



Overcoming wound complications in head and neck salvage surgery[☆]

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ABSTRACT

Background: Loco-regional treatment failure after radiotherapy with or without chemotherapy and/or prior surgery represents a significant portion of head and neck cancer patients. Due to a wide array of biological interactions, these patients have a significantly increased risk of complications related to wound healing.

Methods: Review of the current literature was performed for wound healing pathophysiology, head and neck salvage surgery, and wound therapy.

Results: The biology of altered wound healing in the face of previous surgery and chemoradiotherapy is well described in the literature. This is reflected in multiple clinical studies demonstrating increased rates of wound healing complications in salvage surgery, most commonly in the context of previous irradiation. Despite these disadvantages, multiple studies have described strategies to optimize healing outcomes. The literature supports preoperative optimization of known wound healing factors, adjunctive wound care modalities, and microvascular free tissue transfer for salvage surgery defects and wounds.

Conclusion: Previously treated head and neck patients requiring salvage surgery have had a variety of disadvantages related to wound healing. Recognition and treatment of these factors can help to reverse adverse tissue conditions. A well-informed approach to salvage surgery with utilization of free vascularized or pedicled tissue transfer as well as optimizing wound healing factors is essential to obtaining favorable outcomes.

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1. Introduction

The treatment of recurrent head and neck cancer (HNC) remains a difficult clinical problem for head and neck surgeons. In the era of widespread radiation, salvage surgery has become more prevalent over the past several decades. Salvage surgery occurs in a compromised tissue field which is suboptimal from several aspects. Previously irradiated tissue is characterized by impaired vascularity and altered biochemical cell function. Additionally, other factors such as malnutrition, low performance status, poor general medical condition and smoking are ubiquitous in this patient population. These factors, along with the anatomy of the upper aerodigestive tract, often lead to fistulas, abnormal scarring, chronic infection, and other wound complications after salvage surgery. Microvascular free tissue transfer has become the standard treatment for defects in head and neck salvage surgery with success rates of >90% in most series, even in the setting of previous irradiation or surgery [1]. Nevertheless, functional and reconstructive outcomes are often compromised after salvage cases due to a high prevalence of wound healing complications. Reconstruction of non-healing wounds and salvage surgery defects remain a challenge to surgeons and patients. We will review the biological and clinical problems faced in salvage surgery as well as the various evidence-based interventions to optimize clinical outcomes.

2. Wound biology and pathophysiology

The physiology of wound healing has been well studied and is often broadly divided into phases of hemostasis, inflammation, proliferation, and remodeling. Wound healing is typically a well-orchestrated interplay of multiple cell types, cytokines and growth factor that work to regulate one another [2]. Abnormal healing and chronic wounds arise from the disruption of these processes. There are multiple local and systemic factors that directly, non-directly, and synergistically contribute to poor wound healing. Local factors include vascularity, oxygenation, infection, foreign bodies, non-viable tissue, tension, and malignancy. Systemic factors include advanced age, malnutrition, chemotherapy, radiation, immunosuppression, stress, pharmacology, endocrinopathy, smoking, alcohol, and other systemic disease. Salvage surgery in the setting of head and neck cancer invariably involves several of these factors including general performance measures such as Karnofsky Performance Score and ECOG Performance Status which have been independently associated with wound complications in head and neck surgery [3,4].

A significant subset of head and neck oncologic patients suffer from loco-regional recurrence or secondary primary cancers that require multiple surgeries as well as radiation and chemotherapy to achieve disease control. Previously operated fields exhibit distorted tissue planes and fascial compartments which are replaced with scarring (see Fig. 1). Additionally, disruptions of normal vascular and lymphatic pathways lead to hypo-perfused tissue, higher tension, and susceptibility to poor healing. Previous neck dissection has been associated with increased wound infections, revision surgery, and free flap failure [4]. Most head and neck cancers involve the upper



Fig. 1. Wound break down and fistula following salvage surgery for a recurrent laryngeal cancer. Radiation has a significant impact on not only the mucosa but also the overlying skin.

digestive tract which introduces micro-organisms through saliva, potentially leading to infection, fistula formation, or other healing complications. Perhaps most importantly, vessel depletion and absence of landmarks can be problematic for additional surgery and reconstructive efforts. Surgery in a previously treated field greatly increases the technical difficulty of surgery and consequently increases operative time, surgical complications, and often requires more complex reconstruction techniques [4–6]. These cumulative factors present a hostile environment for reconstruction and healing.

The effects of chemotherapy and radiotherapy are pervasive and represent fundamental cellular changes. The hallmarks of biochemical tissue response to radiation are inflammation, hypercoagulability and fibrosis. These changes are initiated by acute cellular injury followed by an abnormal inflammatory response that leads to excess collagen deposition. These effects are perhaps most evident in blood vessel changes. Morphologic changes to all layers of irradiated vessels have been described. The inflammatory cell content is increased in the intima of the irradiated head and neck vessels [7]. The tunica intima layer is thickened and dehiscence from the tunica media with reduced numbers of endothelial cells that are vacuolized. Similarly, the tunica media and adventitia show fibrosis, hyalinosis and loss of smooth muscle cells and fibroblasts. Normal extracellular matrix is reduced and replaced by calcification [8]. These changes are initially acute but become chronic with abnormal collagen deposition without appropriate remodeling. Radiation induced vascular pathology differs quantitatively and qualitatively from age-related atherosclerosis [9]. Similar to the blood vessel changes, surrounding connective tissue and lymphatic systems become chronically hypoxic and hypocellular [8]. In the larger tissue bed, blood vessels become sparse, smaller in diameter, and less responsive to angiogenic or vasodilatory mediators [1]. Ultimately, tissue treated with radiation is poorly equipped to react to new injury and thus more prone to developing wound complications.

The specific role of chemotherapy to local tissue has not been well studied. Studies have drawn statistically significant associations with prior chemotherapy to wound complications [8]. However, in curative head and neck oncologic treatment,

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