ASSESMENT OF PATENCY OF CAROTID ARTERY AND INTERNAL JUGULAR VEIN FOLOWING NECK DISSECTION AND ADJUVANT RADIOTHERAPY IN HEAD AND NECK CANCER PATIENTS-A PROSPECTIVE OBSERVATIONAL STUDY

By

DR. GOKUL P.M

DISSERTATION SUBMITTED TO

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, KOLAR

In partial fulfillment of the requirements for the degree of

MASTER OF SURGERY

IN

OTORHINOLARYNGOLOGY

Under the guidance of

DR. AZEEM MOHIYIDDIN
MBBS, MS (E.N.T), FICS.,FACS.,MNAMS,
SEKHSARIA FELLOWSHIP IN HEAD AND NECK SURGERY ,
AND
DR.ANIL KUMAR SAKALECHA
MBBS,MD RADIODIAGNOSIS

DEPARTMENT OF OTORHINOLARYNGOLOGY
SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA KOLAR
2025

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Dr. S.M AZEEM MOHIYUDDIN,
MBBS, MS, FACS, FICS, MNAMS,
SEKHSARIA FELLOWSHIP
IN HEAD AND NECK SURGERY
Professor AND HOD
Department of Otorhinolaryngology,
Sri Devaraj Urs Medical College,
Tamaka, Kolar

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DATE :

PLACE :

DR.ANIL KUMAR SAKALECHA
MBBS,MD RADIODIAGNOSIS
PROFESSOR AND HOD
Department of radio-diagnosis
sri devaraj urs medical college
tamaka, kolar- 563101

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DATE :
PLACE:

Signature of the HOD

Dr. S.M AZEEM MOHIYUDDIN,
MBBS, MS, FACS, FICS, MNAMS,
SEKHSARIA FELLOWSHIP
IN HEAD AND NECK SURGERY
Professor AND HOD
Department of Otorhinolaryngology,
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

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SIGNATURE OF HOD

Dr. S.M.AZEEM MOHIYUDDIN,
M.B.B.S.,M.S, FICS.,FACS.,MNAMS,
SEKHSARIA FELLOWSHIP
IN HEAD AND NECK SURGERY
Professor and HOD
Department of Otorhinolaryngology
Sri Devaraj Urs Medical College,
Tamaka, Kolar.

Date:

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DR. PRABHAKAR .K
M.B.B.S, M.D, MNAMS,FICP,AFISC,FIAMS
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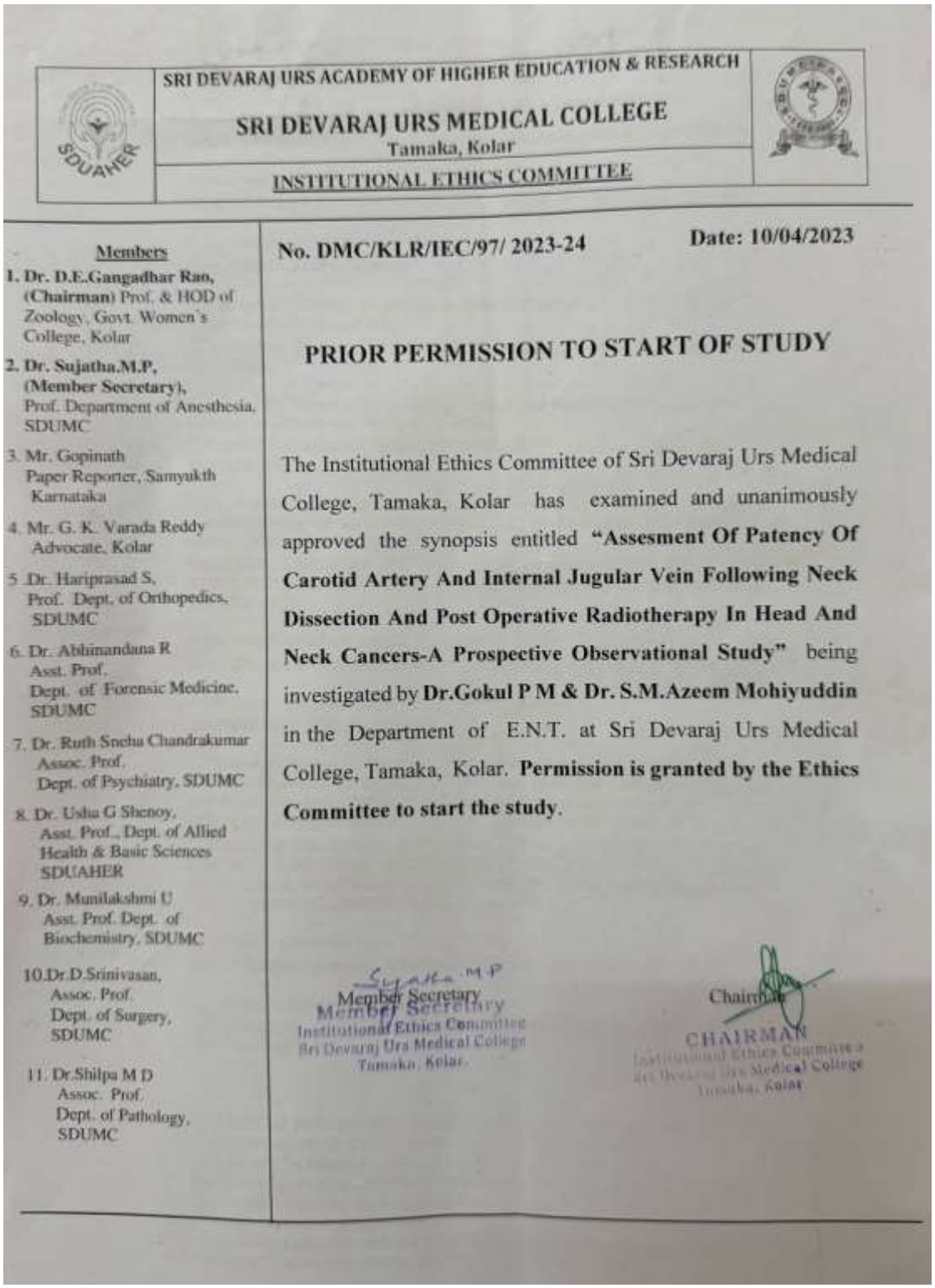

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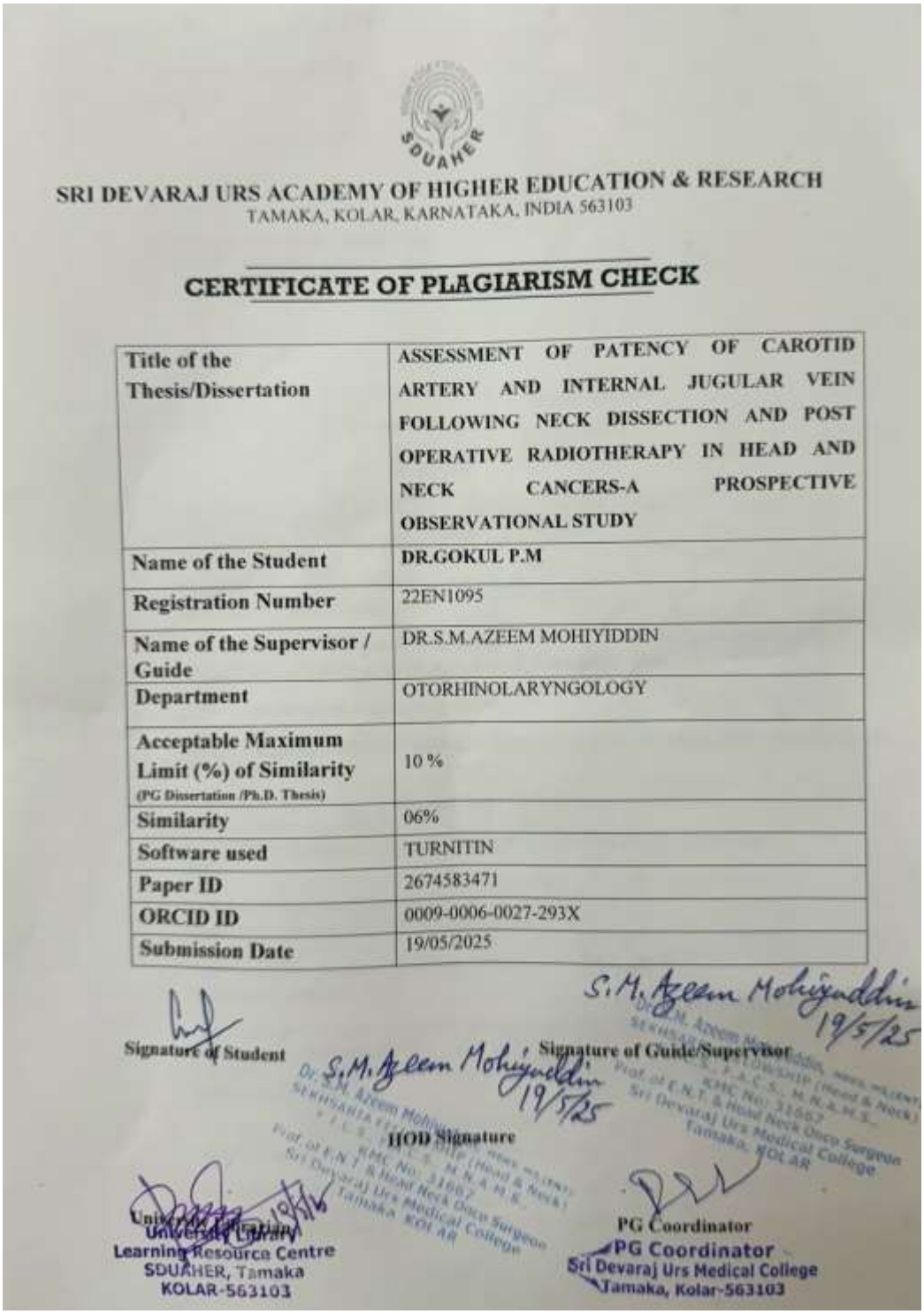

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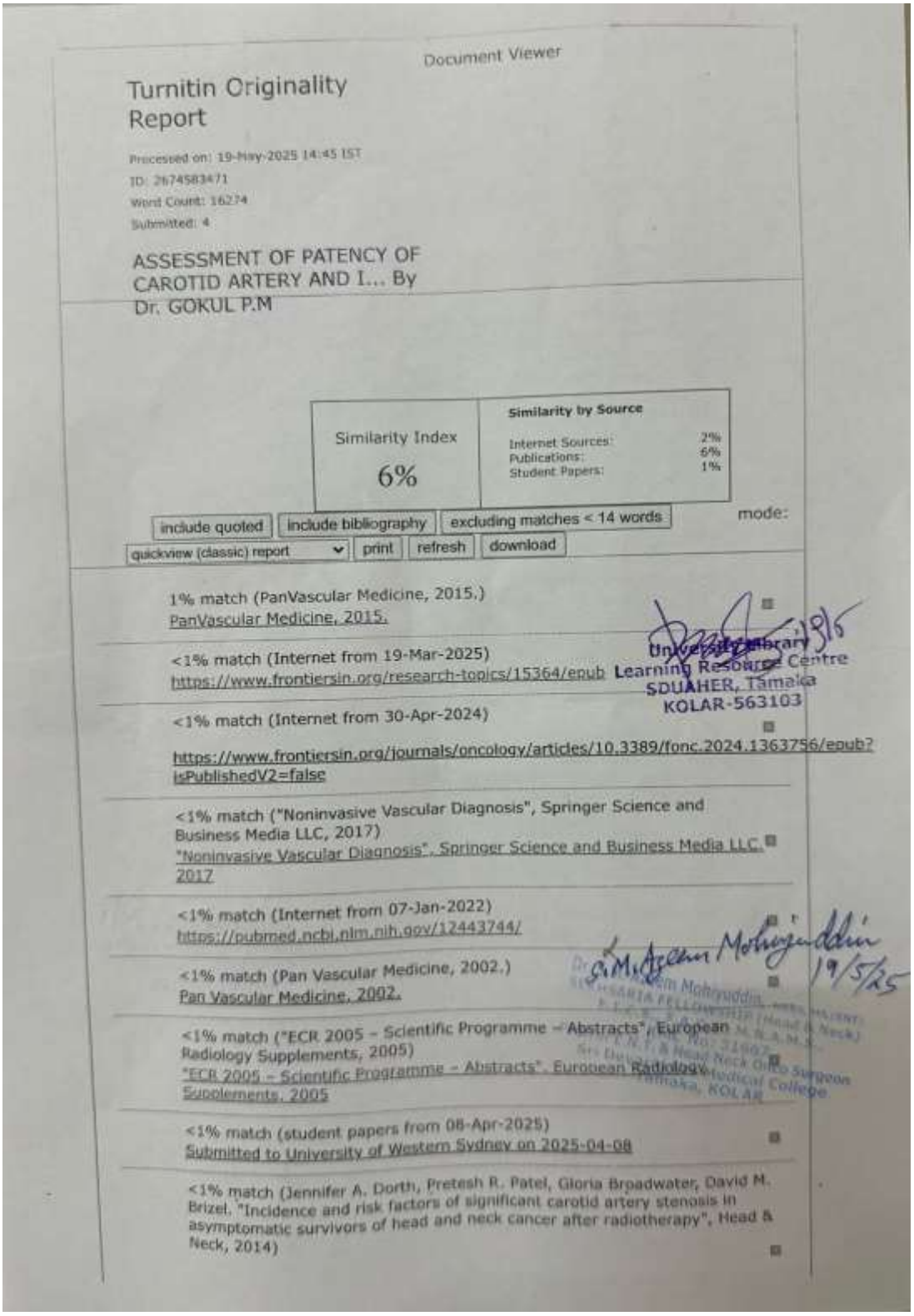
S.M. Azeem Mohiyuddin
19/5/25

Dr. S.M. Azeem Mohiyuddin, MBBS, MS(ENT),
SEKHSARIA FELLOWSHIP (Head & Neck)
F.I.C.S., F.A.C.S., M.N.A.M.S.,
KMC No: 31657
Prof. of E.N.T. & Head Neck Onco Surgeon
Sri Devaraj Urs Medical College
Tamaka, KOLAR



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ASSESSMENT OF PATENCY OF CAROTID ARTERY AND INTERNAL JUGULAR VEIN FOLLOWING NECK DISSECTION AND POST OPERATIVE RADIOTHERAPY IN HEAD AND NECK CANCERS - A PROSPECTIVE OBSERVATIONAL STUDY

ABSTRACT

Background

Oral Head and neck cancer treatment often involves neck dissection and adjuvant RT, which may compromise vascular integrity with potential long-term cerebrovascular sequelae. While radiation-induced CS is well-documented, the immediate vascular effects of multimodality treatment remain incompletely characterized. Contemporary techniques including IMRT and refined surgical approaches may modify vascular risks compared to historical cohorts.

Aim and objective

1. To evaluate CCA and IJV preoperatively by carotid doppler ultrasound and document the patency, size of lumen and flow of blood.
2. To evaluate patency, size of lumen, and flow of blood of CCA and IJV in patients who have undergone neck dissection after 2 weeks
3. To assess whether adjuvant RT has affected the lumen, Blood flow and patency of CCA and IJV in above patients after 4 weeks and all these results are compared preoperative, postoperative and after RT

Methodology

This prospective observational study enrolled 60 patients (71.7% female; mean age 55.48±11.8 years) diagnosed with head and neck malignancies requiring neck dissection and adjuvant RT between May 2023 and October 2024. Patients with previous neck radiation, cervical trauma, or severe kyphoscoliosis were excluded. All participants underwent MRND (58.3% left-sided, 41.7% right-sided) followed predominantly by RT alone (93.3%), with most receiving 64 Gy (41.7%) or 66 Gy (25.0%) delivered in 32 fractions (45.0%). Vascular assessment was performed via Doppler ultrasonography at three sequential timepoints: preoperative (baseline), post-operative (2 weeks), and post-RT (4 weeks). Comprehensive hemodynamic profiling included PSV, EDV, and PSV ratios of CCA and ICA, with bilateral CIMT measurements. Statistical analysis employed Wilcoxon signed-rank tests for paired comparisons across treatment phases, with significance established at $p<0.05$.

