its stability. The Tessari technique is shown in Figure 2.

After preparation, the foam is injected into the vascular lesion through a needle placed under imaging guidance. The clinician carefully observes the distribution of the foam within the lesion to ensure adequate filling of the abnormal vascular spaces while avoiding surrounding tissues.[16]



Figs 2A and B: (A) Two 10-mL syringes, one containing 2 mL of 5% ethanolamine oleate and the other containing 8 mL of air. The syringes are connected with a three-way connector. Also, one scalp vein set (25 G) and an empty ampoule of 5% ethanolamine oleate are shown; (B) After 20 cycles, 5% ethanolamine oleate foam (with a characteristic white color) was present in one syringe[3]

Careful preparation of the sclerosant, accurate needle placement, and controlled delivery of the agent are fundamental steps for achieving safe and effective sclerotherapy, particularly in vascular lesions of the head and neck where anatomical precision is essential.[20]

Clinical Outcomes of Sclerotherapy

Clinical success in sclerotherapy is gauged by volumetric reduction, symptom relief, and patient-reported outcomes.[2] Foam-based sclerosants consistently demonstrate high efficacy, with short-term improvement in over 90% of treated lesions and durable long-term benefit in many cases.[9] Reduction in lesion size correlates with alleviation of pain, decreased bleeding, and restoration of vital functions such as speech and swallowing.[14] Outcomes are strongly influenced by hemodynamics: low-flow venous and macrocystic LMs respond predictably due to prolonged sclerosant contact,[12] whereas high-flow lesions often require adjunctive measures to achieve stability.[4]

Morphology further dictates response. Macrocystic lesions allow effective sclerosant distribution and greater volume reduction,[6] while microcystic variants yield partial regression, necessitating repeated sessions or combined therapy.[12] Comparative analyses reveal that potent agents achieve rapid regression but carry higher complication risks,[11] whereas agents with controlled mechanisms provide comparable efficacy with safer profiles, particularly in anatomically sensitive regions.[3] No single sclerosant has proven universally superior, reinforcing the need for individualized treatment planning.[7] Functional outcomes are especially relevant in oral and maxillofacial practice, where reduction in lesion bulk improves airway patency, speech articulation, and masticatory function.[6,13] Lesions of the tongue and floor of the mouth benefit from enhanced swallowing and reduced obstruction.[14]

Esthetic improvement is equally significant, with gradual reduction in vascular prominence enhancing facial symmetry and patient satisfaction.[2,3,7] Multiple staged sessions are often required, permitting progressive reduction while minimizing risks of high-dose toxicity.[6,12] Long-term data indicate low recurrence in adequately treated low-flow lesions, though incomplete therapy predisposes patients to residual disease.[4] In extensive or complex cases, integration of sclerotherapy with surgery achieves durable outcomes.[10] Thus, sclerotherapy remains a cornerstone in oral and

maxillofacial surgery, balancing efficacy, safety, and functional restoration in the management of head and neck vascular malformations.

Complications of Sclerotherapy

Local Tissue Effects

Progressive expansion distorts anatomy and compromises vital structures.[5] Venous/LMs enlarge gradually, with episodes of thrombosis, infection, or intralesional hemorrhage causing pain and swelling.[6] LMs may acutely enlarge due to infection or fluid accumulation, particularly in children.[7]

Airway Compromise

Lesions of the tongue, floor of the mouth, parapharyngeal space, and cervical region pose a high risk.[8] Progressive growth or acute inflammation can precipitate obstruction, sometimes requiring intubation or tracheostomy.[6] Pediatric patients are especially vulnerable due to smaller airway dimensions.[8]

Hemorrhagic Events

High-flow arteriovenous malformations predispose to spontaneous bleeding, ulceration, necrosis, and intraoperative blood loss.[4] Even low-flow malformations may bleed following trauma or infection, leading to sudden enlargement and pain.[3]

Neurological Complications

Compression of neural structures in parotid, infratemporal, or skull base regions may occur.[5] Facial nerve involvement can cause paresis/paralysis; deeper lesions may impair cranial nerves affecting speech, swallowing, and facial expression.[2]

Functional Impairment

Oral cavity lesions compromise mastication, speech, and deglutition.[6] Tongue/floor-of-the-mouth lesions interfere with articulation and airway protection, contributing to nutritional deficits and reduced quality of life.[7,12]

Treatment-related Morbidity

Sclerotherapy risks include tissue necrosis, ulceration, nerve injury, and localized inflammation; major complication rates are ≈10%–15%, higher in high-flow lesions.[11]

Surgery Risks

Excessive bleeding, incomplete excision, recurrence, and damage to adjacent structures.[5] Microcystic LMs are particularly prone to recurrence after surgery.[12] Combined approaches may increase cumulative morbidity if not carefully planned.[10]

Psychosocial Impact

Visible facial lesions cause esthetic deformity, psychological distress, and social withdrawal.[2] This is particularly significant in pediatric/adolescent patients where facial appearance influences social development.[7]

Clinical Perspective

Management of these complications demands a multidisciplinary approach, integrating precise imaging, individualized planning, and protocol-driven intervention to minimize morbidity while optimizing both functional and esthetic outcomes in oral and maxillofacial practice.[8]

Limitations

This review has several limitations. As a narrative review, it does not follow a formal systematic methodology, and therefore selection bias cannot be completely excluded. The included studies are heterogeneous in terms of design, sample size, lesion type, sclerosant used, and follow-up duration, limiting direct comparison between agents. Quantitative synthesis and meta-analysis were not feasible due to variability in outcome measures and reporting standards. Additionally, most available evidence is derived from retrospective studies and case series, with limited high-quality comparative trials. Long-term outcomes and standardized assessment criteria remain inconsistently reported across the literature.

Conclusion

Sclerotherapy stands as a cornerstone in maxillofacial surgery, being minimally invasive, function-preserving, and esthetically restorative. Success hinges on precise diagnosis, tailored agent selection, and expert technique, ensuring durable outcomes while safeguarding airway, speech, and facial harmony in complex vascular malformations.

Declarations

Ethics Approval

Not applicable.

Patient Consent for Publication

Not applicable.

Animal Studies

Not applicable.

Author Contributions

Dishantkumar Sonpal: Conceptualization, literature search, and writing—original draft.
Khalid Mohammed Agwani: Supervision and writing—review and editing.
Ramank Mathur: Literature evaluation and validation.

All authors have read and approved the final manuscript and agree to be accountable for all aspects of the work.

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Clinical Trial Registry Number

Not applicable.

PROSPERO Registry Number

Not applicable.

Data Availability Statement

No new data were generated or analyzed in this study. All data supporting the findings of this review are derived from previously published articles, which are cited within the manuscript. Additional information is available from the corresponding author upon reasonable request.

AI Use Declaration Statement

The authors confirm that language refinement and editorial assistance were supported using artificial intelligence tools to enhance clarity, grammar, and readability. The scientific content, interpretation, and conclusions of the manuscript remain the sole responsibility of the authors. All outputs were critically reviewed, modified, and approved to ensure accuracy and originality.

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