Results

Hemodynamic parameters demonstrated remarkable stability throughout the treatment course for the overall cohort. The proportion of patients with normal CCA PSV slightly increased from preoperative (96.7%) to post-operative and post-RT phases (both 98.3%), with no statistically significant differences ($p=0.77$). All participants maintained normal ICA PSV, normal EDV, and normal PSV ratios (<2) throughout the assessment period. Mean CCA PSV showed

modest progressive increases from baseline (93.73±11.29 cm/s) to post-operative (94.23±9.79 cm/s) and post-RT (98.20±8.58 cm/s) phases, representing a cumulative 4.77% increment that remained well below the threshold for hemodynamically significant stenosis. In contrast, CIMT demonstrated statistically significant progressive increases bilaterally across all treatment phases. Mean CIMT values increased from preoperative (right: 0.44±0.10 mm; left: 0.51±0.12 mm) to post-operative (right: 0.48±0.12 mm; left: 0.55±0.15 mm) and post-RT (right: 0.54±0.14 mm; left: 0.60±0.18 mm), constituting cumulative increases of 22.7% and 17.6% from baseline, respectively. Notably, comorbidity-specific subgroup analysis revealed significant differences in vascular response patterns. The hyperlipidaemia subgroup exhibited the most pronounced alterations, with statistically significant increases in both PSV (104.0±19.4 cm/s to 108.2±6.0 cm/s, p=0.013) and CIMT (0.51±0.15 mm to 0.61±0.16 mm, p=0.018). Diabetic patients showed significant PSV increases (p=0.041) but non-significant CIMT progression (p=0.071), while hypertensive patients demonstrated non-significant changes in both parameters.

Conclusion

This prospective study demonstrates a dissociation between morphological vascular alterations (significant CIMT increases) and hemodynamic compromise (preserved flow parameters) following multimodality head and neck cancer treatment. The identification of comorbidity-specific vascular response patterns,

with hyperlipidaemia demonstrating the most pronounced alterations, provides a foundation for risk-stratified surveillance protocols. The documentation of surgery-attributable CIMT increases highlights the potential vascular impact of neck dissection independent of RT effects. These findings suggest that early post-treatment surveillance focused exclusively on flow parameters may underestimate evolving vasculopathy, particularly in hyperlipidaemic patients. While immediate hemodynamic compromise appears minimal, the progressive CIMT increases observed warrant extended follow-up to determine long-term cerebrovascular implications and support consideration of targeted preventive interventions in high-risk subgroups.

Keywords

Carotid Stenosis; Radiotherapy; Head and Neck Neoplasms; Neck Dissection; Carotid Intima-Media Thickness; Ultrasonography, Doppler; Vascular Patency

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ABBREVIATIONS

Abbreviation	Explanation
CCA	Common Carotid Artery
ICA	Internal Carotid Artery
PSV	Peak Systolic Velocity
EDV	End-Diastolic Velocity
CIMT	Carotid Intima-Media Thickness
IJV	Internal Jugular Vein
HNC	Head And Neck Cancers
HPV	Human Papillomavirus
TORS	Transoral Robotic Surgery
IMRT	Intensity-Modulated Radiotherapy
MRA	Magnetic Resonance Angiography
CTA	Computed Tomography Angiography
TIA	Transient Ischemic Attacks
RI	Resistive Index

IMT	Intima-Media Thickness
CEA	Carotid Endarterectomy
CAS	Carotid Artery Stenting
CVD	Cardiovascular Disease
MRND	Modified Radical Neck Dissection
IMRT	Intensity-Modulated Radiation Therapy
VMAT	Volumetric Modulated Arc Therapy
DM	Diabetes Mellitus
HTN	Hypertension
RT	Radiotherapy
CT	Chemotherapy
CS	Carotid artery stenosis
ND	Neck Dissection
ECA	External Carotid Artery
QOL	Quality of Life

INTRODUCTION

HNCs constitute a major health burden, with documenting 890,000 new cases diagnosed annually worldwide.[1] The management of these malignancies frequently involves radical neck dissection, a surgical procedure that entails the removal of lymph nodes and surrounding soft tissues, often followed by adjuvant RT for patients with adverse pathological features.[2] While these therapeutic interventions have significantly improved oncological outcomes, they may induce considerable morbidity, including potential alterations in the vascular structures of the neck region.[3]

The CCA and IJV represent critical vascular structures in the neck that play fundamental roles in cerebral perfusion and venous drainage, respectively. Neck dissection procedures, particularly radical and modified radical variants, can significantly impact these vessels through direct manipulation, devascularization, or denervation of surrounding tissues.[4] Moreover, post-operative RT, while essential for locoregional control, may induce progressive radiation-induced vasculopathy characterized by endothelial damage, medial necrosis, and adventitial fibrosis, potentially leading to stenosis, thrombosis, or occlusion of these vital vessels.[5]

Vascular complications following neck dissection and RT have been well-documented in the literature, with reported incidences of carotid stenosis ranging

from 3% to 25% depending on the extent of surgery, radiation dose, treatment modality, and patient-specific risk factors.[6–8] These complications may manifest as CS, accelerated atherosclerosis, carotid blowout syndrome, or IJV thrombosis, which can have devastating consequences including stroke, intracranial HTN, or even death.[9,10]

Radiation-induced carotid artery stenosis represents a particularly concerning late complication, with studies indicating a significantly higher prevalence of CS in patients who have undergone RT for HNCs compared to age-matched controls.[11,12] The pathophysiology involves radiation-induced inflammation, fibrosis, and accelerated atherosclerosis, leading to progressive narrowing of the vessel lumen and compromised cerebral blood flow.[13] Recent meta-analyses have demonstrated that RT for nasopharyngeal carcinoma significantly increases the risk of carotid stenosis, with a pooled odds ratio of 4.07.[14]

The integration of advanced imaging modalities, particularly carotid Doppler ultrasonography, has facilitated the non-invasive assessment of vascular patency and flow dynamics in head and neck cancer survivors.[15,16] These techniques offer valuable insights into treatment-related vascular changes, enabling the early detection of subclinical abnormalities before the onset of clinically apparent complications.[17,18]

Despite the clinical significance of these vascular sequelae, there remains a paucity of prospective data systematically evaluating the evolution of CCA and

IJV changes following combined modality treatment for HNCs. Most existing studies are retrospective in nature, with heterogeneous patient populations, variable treatment protocols, and inconsistent follow-up periods, limiting the generalizability of their findings.[19,20]

Understanding the evolution, risk factors, and clinical implications of vascular changes following neck dissection and RT is essential for optimizing surveillance strategies, implementing preventive measures, and developing targeted interventions to mitigate the risk of vascular complications in this vulnerable patient population.[21] Additionally, such knowledge may inform treatment decisions, including the extent of surgical resection, radiation dose constraints, and the potential role of antiplatelet or anticoagulant therapies in selected high-risk patients.[22,23]

The present prospective observational study aims to address this knowledge gap by systematically evaluating the patency of the CCA and IJV in patients with HNCs who have undergone neck dissection and post-operative RT, thereby providing valuable evidence to guide clinical practice and future research directions in this important area of head and neck oncology.

OBJECTIVES

1. To evaluate CCA and IJV preoperatively by carotid doppler ultrasound and document the patency, size of lumen and flow of blood.
2. To evaluate patency, size of lumen, and flow of blood of CCA and IJV in patients who have undergone neck dissection after 2 weeks
3. To assess whether adjuvant RT has affected the lumen, Blood flow and patency of CCA and IJV in above patients after 4 weeks and all these results are compared preoperative, postoperative and after RT

REVIEW OF LITERATURE

Embryology of head and neck structures

The embryology of head and neck structures is an intricate process that involves the formation of various tissues and organs from the initial embryonic layers. During the fourth week of embryonic development, the pharyngeal arches, also known as branchial arches, emerge. These arches contribute to the formation of the face, neck, and associated structures. Each arch consists of an ectodermal outer layer, a mesodermal core, and an endodermal inner lining. Neural crest cells migrate into these arches, playing a crucial role in the development of craniofacial elements, including the bones, cartilage, and connective tissues. Additionally, the development of the thyroid, thymus, and parathyroid glands originates from the endodermal pouches of these arches. Disturbances in this meticulous process can lead to congenital anomalies such as cleft lip or palate.[24,25]

Epidemiology as well as Burden of HNCs

Global and Regional Prevalence

HNCs represent a significant global health burden, ranking as the sixth most common malignancy worldwide. According to the GLOBOCAN 2018 estimates, approximately 834,000 new cases of HNCs were diagnosed globally, accounting for 4.6% of all cancer cases.[1] The geographic distribution of HNCs demonstrates

marked heterogeneity, with the highest incidence rates observed in South and Southeast Asia, Eastern Europe, and specific regions of Latin America along with the Caribbean. Recent trend analyses indicate a concerning escalation in global HNC incidence, with Zhou et al. (2024) reporting a 59.4% increase in incident cases between 1990 and 2019, rising from 461,600 to 736,000 cases annually.[26]

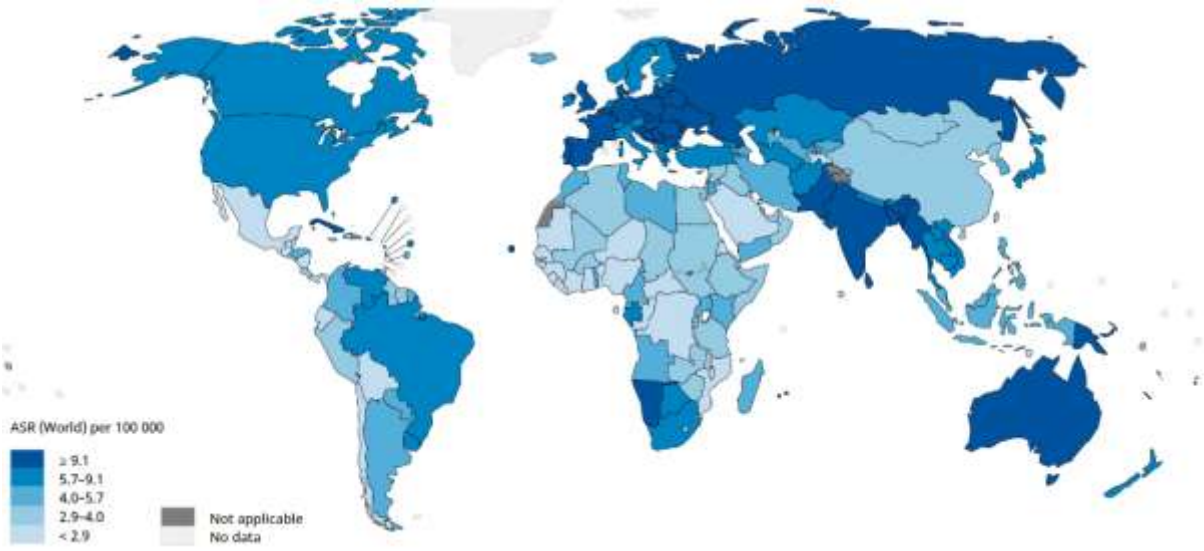

Figure 1: Age-standardised occurence rates of HNC.

The burden of HNCs exhibits pronounced regional disparities. India alone accounts for approximately one-third of the global HNC burden, with an age-standardized incidence rate of 33.4 per 100,000 population, significantly exceeding the global average of 14.3 per 100,000.[27] Analysis of 37 population-based cancer registries in India revealed that HNCs constitute 30% of all cancers among males and 11% among females in certain regions.[27] In contrast, Western countries demonstrate comparatively lower incidence rates but are experiencing

a recent surge in oropharyngeal cancers, predominantly attributed to HPV infections.[28]

Mortality and Morbidity Statistics

HNCs are associated with substantial mortality and morbidity globally. The GLOBOCAN 2018 data documented approximately 430,000 annual deaths from HNCs worldwide, representing 4.2% of total cancer mortality.[1] The mortality-to-incidence ratio for HNCs approximates 0.5, indicating that nearly half of diagnosed patients succumb to their disease, though this ratio varies significantly across regions.[29]

Lower-income countries bear a disproportionate mortality burden, with 5-year survival rates of 30-40% compared to 60-70% in high-income nations.[29] In India, HNCs account for approximately 48,000 deaths annually, with mortality rates significantly higher among males than females.[30] The morbidity associated with HNCs and their treatment is equally substantial, encompassing functional impairments in speech, swallowing, and respiration, as well as profound psychosocial consequences affecting quality of life.[31]

Risk Factors and Etiological Considerations

The aetiology of HNCs is multifactorial, with tobacco use and alcohol consumption established as primary risk factors, exhibiting both independent and synergistic effects. Tobacco use, including smokeless tobacco products such as

gutkha and khaini, is implicated in approximately 80% of HNC cases in South Asia.[32] A comprehensive study by Chauhan et al. (2022) demonstrated that 84% of HNC patients had a history of tobacco consumption, with a significant male preponderance (male-to-female ratio of 4.5:1).

HPV infection, particularly HPV-16, has emerged as a crucial etiological factor in oropharyngeal cancers, associated with distinct clinicopathological characteristics and improved prognosis compared to tobacco-related malignancies.[33] Additional risk factors include betel quid chewing, occupational exposures to carcinogens, nutritional deficiencies, and genetic predisposition.[34]

The epidemiological landscape of HNCs is evolving, with declining tobacco-related oral cavity cancers in certain regions juxtaposed against increasing HPV-associated oropharyngeal cancers, particularly among younger populations without traditional risk factors.[28] This shifting epidemiological profile necessitates adaptation of preventive strategies and management protocols to address the changing etiological spectrum of HNCs.

Management of HNCs

Surgical Approaches with Focus on Neck Dissection Techniques

Surgical intervention is a cornerstone in the management of HNCs, with neck dissection being a critical component for assessing and controlling regional lymphatic spread. Modern surgical approaches have evolved to include

minimally invasive techniques, such as TORS as well as laser microsurgery, which aim to reduce patient morbidity while maintaining oncological efficacy. These techniques allow for precise tumour resection with minimal impact on surrounding healthy tissues, thus preserving function and quality of life. Neck dissection, specifically, has shifted from radical procedures to more targeted approaches, such as selective neck dissection, which involves removing only the lymph nodes at highest risk of metastasis. This evolution reflects a broader trend towards personalized medicine, where the extent of surgery is tailored to the individual patient's disease characteristics.[35]

Evolution of Neck Dissection Classification

The classification of neck dissection has undergone significant refinement over the years, driven by a better understanding of the patterns of lymphatic drainage and metastasis in HNCs. The traditional radical neck dissection, which involved the removal of all cervical lymph nodes along with non-lymphatic structures such as the sternocleidomastoid muscle, IJV, along with spinal accessory nerve, has largely been replaced by more conservative approaches. MRND preserves one or more non-lymphatic structures, reducing surgical morbidity without compromising oncological outcomes. The introduction of the selective neck dissection, which targets specific lymph node groups based on the primary tumour’s site and stage, represents a significant advancement in surgical

management. This approach minimizes unnecessary tissue removal, thereby reducing complications and enhancing recovery.

Indications for Adjuvant Therapy

Adjuvant therapy, including RT and chemoradiotherapy, plays a pivotal role in the comprehensive management of HNCs, particularly in patients with high-risk pathological features. Indications for adjuvant RT typically include positive surgical margins, perineural invasion, and high lymph node burden. Chemoradiotherapy is often indicated in cases with extracapsular spread of nodal disease or involved surgical margins, offering a synergistic effect that enhances local and regional control. The decision to employ adjuvant therapy is multifactorial, considering tumour histology, stage, and patient comorbidities. The advancements in RT techniques, such as IMRT, have improved the precision of radiation delivery, minimizing exposure to surrounding healthy tissues and thereby reducing treatment-related toxicities. The integration of adjuvant therapies into treatment protocols underscores the importance of a multidisciplinary approach in optimizing patient outcomes.[36]

Vascular Anatomy of the Neck

Normal Anatomy of Carotid Artery

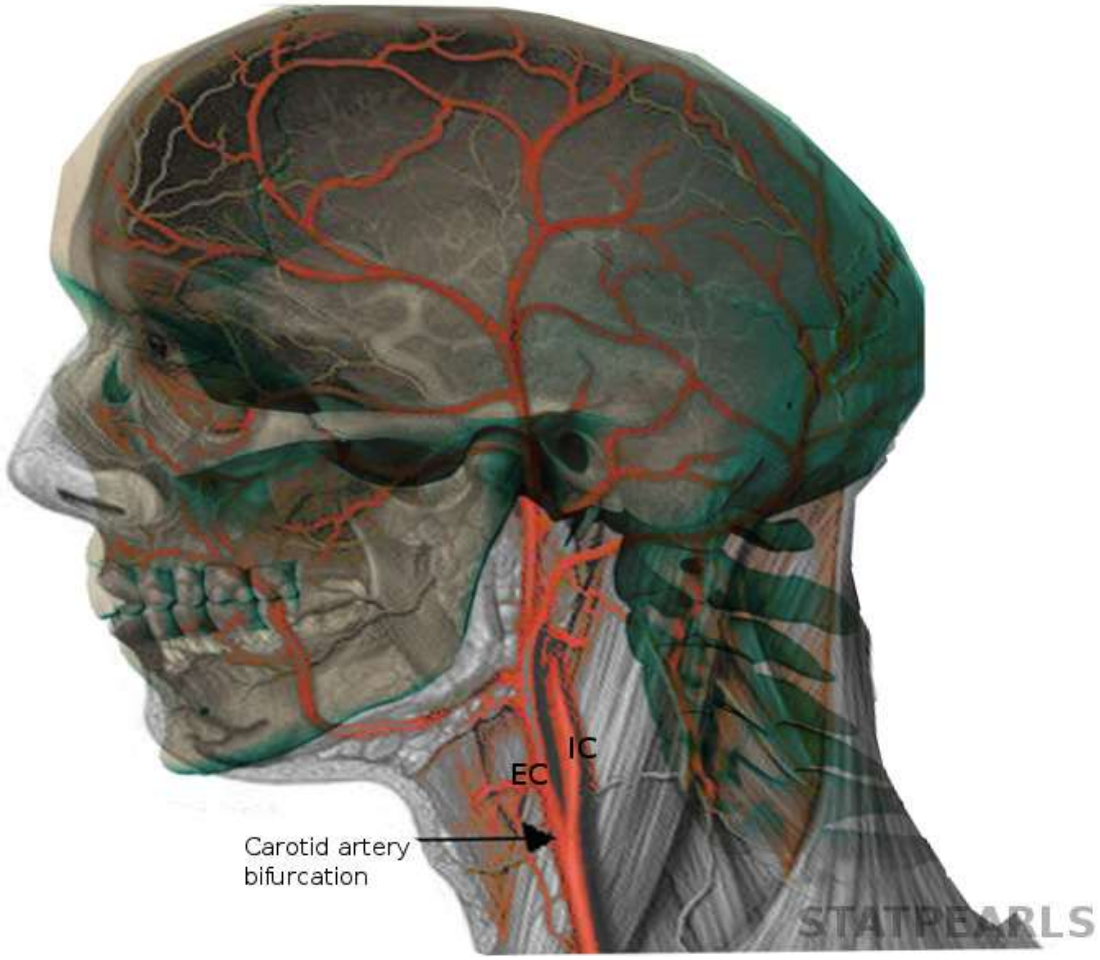

Figure 2: ***Carotid artery***[37]

They are considered as foremost blood vessels located on each side of the neck, accountable for providing oxygenated blood to the brain, neck, along with face. They originate from the aorta, with the right carotid artery branching off the brachiocephalic trunk and the left directly from the aortic arch. Each carotid

artery bifurcates into two main branches: the ICA, which supplies blood to the brain, as well as the ECA, which supplies the face and neck.[38]

The ICA ascends to the base of the skull without branching in the neck, entering the cranial cavity through the carotid canal. Within the skull, it branches into the ophthalmic artery, anterior choroidal artery, and posterior communicating artery, before dividing into the anterior and middle cerebral arteries. The carotid sinus, located at the bifurcation of the CCA into the internal and external branches, contains baroreceptors that help regulate blood pressure.[39]

The external carotid artery, in contrast, gives rise to several branches in the neck, including the superior thyroid, lingual, facial, occipital, posterior auricular, ascending pharyngeal, maxillary, as well as superficial temporal arteries. These branches supply blood to various structures in the neck, face, scalp, and base of the skull. This anatomical distinction between the ICA and ECA is crucial for surgical interventions and diagnostic imaging.[40]

Understanding the normal anatomy of the carotid arteries is essential for diagnosing and managing vascular diseases, such as carotid artery stenosis, which can lead to serious complications like stroke. Advancements in imaging techniques, such as MRA and CTA, have enhanced the visualization of carotid artery anatomy, allowing for more accurate assessments of vascular health.[41]

Histology of CCA

The CCA exhibits the classical three-layered structure characteristic of elastic arteries. The tunica intima consists of a continuous monolayer of endothelial cells resting on a basal lamina with an underlying subendothelial layer containing sparse smooth muscle cells as well as connective tissue elements.[42] The endothelium expresses von Willebrand factor and CD31, serving as key immunohistochemical markers. The internal elastic lamina forms a prominent, corrugated boundary separating the intima from the media.[43]

The tunica media, the thickest layer, contains numerous concentric elastic lamellae (40-70 in number) interspersed with smooth muscle cells arranged circumferentially, along with collagen fibres and proteoglycans. This elastic framework confers significant distensibility, allowing the vessel to accommodate the pulsatile pressure generated by cardiac contraction. The external elastic lamina delineates the media from the adventitia.[44]

The tunica adventitia, composed primarily of collagen fibres (predominantly type I), contains fibroblasts, adipocytes, and vasa vasorum that provide nutrients to the outer portions of the vessel wall. Sympathetic nerve fibres are distributed throughout this layer, regulating vascular tone.[45]

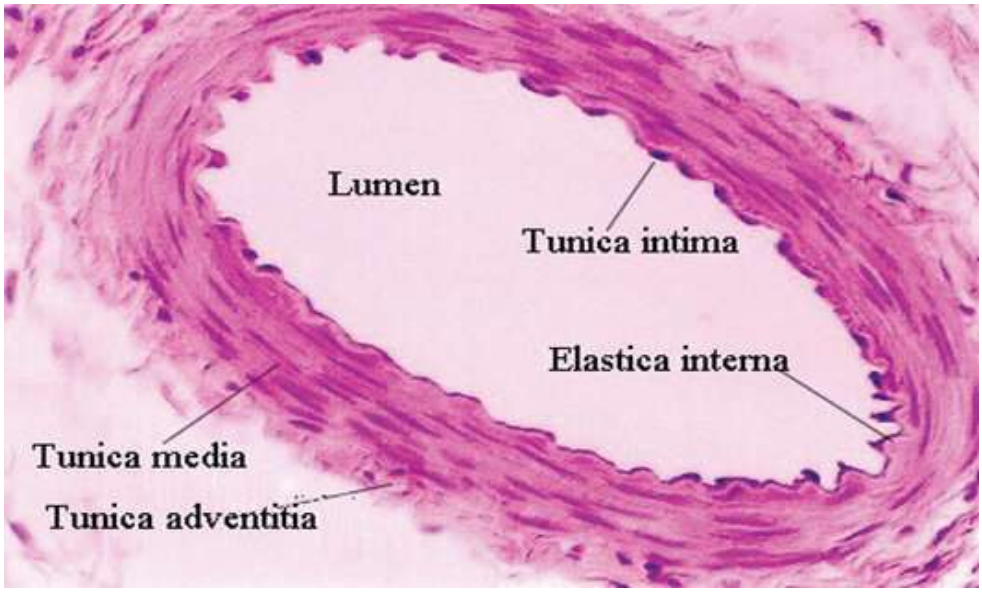

***Figure 3:* Histology of CCA**[46]

Normal Anatomy of IJV

They are a chief venous structure in the neck, responsible for draining blood from the brain, face, and neck. It originates at the base of the skull, as a continuation of the sigmoid sinus, and descends within the carotid sheath. The carotid sheath is a connective tissue structure that also contains the CCA and the vagus nerve, reflecting the close anatomical relationship among these critical structures.[47]

As the IJV travels down the neck, it receives blood from several tributaries, including the facial, lingual, pharyngeal, superior thyroid, along with middle thyroid veins. These tributaries ensure efficient drainage from the head and neck into the venous system. The IJV terminates by joining the subclavian vein to form the brachiocephalic vein, which subsequently drains into the superior vena cava, ultimately returning deoxygenated blood to the heart.[48]

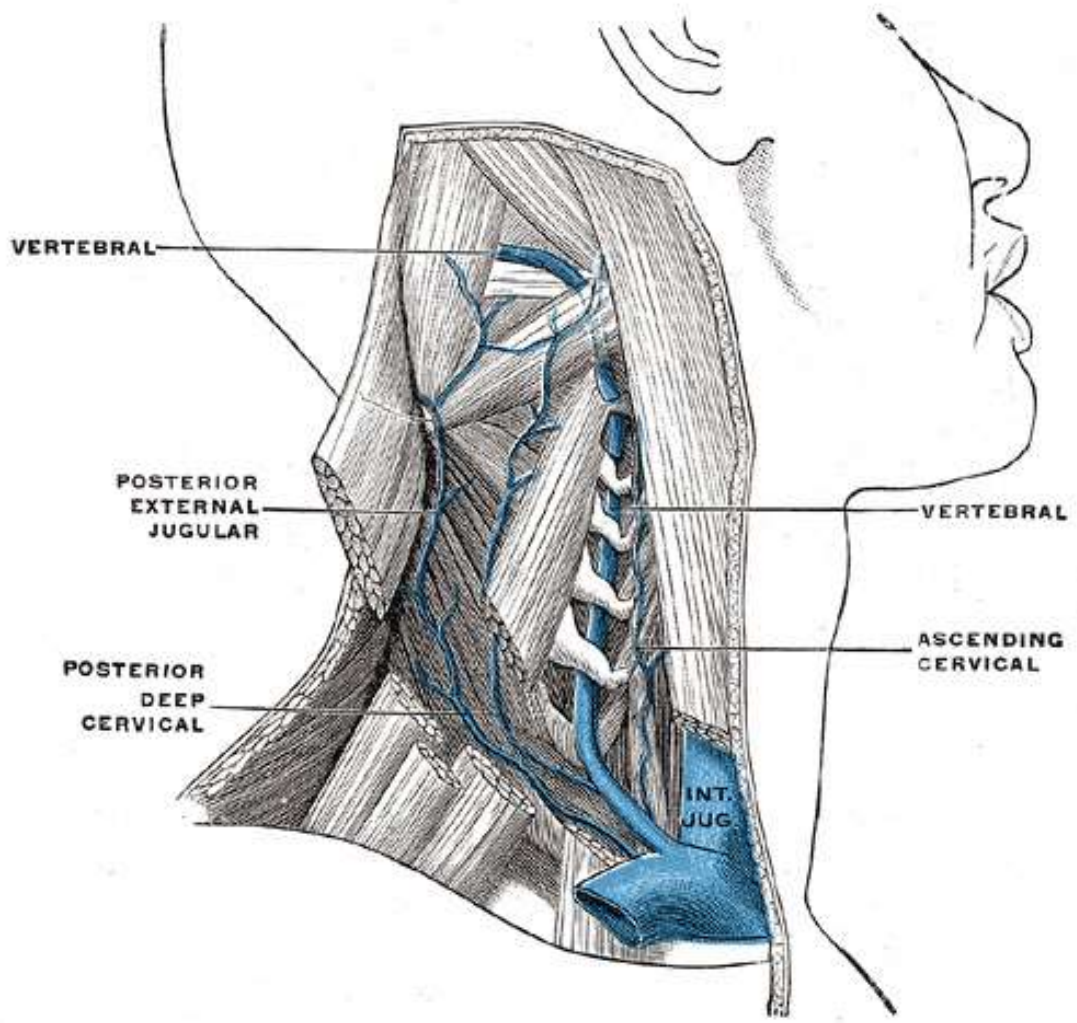

Figure 4: IJV and its Branches[49]

The IJV's location and size make it a preferred site for central venous access and catheterization, commonly performed in critical care settings. Its palpable nature, especially when the patient is in the Trendelenburg position, facilitates the insertion of catheters for monitoring central venous pressure or administering medications and fluids. The IJV's anatomical variations, such as differences in diameter or its relationship to the carotid artery, can influence the success of these procedures and necessitate careful imaging and technique.[50]

Understanding the normal anatomy and variations of the IJV is crucial for clinicians performing surgical and diagnostic interventions in the neck region. Advances in imaging techniques, such as ultrasound and CT angiography, have enhanced the ability to visualize the IJV and its surrounding structures, improving procedural safety and outcomes. [47]

IJV Histology

The IJV demonstrates a thinner wall compared to the CCA, with less distinct layering. The tunica intima consists of endothelial cells with an underlying delicate subendothelial layer. Notably, the IJV possesses bicuspid valves, particularly at its junction with the subclavian vein, preventing retrograde blood flow.[51]

The tunica media is significantly thinner than in arteries, containing fewer smooth muscle cells arranged both longitudinally and circumferentially, with minimal elastic fibers. This structural arrangement permits substantial distensibility, accommodating variations in blood volume.

The tunica adventitia constitutes the thickest layer, composed predominantly of collagen fibers with scattered fibroblasts, providing structural support. The IJV lacks vasa vasorum due to its thin wall and lower metabolic demands.[52]

Comparative immunohistochemical analysis reveals differential expression patterns, with smooth muscle actin and desmin prominently expressed in the CCA

media, while the IJV demonstrates lower expression levels of these contractile proteins.

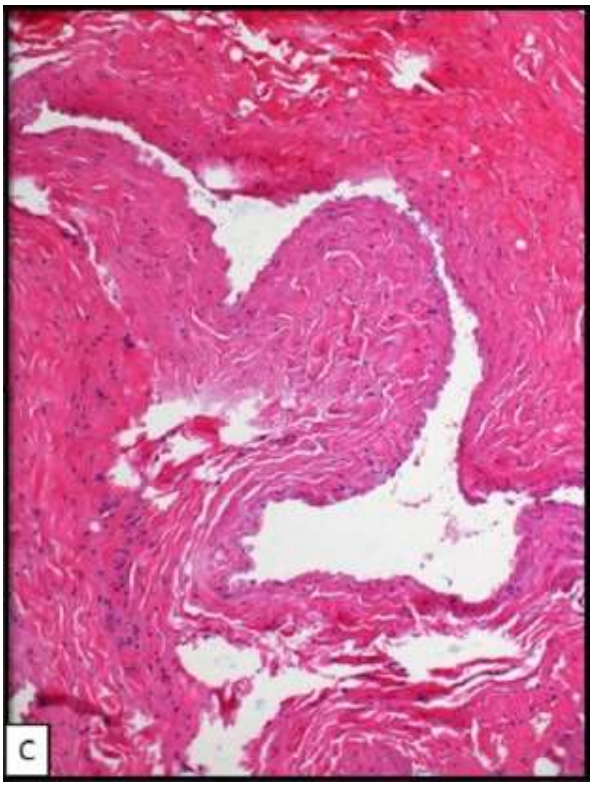

***Figure 5:* H&E stain of left IJV.**[53]

Importance in Cerebral Circulation

The carotid arteries are paramount in maintaining cerebral circulation. The ICA, through its branches, supplies critical structures within the brain, including the anterior as well as middle cerebral arteries, which are vital for cognitive and motor functions. Blood flow through the carotid arteries is meticulously regulated to ensure adequate cerebral perfusion as well as oxygenation, which are crucial for neurological health. The IJV complements this system by facilitating the drainage of deoxygenated blood and metabolic waste from the brain. The patency

and functional integrity of these vessels are essential for preventing cerebrovascular accidents such as strokes, which can result from compromised blood flow or venous drainage.[54]

Anatomical Variations Relevant to Surgical Procedures

Anatomical variations in the carotid artery and IJV can have significant implications for surgical procedures in the neck. Variations such as bifurcation anomalies, tortuosity, and differences in vessel diameter can affect surgical access and the risk of intraoperative complications. For instance, a high bifurcation of the carotid artery may necessitate adjustments in surgical technique during CEA or neck dissection. Similarly, variations in the course or position of the IJV can impact procedures such as central venous catheter placement. Understanding these variations is critical for surgeons to minimize risks and optimize surgical outcomes.[55]

In addition to these common variations, rare anatomical anomalies such as the presence of a persistent hypoglossal artery or an absent IJV can pose unique challenges. Preoperative imaging, including Doppler ultrasound and CT angiography, are invaluable tools for identifying these anomalies and planning surgical interventions accordingly. Surgeons must be adept at recognizing and adapting to these variations to prevent inadvertent injury to these critical structures during head and neck surgeries.[56]

Vascular Complications Following ND

Incidence and Types of Vascular Complications

ND, a regular surgical method in the treatment of HNCs, is associated with various vascular complications. The incidence of these complications varies widely, reported to be between 1% and 25%, depending on the extent of dissection and the individual patient's vascular anatomy.[57]

Types of complications include arterial injury, venous thrombosis, and haemorrhage. Arterial trauma, although less common, can lead to significant morbidity and may result from inadvertent injury to the carotid artery or its branches during the procedure.[58] Venous complications, particularly involving the IJV, can manifest as thrombosis or stenosis, impacting cerebral venous outflow and potentially leading to increased intracranial pressure.[59]

Immediate vs. Delayed Complications

Vascular complications following neck dissection can be categorized into immediate and delayed types. Immediate complications are those that occur intraoperatively or shortly after surgery. These include acute haemorrhage due to vessel laceration and acute thrombosis, often presenting with sudden swelling and pain in the neck. Prompt recognition as well as management are crucial to prevent further complications such as airway obstruction or stroke.[57] Delayed complications may arise days to weeks post-surgery and include

pseudoaneurysms, which are false aneurysms formed due to vessel wall injury, and delayed haemorrhage from previously unrecognized vessel trauma. These complications can be life-threatening and also may require urgent surgical or endovascular intervention.[48]

Pathophysiology of Surgical Trauma to Vessels

The pathophysiology underlying surgical trauma to vessels during neck dissection involves several mechanisms. Mechanical trauma during dissection can lead to direct vessel wall injury, causing disruption of the endothelial layer and exposure of the underlying subendothelial matrix. This exposure initiates a cascade of coagulation and inflammatory responses, resulting in thrombus formation and potential vessel occlusion.[60] Additionally, the manipulation of vessels can cause vasospasm, further compromising blood flow. In cases where the carotid artery is involved, this can lead to ischemic complications due to reduced cerebral perfusion. The inflammatory response following vessel injury can also contribute to fibrosis and stenosis, impacting long-term vessel patency.[47]

Moreover, the use of energy devices for haemostasis during surgery can exacerbate vascular injury. These devices, while effective in reducing blood loss, can cause thermal damage to vessel walls, increasing the risk of delayed haemorrhage and pseudoaneurysm formation.[61] Understanding these pathophysiological mechanisms is essential for developing strategies to minimize vascular complications during neck dissection, such as careful surgical technique,

intraoperative monitoring of vessel integrity, and the use of adjunctive measures like intraoperative Doppler ultrasound.[62]

Radiation-Induced Vascular Changes

Mechanisms of Radiation-Induced Vasculopathy

Radiation-induced vasculopathy is a complex process involving multiple mechanisms that contribute to vascular damage. The primary mechanism is the direct damage to endothelial cells lining the blood vessels. Radiation causes DNA damage, leading to cell apoptosis and the release of inflammatory cytokines. This inflammatory response results in increased vascular permeability and leukocyte adhesion, promoting a pro-thrombotic environment.[63] Additionally, radiation induces oxidative stress, which further damages endothelial cells and disrupts the balance between vasodilators and vasoconstrictors, leading to vasospasm and reduced blood flow.[64] The combination of these factors results in progressive vessel wall thickening and luminal narrowing, characteristic of radiation-induced vasculopathy.[63]

Histopathological Changes in Vessel Walls

The histopathological changes in vessel walls following radiation are well-documented and include endothelial cell loss, intimal thickening, and medial fibrosis. These changes are often accompanied by increased collagen deposition within the vessel wall, contributing to its rigidity and reduced compliance.[63]

Inflammation plays a crucial role in these changes, as the infiltration of inflammatory cells into the vessel wall leads to further tissue damage and fibrosis.[63] Additionally, radiation exposure can lead to the formation of microthrombi within the vessel lumen, exacerbating ischemic damage to downstream tissues.[64] Over time, these histopathological alterations can progress to complete vessel occlusion, significantly impairing blood flow and increasing the risk of ischemic events.[65]

Dose-Response Relationships

The relationship between radiation dose and the extent of vascular damage is dose-dependent, with higher doses leading to more severe vascular injury. Studies have shown that doses above 50 Gy are particularly damaging to the vasculature, with significant endothelial cell loss and increased risk of thrombosis.[63] The dose-response relationship is also influenced by the fractionation schedule, with hyper fractionated regimens potentially reducing the severity of vascular damage by allowing time for endothelial repair between doses.[64] However, even at lower doses, chronic exposure to radiation can lead to cumulative vascular damage, highlighting the importance of optimizing radiation therapy protocols to minimize vascular side effects.[63]

Time Course of Radiation Effects

Radiation-induced vascular changes can manifest at various time points post-exposure, with acute effects occurring within days to weeks and chronic effects developing over months to years. Acute changes are primarily characterized by endothelial cell apoptosis and increased vascular permeability, leading to oedema and inflammation.[63] These acute effects can progress to chronic changes, including vessel wall fibrosis and luminal narrowing, as the inflammatory response persists, and fibrotic tissue replaces normal vascular architecture.[64] The time course of these changes is influenced by factors such as radiation dose, fractionation schedule, and individual patient susceptibility, underscoring the need for long-term monitoring of patients who have undergone radiation therapy.[65] Understanding the progression of radiation effects is crucial for developing strategies to mitigate vascular damage and improve clinical outcomes.[63]

CS Following RT

Incidence and Prevalence Rates

They are a recognized complication following RT for HNCs. The incidence of stenosis varies depending on factors such as the RT dose and the techniques used. Studies have reported an incidence range from 30% to 50% in patients who have undergone RT, with some studies indicating even higher rates.[66] The prevalence

is influenced by the duration of follow-up, with longer follow-up periods often revealing higher rates of stenosis. A retrospective study highlighted that the cumulative incidence of carotid stenosis significantly increases over time, underscoring the importance of long-term monitoring in these patients.[67]

Risk Factors for Stenosis Development

Several risk factors predispose patients to the development of CS following RT. High radiation doses, particularly above 50 Gy, are strongly associated with increased risk. Additionally, the presence of traditional cardiovascular risk factors such as HTN, hyperlipidaemia, and smoking can exacerbate the effects of radiation on the vasculature, accelerating the development of stenosis.[68] Age is another important factor, with older patients demonstrating a higher susceptibility to radiation-induced vascular damage. The anatomical location of the tumour and the proximity of the carotid arteries to the radiation field also play a crucial role in the risk of stenosis.[69]

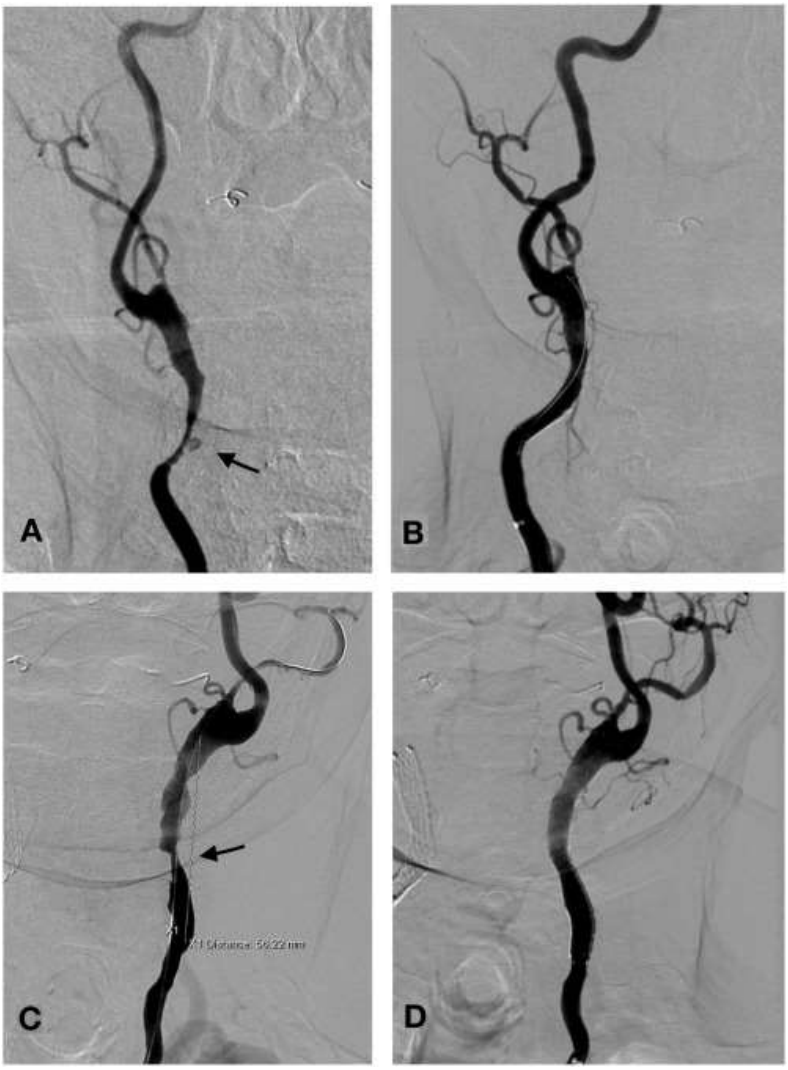

Figure 6: ***The right CCA arteriography shows severe stenosis after laryngeal cancer resection and RT***[13]

Clinical Significance and Outcomes

The development of carotid artery stenosis post-RT has significant clinical implications. Stenosis can lead to reduced cerebral perfusion, increasing the risk of ischemic cerebrovascular events such as TIAs and strokes.[66] The clinical outcomes of radiation-induced stenosis can be severe, with patients experiencing neurological deficits that impact their quality of life and functional

independence.[70] Early detection and management of stenosis are critical to prevent these adverse outcomes. Regular screening using duplex ultrasonography is recommended for early identification of carotid artery changes in patients treated with RT.[71]

Management strategies for carotid stenosis include medical therapy to control cardiovascular risk factors, CEA, or CAS, depending on the severity and progression of the stenosis.[72] The choice of intervention is influenced by the patient's overall health, the degree of stenosis, and the presence of symptoms. Recent advancements in endovascular techniques have provided less invasive options for managing stenosis, offering reduced recovery times and lower complication rates compared to traditional surgical approaches.[73]

IJV Complications

Incidence of Thrombosis and Stenosis

IJV complications, including thrombosis and stenosis, are significant concerns following neck dissection and RT in patients with HNCs. The incidence of IJV thrombosis varies in the literature, with reports suggesting it about 5% to 15% of patients undergoing these procedures.[59] Thrombosis is often associated with catheterization, which can cause endothelial damage and subsequent thrombus formation. Similarly, IJV stenosis, which involves the narrowing of the vein, can result from fibrotic changes due to surgical manipulation and radiation

exposure.[74] The prevalence of stenosis increases with higher radiation doses and longer follow-up periods, indicating a dose-response relationship.[75]

Clinical Implications

The clinical implications of IJV complications are substantial. Thrombosis can lead to venous congestion, facial oedema, and increased intracranial pressure, potentially causing neurological symptoms such as headaches and visual disturbances.[57] In severe cases, thrombosis may result in pulmonary embolism if the thrombus dislodges and travels to the lungs, posing a life-threatening risk. Stenosis, on the other hand, can impair venous drainage from the brain and head, leading to chronic venous insufficiency and contributing to the development of collateral circulation.[47] These complications can significantly impact the quality of life and may require long-term medical management.

Management Strategies

Management of IJV complications focuses on both prevention and treatment. For thrombosis, anticoagulation therapy is the mainstay of treatment, with agents such as low-molecular-weight heparin or direct oral anticoagulants being commonly used to prevent thrombus propagation and facilitate resolution.[57] In cases of catheter-related thrombosis, catheter removal is often necessary to resolve the underlying cause. For stenosis, management may involve endovascular interventions such as angioplasty or stenting to restore patency and

improve venous flow.[76] Surgical options, although less common, may be considered for severe or refractory cases.

Preventive strategies are equally important in managing IJV complications. These include careful surgical technique to minimize vascular injury, judicious use of catheters, and consideration of radiation dose and fractionation to reduce the risk of stenosis.[59] Regular monitoring through imaging modalities such as Doppler ultrasound or MR venography can aid in early detection and intervention, preventing the progression of complications.[75]

Imaging Modalities for Vascular Assessment

Ultrasonography Techniques and Parameters

Doppler ultrasonography is a vital tool for assessing the patency, size, and blood flow of neck blood vessels, particularly in patients undergoing neck dissection for head and neck cancer. This non-invasive imaging technique provides crucial insights into vascular conditions post-surgery, aiding in the management and prognostication of patient outcomes.

Probe Used

The choice of probe in Doppler ultrasonography is critical for obtaining accurate measurements of the neck blood vessels. Typically, a high-frequency linear array transducer is employed, ranging from 7.5 to 12 MHz. This frequency range offers

excellent resolution, crucial for visualizing superficial structures like the CCA and IJV. The linear probe allows for high-resolution imaging, which is essential for detecting subtle changes in blood vessel walls and lumen size, as well as for assessing blood flow dynamics.[77]

Additionally, specialized Doppler probes with capabilities for colour and spectral Doppler imaging are used to evaluate blood flow velocities and patterns. These probes provide both B-mode imaging, which visualizes the structure of the vessels, and Doppler mode, which assesses the direction and speed of blood flow, thereby offering a comprehensive evaluation of the vascular system.

Procedure

Doppler ultrasonography of the neck involves a systematic approach to ensure accurate and reproducible results. The procedure begins with the patient in a supine position with the neck slightly extended and turned away from the side being examined. This positioning optimizes access to the carotid artery and IJV.

The examination starts with B-mode imaging to assess the structural integrity of the vessels. The sonographer evaluates the vessel walls for thickness, plaques, or irregularities that may indicate atherosclerotic changes. Following this, color Doppler imaging is conducted to visualize blood flow patterns, detecting any turbulence or flow disturbances that could suggest stenosis or other vascular abnormalities.[78]

Spectral Doppler analysis is then performed to measure blood flow velocities within the vessels. This involves placing the Doppler cursor within the lumen of the vessel and recording the waveform patterns. The peak systolic and end-diastolic velocities are key parameters measured during this phase, providing insights into the hemodynamic status of the vessels.

Throughout the procedure, care is taken to maintain consistent probe positioning and pressure to avoid artifacts that could affect the accuracy of the readings. The entire examination is typically completed within 15 to 30 minutes, depending on the complexity of the vascular assessment required.

Key parameters assessed include the IMT, PSV, and EDV, which are crucial for evaluating the degree of stenosis.[38]

- **PSV:** This parameter measures the maximum speed of blood flow during cardiac systole. Elevated PSV values can indicate stenosis, while reduced values may suggest occlusion or other flow-restricting conditions. Values <125 cm/s indicate normal flow dynamics without significant stenosis. The intermediate range (125-230 cm/s) correlates with approximately 50-69% stenosis, while measurements >230 cm/s strongly suggest hemodynamically significant stenosis (≥70%). The "Undetectable" category indicates complete or near-complete occlusion. These velocity thresholds demonstrate high correlation with angiographic findings and are

considered the most reliable sonographic indicators of stenosis severity in standard clinical practice.[79]

- **EDV:** This measures the blood flow speed during diastole. Changes in EDV can provide additional information about peripheral resistance and vessel patency. Normal values (<40 cm/s) indicate preserved diastolic flow without significant resistance. Moderate elevation (40-100 cm/s) suggests developing stenosis with increasing resistance, while severe elevation (>100 cm/s) typically accompanies critical stenosis (>70%) with substantially increased resistance. EDV serves as an important confirmatory parameter when PSV values are borderline or ambiguous, enhancing diagnostic accuracy. [79]

- **RI:** Calculated as (PSV - EDV)/PSV, the RI is a measure of vascular resistance. Increased RI values can indicate downstream resistance, often associated with vascular narrowing or pathological changes.

- **Lumen Diameter and Cross-Sectional Area:** These are assessed using B-mode imaging to evaluate the size and structural integrity of the vessels. Changes in lumen size can reflect pathological enlargement or narrowing.

- **Flow Patterns:** Color Doppler imaging assesses the direction and uniformity of blood flow. Turbulent flow can indicate stenosis or vessel irregularities.

- **Plaque Characteristics:** B-mode imaging is used to assess plaque morphology and echogenicity, which are important for evaluating the risk of embolic events.

These parameters collectively provide a comprehensive assessment of the vascular status, aiding clinicians in making informed decisions regarding patient management post-neck dissection.[80] Color Doppler ultrasound provides real-time visualization of blood flow patterns, aiding in the detection of turbulence indicative of stenosis or occlusion. The sensitivity of ultrasound in detecting significant stenosis makes it an invaluable first-line tool in vascular assessment.[81]

CTA and MRA

They are advanced imaging modalities that offer detailed visualization of vascular anatomy. CTA involves the use of contrast material and X-ray technology to create cross-sectional images of blood vessels. They are very helpful in measuring the extent as well as severity of stenosis, as well as identifying calcifications within the vessel walls.[62]

MR angiography, on the other hand, employs magnetic fields and radio waves to produce images without ionizing radiation. MRA is advantageous in patients with renal impairment or those requiring repeated imaging, as it avoids nephrotoxic contrast agents used in CTA.[40] Both modalities provide comprehensive insights into vascular health, allowing for accurate diagnosis and treatment planning.

Comparison of Different Imaging Modalities

When comparing these imaging modalities, each has its unique strengths and limitations. Ultrasonography is cost-effective, widely available, and devoid of radiation, making it suitable for routine screening and follow-up.[38] However, its accuracy can be operator-dependent, and it may be limited in obese patients or those with calcified vessels. CT angiography provides high-resolution images and is excellent for evaluating complex vascular anatomy and acute pathologies. Nonetheless, exposure to radiation and contrast-induced nephropathy are potential drawbacks.[62] MR angiography, while avoiding radiation, can be time-consuming and may be contraindicated in patients with certain implants or claustrophobia.[40]

The choice of imaging modality often depends on the clinical context, patient-specific factors, and the level of detail required. For instance, in acute settings where rapid assessment is crucial, CTA is often preferred due to its speed and clarity. In contrast, MRA is ideal for patients requiring frequent evaluations or those with contraindications to iodinated contrast agents. Ultimately, the integration of these imaging techniques allows for a comprehensive approach to vascular assessment, optimizing diagnostic accuracy and enhancing patient outcomes.[82]

Hemodynamic Parameters in Vascular Assessment

PSV and EDV

In vascular assessment, PSV and EDV are critical parameters measured using Doppler ultrasonography. PSV refers to the maximum blood flow velocity during heart contraction, while EDV represents the flow velocity during heart relaxation. These measurements are instrumental in evaluating the haemodynamics of blood flow within vessels, such as the carotid arteries. High PSV values often indicate stenosis, as the blood velocity increases to overcome narrowing. Conversely, reductions in EDV can suggest downstream resistance or reduced compliance in peripheral vessels.[83] Together, these velocities provide a comprehensive picture of vascular health and are used to stratify the risk of cardiovascular events in patients with potential arterial disease.[84]

IMT Measurements

IMT is a well-established marker of atherosclerosis and is measured using B-mode ultrasonography. This parameter reflects the combined thickness of the intima and media layers of the arterial wall. Increased IMT is associated with a higher risk of CVDs, including stroke and myocardial infarction, making it a valuable predictor of systemic atherosclerosis.[38] IMT measurement is not only non-invasive but also repeatable, allowing for regular monitoring of disease progression or regression in response to treatment. The standardization of IMT

measurement protocols has enhanced its utility in both clinical and research settings, facilitating comparative studies across different populations.[81]

Velocity Ratios and Their Significance

Velocity ratios, particularly the ratio of PSV to EDV, are crucial in assessing the severity of vascular stenosis. A higher velocity ratio suggests a significant narrowing, as the blood must accelerate to maintain flow across a constricted segment. These ratios help differentiate between mild, moderate, and severe stenosis, guiding clinical decision-making regarding intervention.[38] Additionally, the ratio of velocities at different points along the vessel, such as the CCA compared to the ICA, can provide insights into the location and extent of the obstruction.[40] Understanding these ratios is essential for accurately interpreting Doppler ultrasound findings and correlating them with clinical symptoms.

ICA/CCA PSV Ratio: Values <2.0 indicate normal flow proportionality without significant stenosis. Ratios between 2.0-4.0 correspond to moderate stenosis (50-69%), while ratios >4.0 strongly suggest severe stenosis (≥70%). This parameter demonstrates particular utility in patients with globally increased or decreased velocities due to cardiac output variations, significantly improving diagnostic accuracy compared to absolute velocity measurements alone. [79]

Management of Radiation-Induced Carotid Stenosis

Surveillance Protocols

Surveillance of radiation-induced carotid stenosis is crucial for early detection and management. Regular monitoring using Doppler ultrasound is recommended for patients who have undergone RT for HNCs, as this non-invasive method provides detailed information on blood flow and vessel wall integrity.[67] The frequency of surveillance depends on the patient's risk factors and the severity of stenosis detected during initial assessments. High-risk patients, such as those with significant cardiovascular risk factors or higher radiation doses, may require more frequent evaluations. Advanced imaging modalities like CT angiography and MR angiography can be utilized for more detailed assessments if ultrasound findings indicate significant stenosis.[66]

Indications for Intervention

Intervention is indicated in patients with severe CS, typically defined as a narrowing of 70% or greater, or in those who are symptomatic, experiencing TIAs or strokes.[70] The decision to intervene is influenced by factors such as the patient's overall health status, the presence of comorbidities, and life expectancy. In asymptomatic patients, the degree of stenosis and the rate of progression are critical considerations. Current guidelines suggest a more conservative approach

in asymptomatic patients with less than 70% stenosis, emphasizing the importance of individualized care plans.[67]

Medical Management vs. Surgical Options

Medical management is the cornerstone of treatment for radiation-induced carotid stenosis, focusing on controlling risk factors such as HTN, hyperlipidaemia, and diabetes. Antiplatelet therapy, typically with aspirin or clopidogrel, is recommended to reduce the risk of thromboembolic events.[85] Lifestyle modifications, including smoking cessation and regular physical activity, are also essential components of medical management.[66]

Surgical options, including CEA and CAS, are considered in patients with significant stenosis or those who are symptomatic. CEA is the traditional surgical approach and involves the removal of atherosclerotic plaque from the carotid artery. However, radiation-induced changes in the neck, such as fibrosis, can complicate surgery, increasing the risk of complications.[86] In these cases, CAS may be a preferable option, offering a less invasive alternative with comparable outcomes.[67]

The choice between CEA and CAS depends on several factors, including the patient's anatomy, the severity of stenosis, and the presence of prior neck surgeries or radiation. Recent studies suggest that CAS may be associated with lower perioperative complication rates, particularly in patients with challenging

anatomical considerations due to prior radiation.[86] Nonetheless, both procedures require careful patient selection and a multidisciplinary approach to optimize outcomes.

QOL Considerations

Impact of Vascular Complications on Patient Outcomes

Vascular complications, such as carotid artery stenosis, significantly impact the quality of life in patients with HNCs. These complications can lead to ischemic events, including strokes and TIAs, which are associated with increased morbidity and mortality.[66] The presence of vascular complications often necessitates ongoing medical management, frequent monitoring, and potential surgical interventions, which can be burdensome for patients and affect their overall well-being. Additionally, the anxiety and stress associated with the risk of ischemic events can further compound the negative impacts on quality of life, highlighting the importance of effective management strategies.[70]

Functional Implications of Vascular Compromise

The functional implications of vascular compromise in head and neck cancer patients are profound. Compromised blood flow resulting from stenosis or occlusion can lead to deficits in neurological function, including cognitive impairment, speech difficulties, and motor skill deterioration.[87] These functional deficits can interfere with daily activities and reduce independence, necessitating

rehabilitation and support services. Moreover, the physical challenges posed by these complications often require adjustments in lifestyle and occupational activities, further affecting patients' social and economic well-being.[88]

Beyond physical impairments, vascular compromise can also have psychosocial consequences. Patients may experience changes in self-image and psychological distress due to their altered physical abilities and reliance on others for assistance. This can lead to social isolation and depression, further diminishing quality of life.[89] Effective communication between healthcare providers, patients, and caregivers is essential to address these challenges, providing holistic care that encompasses both medical and psychosocial needs.

Cardiovascular Risks in Head and Neck Cancer Survivors

Long-term Cardiovascular Morbidity

Head and neck cancer (HNC) survivors face a heightened risk of long-term cardiovascular morbidity, a concern that is increasingly recognized in survivorship care. CVD is one of the leading non-cancer causes of morbidity and mortality in these patients, exacerbated by factors such as RT which can lead to vascular damage and increased atherosclerosis.[90] Studies show that the risk of ischemic heart disease and other cardiovascular events remains elevated long

after treatment, necessitating ongoing monitoring and management of cardiovascular health in these individuals.[91]

Risk Prediction Models

Risk prediction models are crucial tools in identifying HNC survivors at high risk for cardiovascular complications. These models integrate various risk factors, including traditional cardiovascular risk markers and treatment-specific factors like radiation dose and treatment modality.[68] The development of precise predictive models has been informed by large cohort studies that elucidate the complex interactions between cancer treatment and cardiovascular risk. These models are essential for tailoring preventive strategies and interventions, ensuring that high-risk individuals receive appropriate cardiovascular care.[35]

Preventive Strategies

Preventive strategies play a pivotal role in mitigating cardiovascular risks among HNC survivors. A comprehensive approach includes lifestyle modifications, such as smoking cessation, dietary improvements, and regular physical activity, which are effective in reducing cardiovascular risk.[35] Pharmacological interventions, including the use of statins and antihypertensives, are also recommended to manage dyslipidaemia and HTN, common in this population.[92]

Furthermore, the integration of cardioprotective strategies during cancer treatment, such as minimizing radiation exposure and employing advanced RT

techniques, can reduce the risk of cardiovascular damage.[91] Regular cardiovascular screening and monitoring post-treatment are essential components of survivorship care, allowing for early detection and management of cardiovascular issues. By implementing these preventive measures, healthcare providers can significantly improve the long-term health outcomes of HNC survivors, reducing the burden of CVD in this vulnerable population.

Gaps in Current Knowledge and Future Research Directions

Limitations of Existing Studies

The current body of research on CCA and IJV patency following neck dissection and postoperative RT in head and neck cancer is limited by several factors. Many studies suffer from small sample sizes, which restricts the generalizability of their findings.[93] Additionally, there is often a lack of long-term follow-up, which is crucial for understanding the chronic effects of treatment on vascular structures.[94] Further, heterogeneity in study designs and methodologies complicates the synthesis of existing data, making it difficult to draw definitive conclusions.[95]

Areas Requiring Further Investigation

Firstly, there is a need for large-scale, multicentre studies with standardized protocols to better assess the long-term vascular outcomes in this patient population.[93] Additionally, exploring the molecular mechanisms underlying radiation-induced vascular damage could provide insights into potential

therapeutic targets.[96] The role of genetic predispositions in vascular complications post-treatment also warrants further investigation, as this could lead to personalized treatment approaches.

Emerging Technologies and Approaches

Emerging technologies hold promises for advancing the assessment and management of vascular complications in head and neck cancer patients. Innovations in imaging techniques, such as advanced MRI as well as CT modalities, offer more detailed visualization of vascular structures, facilitating early detection of changes in vessel patency.[97] In the surgical domain, the development of minimally invasive techniques and robotic-assisted surgeries may reduce the risk of vascular injury during neck dissections.[98] Furthermore, the integration of artificial intelligence in diagnostic imaging could enhance the accuracy of vascular assessments, enabling more precise monitoring and intervention strategies.[99]

MATERIALS AND METHODS

Study Design and Study Setting

This investigation was designed as a prospective observational study to systematically evaluate the vascular sequelae of multimodality treatment in head and neck cancer patients. The study was conducted at the Department of Otorhinolaryngology in R.L. Jalappa Hospital, Tamaka, Kolar, a tertiary care academic medical center with specialized head and neck oncology services.

Study Period

The study was conducted over a 17-month period from May 2023 to October 2024, encompassing patient recruitment, sequential vascular assessments, and data analysis.

Ethics Committee Approval

Prior to participant enrolment, the study protocol received comprehensive review and approval from the Institutional Ethics Committee (IEC) of R.L. Jalappa Hospital and Research Centre. Written informed consent was obtained from all participants following detailed explanation of the study procedures, potential risks, benefits, and confidentiality provisions. Participants were assured of their right to withdraw from the study at any point without affecting their standard clinical care.

Inclusion Criteria

The study enrolled patients meeting the following criteria:

1. Age between 35-85 years
2. Histopathologically confirmed diagnosis of head and neck malignancy
3. Clinical staging requiring surgical intervention inclusive of neck dissection
4. Adjuvant RT indicated as per multidisciplinary tumour board recommendation
5. Willingness to participate and ability to provide informed consent
6. Availability for follow-up assessments as per study protocol

Exclusion Criteria

Patients were excluded from participation based on the following criteria:

1. Previous history of radiation therapy to the neck region
2. Pre-existing cervical injury or laceration
3. Severe kyphoscoliosis affecting neck anatomy and vascular assessment
4. Pre-existing carotid artery stenosis exceeding 50% on initial screening
5. Known coagulopathy or anticoagulant therapy that could not be temporarily discontinued
6. Medical contraindications to surgical intervention or RT

7. Inability to comply with follow-up assessment protocol

Sample Size Estimation

The sample size was calculated using Cochran's formula for estimating population proportion with specified precision:

$$n = Z^2 \times p \times (1-p) / d^2$$

Where:

- Z = standard normal variate (1.96 for 95% confidence interval)
- p = expected proportion with the outcome of interest
- d = relative precision

The primary outcome parameter for sample size calculation was the proportion of patients expected to develop carotid artery lumen stenosis following neck radiation. Based on the study by Akhavan et al. (2021), who reported that 51.1% of 45 Iranian head and neck cancer patients developed mild CS (<50%) following neck radiation, this prevalence value was adopted for calculation.[79] Assuming a 95% confidence interval ($\alpha = 0.05$) and relative precision of 25%, the minimum required sample size was calculated as:

$n = (1.96)^2 \times 0.511 \times (1-0.511) / (0.25 \times 0.511)^2$ $n = 3.8416 \times 0.511 \times 0.489 / 0.016269$ $n = 59.36$

Based on this calculation, a minimum sample size of 59 participants was required. The study enrolled 60 patients to account for potential attrition and ensure adequate statistical power.

Sampling Method

Consecutive sampling was employed to enrol eligible patients presenting to the Department of Otorhinolaryngology at R.L. Jalappa Hospital during the study period. All patients diagnosed with head and neck malignancy requiring neck dissection and adjuvant RT who met inclusion criteria were approached for participation.

Data Collection Procedure

All patients underwent comprehensive clinical evaluation by a multidisciplinary tumour board comprising specialists in head and neck surgery, radiation oncology, medical oncology, radiology, and pathology. Baseline demographic data (age, gender) and relevant clinical information (comorbidities, tumour characteristics) were documented using a standardized data collection form.

Vascular Assessment Protocol

Vascular assessments were performed by experienced radiologists using a standardized Doppler ultrasonography protocol at three sequential timepoints:

1. Preoperative (baseline): Within one week prior to surgical intervention
2. Post-operative: Two weeks following neck dissection

3. Post-RT: Four weeks following completion of adjuvant radiation therapy

Examinations were conducted using a high-resolution colour Doppler ultrasound system with a 7.5-12 MHz linear array transducer. Patients were positioned supine with the head slightly extended and rotated away from the side being examined. All measurements were performed bilaterally to establish comparative parameters.

The vascular assessment included the following quantitative parameters:

1. CCA PSV: Measured in centimetres per second (cm/s) with normal values defined as <125 cm/s, intermediate as 125-230 cm/s (corresponding to approximately 50-69% stenosis), and elevated as >230 cm/s (suggesting ≥70% stenosis).
2. ICA PSV: Measured in cm/s with normal values defined as <125 cm/s, intermediate as 125-230 cm/s, and elevated as >230 cm/s.
3. ICA EDV: Measured in cm/s with normal values defined as <40 cm/s, intermediate as 40-100 cm/s, and elevated as >100 cm/s.
4. ICA/CCA PSV Ratio: Calculated by dividing ICA PSV by CCA PSV, with normal values defined as <2.0, intermediate as 2.0-4.0 (corresponding to 50-69% stenosis), and elevated as >4.0 (suggesting ≥70% stenosis).
5. CIMT: Measured in millimetres at the far wall of the distal CCA approximately 1 cm proximal to the carotid bulb. Measurements were

obtained during end-diastole from three consecutive cardiac cycles and averaged.

Surgical and RT Protocols

All patients underwent MRND performed by experienced head and neck surgeons according to standardized protocols. The laterality of the procedure (left or right) was documented. Following adequate wound healing, patients proceeded to adjuvant therapy based on histopathological findings and multidisciplinary tumor board recommendations.

RT was delivered using linear accelerator-based IMRT or VMAT techniques. The prescribed radiation dose, fractionation schedule, and concurrent CT (if administered) were documented for each patient. Standard fractionation typically consisted of 2 Gy per fraction delivered once daily, five days per week, to a total dose range of 56-66 Gy depending on tumor characteristics and surgical margins.

Data Analysis

Statistical analysis was performed using SPSS version 26.0. Continuous variables were presented as mean plus/minus standard deviation while categorical variables as frequencies along with percentages. Non-parametric Wilcoxon signed-rank tests were performed for paired comparisons of vascular parameters across the

three assessment timepoints (preoperative vs. post-operative, post-operative vs. post-RT, and preoperative vs. post-RT) due to non-normal distribution of the data. McNemar's test was employed for paired comparison of categorical outcomes. For all analyses, a p-value <0.05 was considered statistically significant.

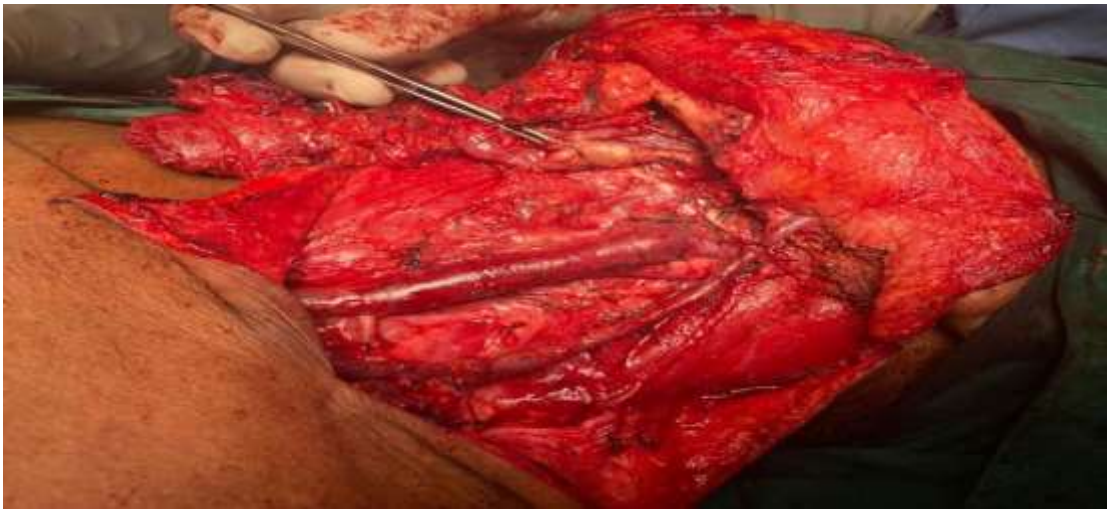

Figure 7: Figure showing the great vessels of neck

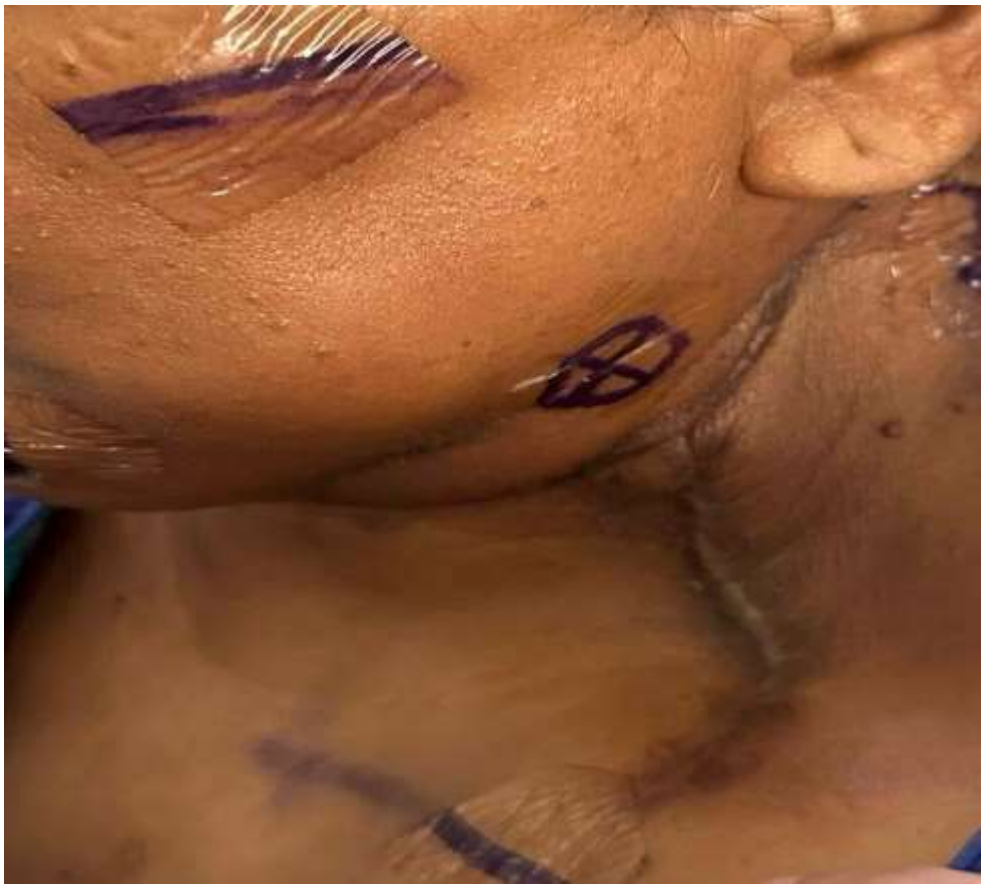

Figure 8: Figure showing skin marking during radiotherapy given to patients

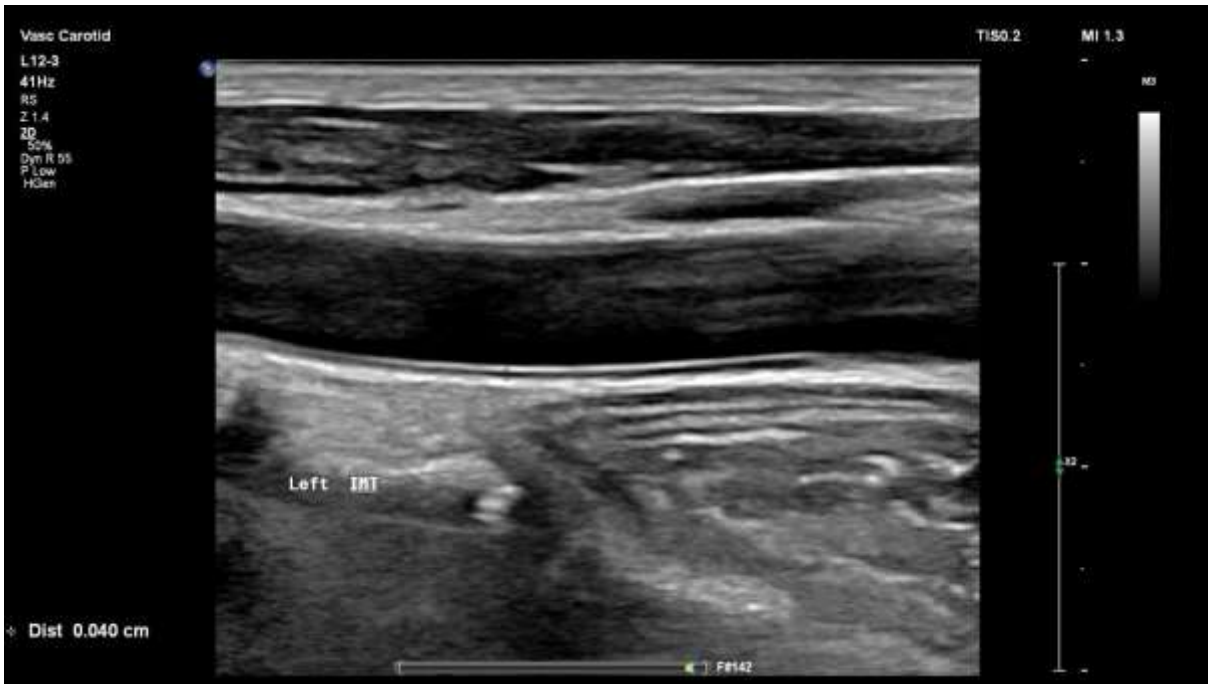

Figure 9: Figure showing preoperative normal intima medial thickness of CCA

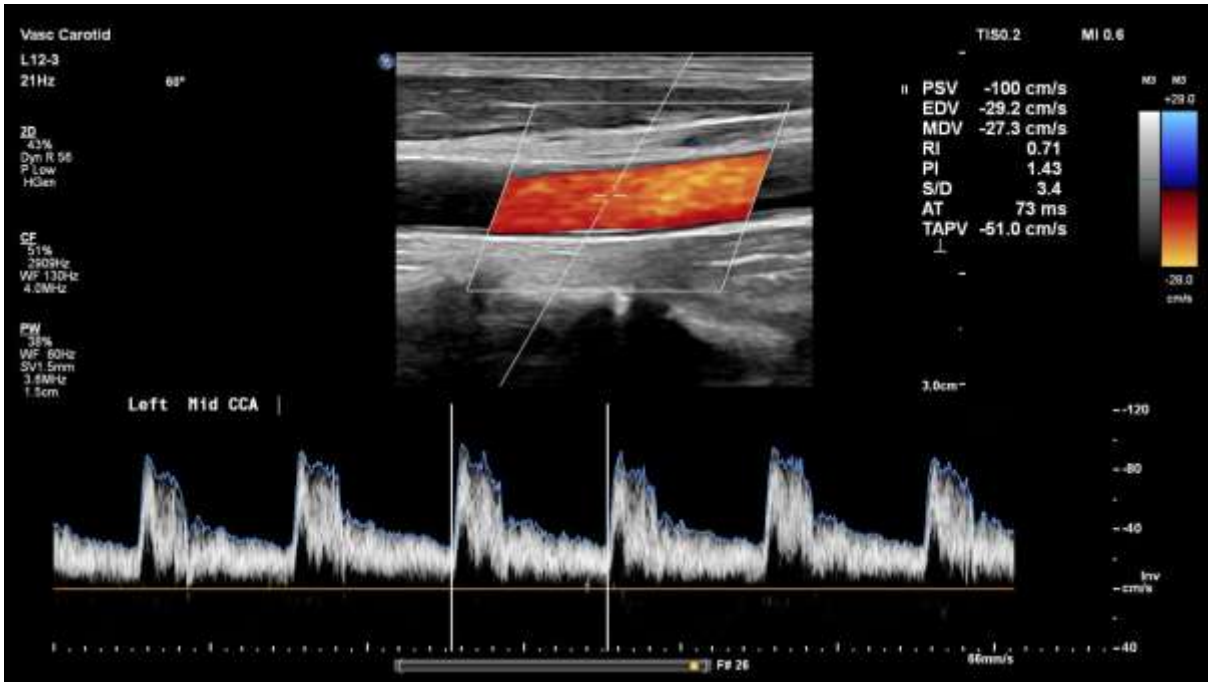

Figure 10: Figure showing preoperative normal peak systolic velocity and end diastolic velocity

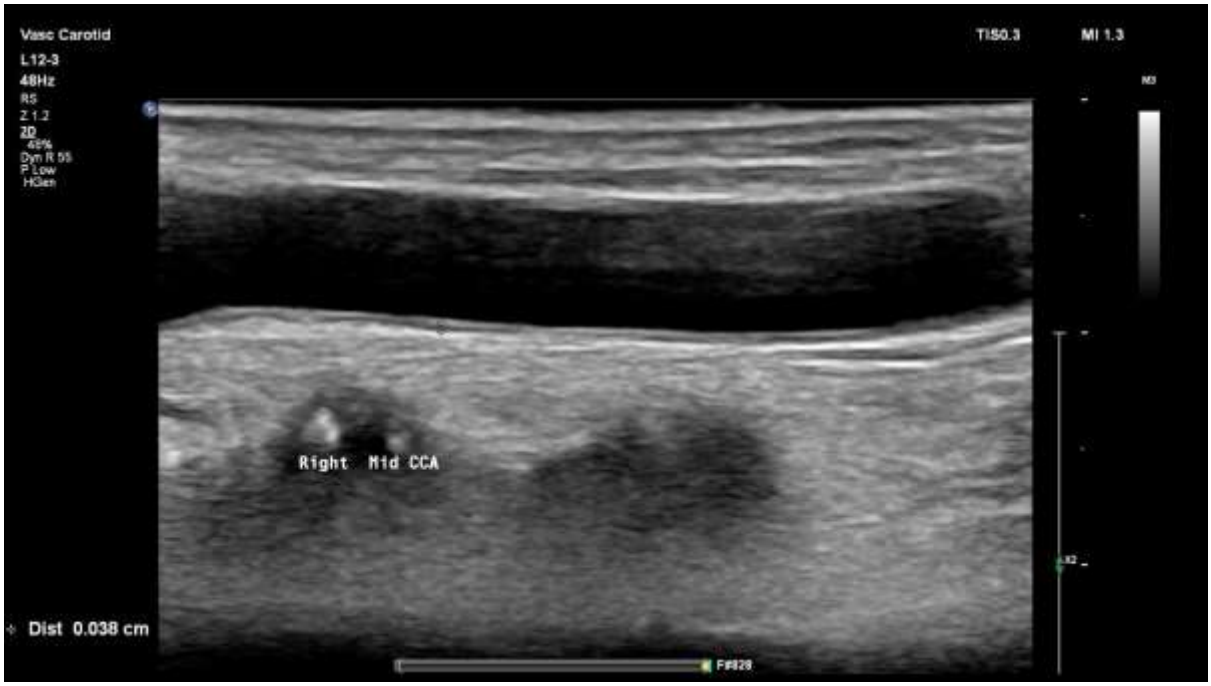

Figure 11: Figure showing post radiotherapy - intima medial thickness of CCA

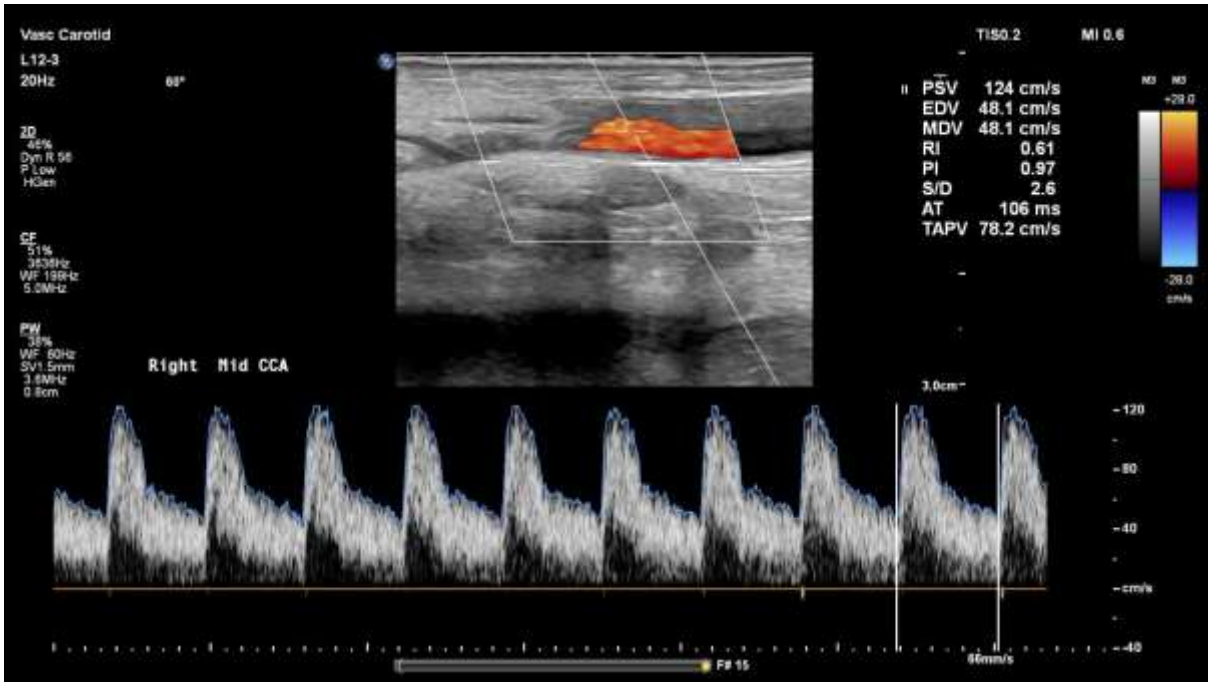

Figure 12: Figure showing the peak systolic velocity and end diastolic volume for post Radiotherapy patient

RESULTS

Table 1: Age data

Age in years	
Mean	55.48
Median	54.5
Standard deviation	11.8
Minimum	35
Maximum	83
Range	49

This table presents the central tendency and dispersion metrics of the age distribution among the study cohort (N=60) undergoing neck dissection for head and neck malignancies. The mean age was 55.48 years (SD=11.8), with a median of 54.5 years, indicating a relatively normal distribution.

Table 2: Gender Distribution

Gender	Frequency	Percent
Female	43	71.7
Male	17	28.3
Total	60	100.0

This table delineates the gender composition of the study population, revealing a predominance of female patients (n=43, 71.7%) compared to male patients (n=17, 28.3%).

Figure 13: Gender Distribution

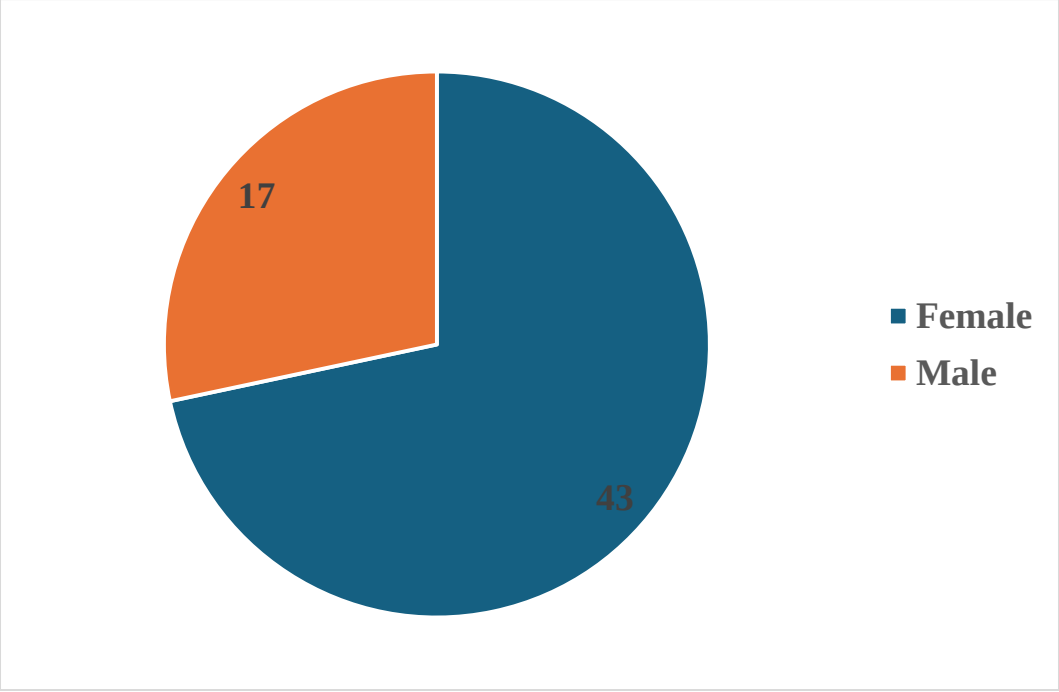

Table 3: Comorbidity Profile of Study Participants

Comorbidities	Frequency	Percent
DM	8	13.3
HTN	6	10
Hypothyroidism	1	1.7
Hyperlipidaemia	12	20
Nil	33	55
Total	60	100.0

The majority of patients (n=33, 55%) presented without significant comorbidities. Among those with comorbidities, hyperlipidemia was most prevalent (n=12, 20%), followed by DM (n=8, 13.3%) and HTN (n=6, 10%). Hypothyroidism was observed in a single patient (1.7%).

Table 4: Complications Correlation Table

Complication	**Hyperlipidaemia (n=12)**	**DM (n=8)**	**HTN (n=6)**
Wound gaping	4	3	2
Flap necrosis	2	1	1
Vessel blowout	0	0	0
Other complications	3	2	2

In the hyperlipidemia group (n=12), wound gaping was the most frequent complication (observed in 33.3% of patients), followed by flap necrosis (16.7%). In the DM group (n=8), wound gaping was also prominent (37.5%), and flap necrosis occurred in 12.5% of patients. HTN patients (n=6) showed a slightly lower incidence of wound gaping (33.3%) and flap necrosis (16.7%). Notably, no vessel blowout events were recorded across all groups, indicating low vascular complication risk in this cohort. The 'other complications' category, comprising issues like flap discoloration, fistula, DVT, edema, pneumonia, and salivary leak, was relatively comparable across comorbidity groups (about 16–25%). These findings suggest that while wound gaping and minor flap-related complications are common, major complications like vessel blowout are rare, irrespective of comorbid status.

Table 5: Distribution of MRND by Laterality

Surgery performed	Frequency	Percent
MRND (Left)	35	58.3
MRND (Right)	25	41.7
Total	60	100.0

This table documents the surgical approach utilized, specifically the laterality of MRND procedures. Left-sided MRND was performed in the majority of cases (n=35, 58.3%), while right-sided MRND was performed in the remainder (n=25, 41.7%).

Figure 14: Distribution of MRND by Laterality

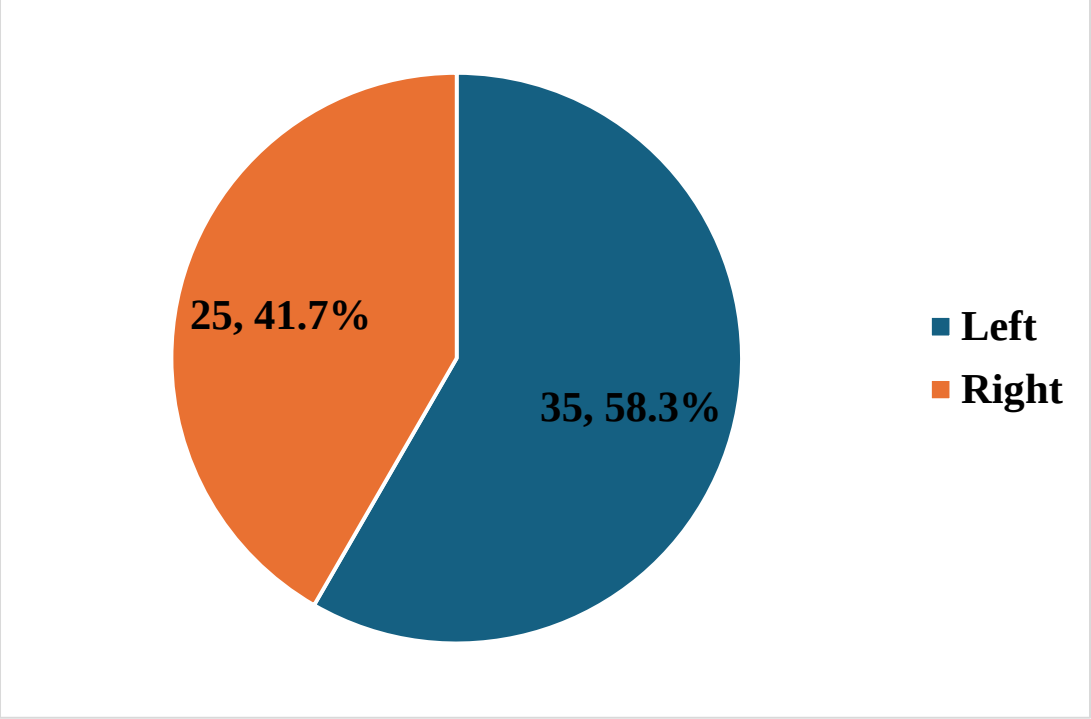

Table 6: Distribution of Adjuvant Therapy Modalities

Adjuvant therapy	Frequency	Percent
CT+RT	4	6.7
RT	56	93.3
Total	60	100.0

The vast majority of patients (n=56, 93.3%) received RT alone, while a small subset (n=4, 6.7%) received combined chemoradiotherapy (CT+RT).

Figure 15: Distribution of Adjuvant Therapy Modalities

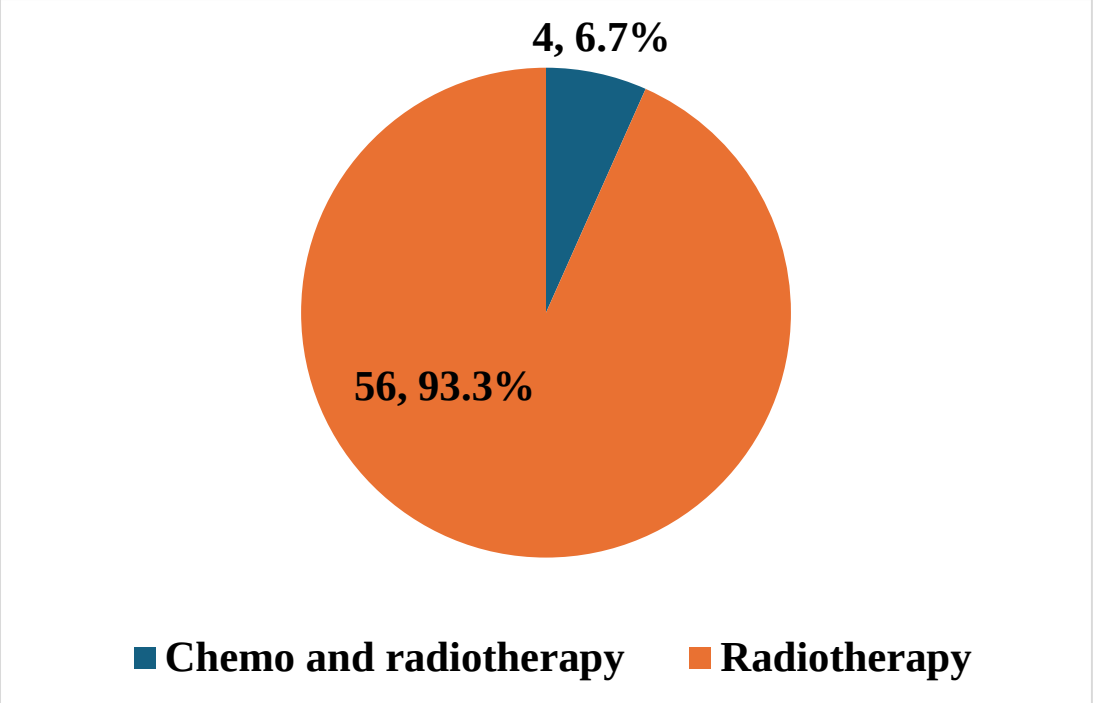

Table 7: Distribution of RT Dose Administration

Dose of RT	Frequency	Percent
52Gy	1	1.7
56GY	11	18.3
60Gy	8	13.3
64Gy	25	41.7
66Gy	15	25
Total	60	100.0

The majority of patients (n=25, 41.7%) received 64 Gy, followed by 66 Gy (n=15, 25.0%), 56 Gy (n=11, 18.3%), and 60 Gy (n=8, 13.3%).

Figure 16: Distribution of RT Dose Administration

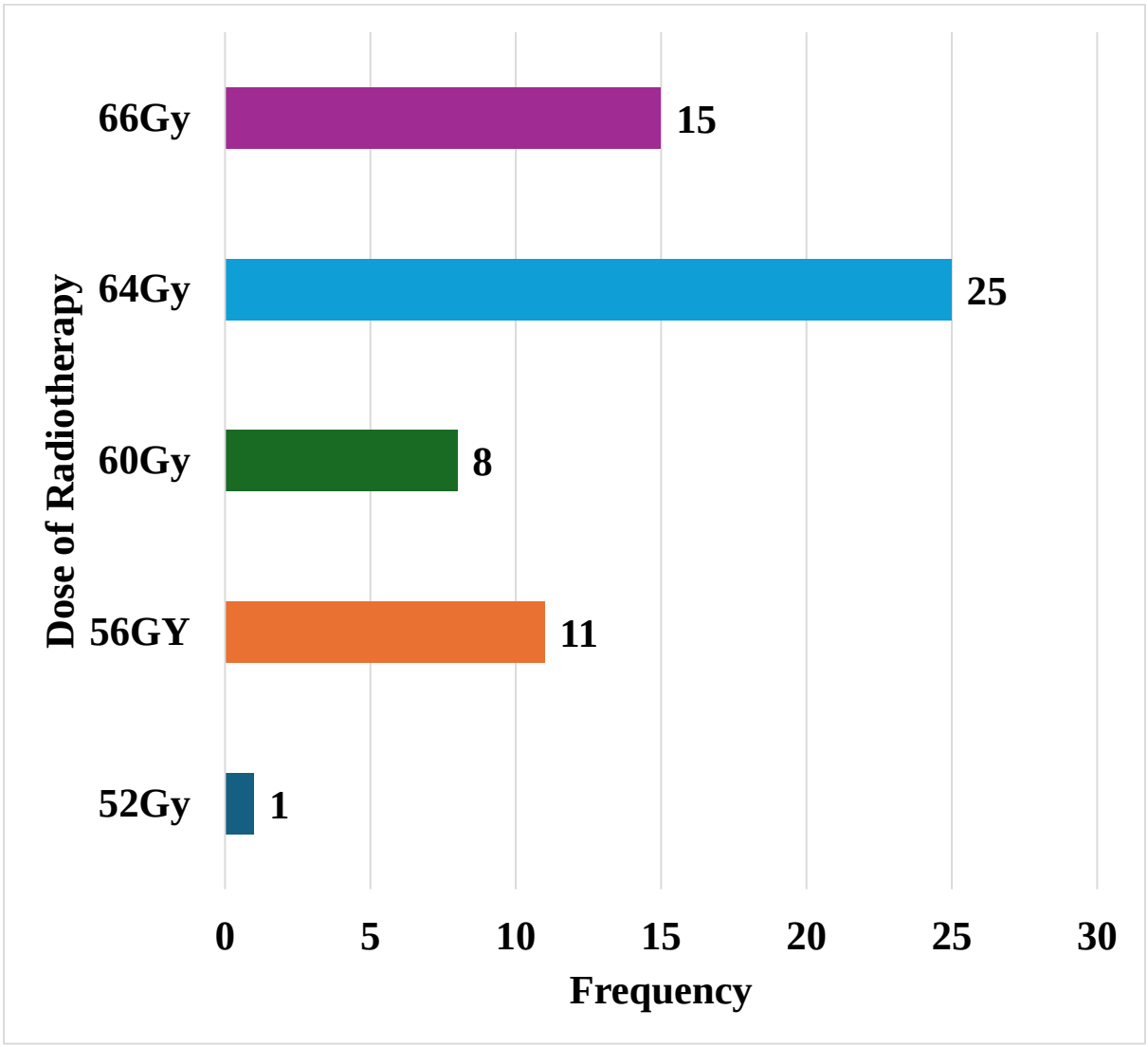

Table 8: Distribution of RT Fractionation Schedules

Number of fractions of RT	Frequency	Percent
26	1	1.7
28	11	18.3
30	8	13.3
32	27	45.0
33	13	21.7
Total	60	100.0

The most common fractionation schedule involved 32 fractions (n=27, 45.0%), followed by 33 fractions (n=13, 21.7%), 28 fractions (n=11, 18.3%), 30 fractions (n=8, 13.3%), and 26 fractions (n=1, 1.7%). This distribution represents standard fractionation approaches in head and neck cancer RT, with the predominant 32-fraction schedule corresponding to approximately 2 Gy per fraction.

Figure 17: Distribution of RT Fractionation Schedules

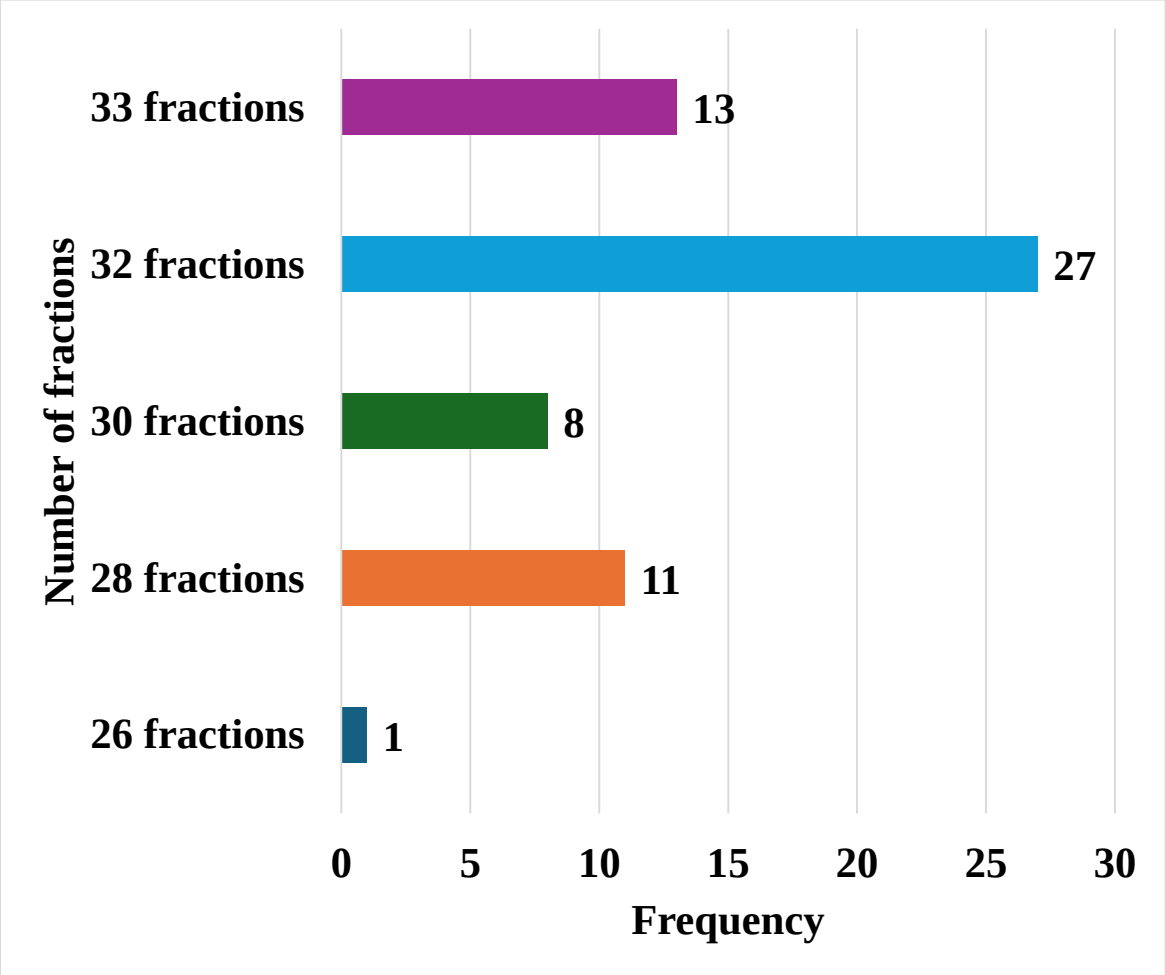

Table 9: Longitudinal Assessment of CCA PSV Following ND and Adjuvant RT

Measures of CCA-PSV (cm/s)	Pre-operative	Post-operative	Post RT
Mean	93.73	94.23	98.20
Std. Deviation	11.29	9.79	8.58

This table presents quantitative hemodynamic analysis of CCA-PSV measured sequentially across three critical treatment timepoints in head and neck cancer patients (N=60) undergoing multimodality therapy. Pre-operatively, mean CCA-PSV was 93.73 cm/s (SD=11.29), establishing baseline flow dynamics. Following neck dissection, a modest increase to 94.23 cm/s (SD=9.79) was observed, representing a marginal increment of 0.53%. Post-RT measurements revealed further progressive elevation to 98.20 cm/s (SD=8.58), constituting a cumulative increase of 4.77% from baseline. Notably, the progressive reduction in standard deviation (11.29→9.79→8.58) suggests increasing homogeneity in vascular response across the cohort. Despite this incremental trend in absolute CCA-PSV values, all measurements remained substantially below the threshold for hemodynamically significant stenosis (125 cm/s), with 98.3% of patients maintaining normal velocity parameters post-RT. This finding demonstrates that

while RT may induce subtle alterations in carotid flow dynamics, these changes remain predominantly within physiological parameters, resulting in negligible clinically significant stenosis rates in the immediate post-treatment period. The preserved functional hemodynamics despite modest velocity increases aligns with the overall vascular safety profile observed in this prospective cohort.

Table 10: Pre-operative Doppler Ultrasound Parameters of Carotid Vasculature

Pre-operative parameters measured	**Category**	**Frequency**	**Percent**
CCA PSV (cm/s)	<125	58	96.7
	125 to 230	2	3.3
ICA PSV (cm/s)	<125	60	100.0
ICA EDV (cm/s)	<40	60	100.0
PSV Ratio	<2	60	100.0

CCA PSV was predominantly within normal limits (<125 cm/s) in 96.7% of patients (n=58), with only 3.3% (n=2) showing borderline elevation (125-230 cm/s). All patients demonstrated normal ICA PSV <125 cm/s), normal ICA end-

diastolic velocity (ICA EDV <40 cm/s), and normal PSV ratios (<2). These baseline measures establish the pre-intervention vascular status, confirming absence of significant pre-existing stenosis in the study population.

Table 11: Post-operative Doppler Ultrasound Parameters of Carotid Vasculature

Post-operative parameters measured	Category	Frequency	Percent
CCA PSV (cm/s)	<125	59	98.3
	125 to 230	1	1.7
ICA PSV (cm/s)	<125	60	100.0
ICA EDV (cm/s)	<40	60	100.0
PSV Ratio	<2	60	100.0

This table documents vascular parameters measured two weeks following neck dissection. Post-operatively, CCA PSV remained predominantly within normal limits (<125 cm/s) in 98.3% of patients (n=59), with only 1.7% (n=1) showing borderline elevation (125-230 cm/s). All patients maintained normal ICA PSV, normal ICA EDV, and normal PSV ratios. These findings suggest minimal immediate impact of surgical intervention on carotid vascular flow dynamics,

with a slight improvement in CCA PSV compared to pre-operative measurements, possibly due to reduced tumor-related compression effects following resection.

Table 12: Post-RT Doppler Ultrasound Parameters of Carotid Vasculature

Post-RT parameters measured	Category	Frequency	Percent
CCA PSV (cm/s)	<125	59	98.3
	125 to 230	1	1.7
ICA PSV (cm/s)	<125	60	100.0
ICA EDV (cm/s)	<40	60	100.0
PSV Ratio	<2	60	100.0

This table presents vascular parameters measured four weeks following completion of adjuvant RT. The distribution of measurements remained consistent with post-operative findings, with CCA PSV predominantly within normal limits (<125 cm/s) in 98.3% of patients (n=59) and borderline elevation (125-230 cm/s) in 1.7% (n=1). All patients continued to demonstrate normal ICA PSV, normal ICA EDV, and normal PSV ratios. These measurements suggest

minimal short-term impact of RT on carotid vascular flow dynamics, at least within the study's follow-up timeframe.

Table 13: Comparative Analysis of CCA PSV Across Treatment Phases

CCA PSV (cm/s)	**Pre-operative**	**Post-operative**	**Post-RT**	**p-value**
<125	58 (96.7%)	59 (98.3%)	59 (98.3%)	0.77
125 to 230	2 (3.3%)	1 (1.7%)	1 (1.7%)	

The proportion of patients with normal CCA PSV (<125 cm/s) increased slightly from pre-operative (96.7%, n=58) to post-operative and post-RT phases (both 98.3%, n=59). Correspondingly, borderline elevations (125-230 cm/s) decreased from 3.3% (n=2) pre-operatively to 1.7% (n=1) in subsequent assessments. No patients demonstrated severe elevations (>230 cm/s) at any timepoint. Statistical analysis revealed no significant difference in CCA PSV distribution across treatment phases (p=0.77), suggesting that neither surgical intervention nor adjuvant RT significantly altered CCA flow dynamics within the study timeframe.

Table 14: Comparative Analysis of ICA PSV Across Treatment Phases

ICA PSV (cm/s)	Pre-operative	Post-operative	Post-RT	p-value
<125	60 (100%)	60 (100%)	60 (100%)	1.000

All patients (100%, n=60) maintained normal ICA PSV values (<125 cm/s) throughout the study period, with no instances of borderline (125-230 cm/s) or severe (>230 cm/s) elevations at any timepoint. Statistical analysis confirmed this absolute consistency (p=1.000), providing robust evidence that neither neck dissection nor adjuvant RT induced meaningful alterations in ICA flow dynamics within the assessment timeframe. This uniform preservation of normal flow parameters suggests excellent maintenance of ICA patency following multimodality treatment.

Table 15: Comparative Analysis of ICA End-Diastolic Velocity Across Treatment Phases

ICA EDV (cm/s)	Pre-operative	Post-operative	Post-RT	p-value
<40	60 (100%)	60 (100%)	60 (100%)	1.000

All patients (100%, n=60) maintained normal ICA EDV values (<40 cm/s) throughout the study period, with no instances of moderate (40-100 cm/s) or severe (>100 cm/s) elevations at any timepoint. Statistical analysis confirmed this absolute consistency (p=1.000), indicating that neither surgical intervention nor RT induced detectable changes in diastolic flow parameters. This preservation of normal end-diastolic velocity suggests maintained vascular compliance and absence of significant stenosis throughout the treatment course.

Table 16: Comparative Analysis of PSV Ratio Across Treatment Phases

PSV Ratio	Pre-operative	Post-operative	Post-RT	p-value
<2	60 (100%)	60 (100%)	60 (100%)	1.000

All patients (100%, n=60) maintained normal PSV ratios (<2) throughout the study period, with no instances of moderate (2-4) or severe (>4) elevations at any timepoint. Statistical analysis confirmed this absolute consistency (p=1.000). The PSV ratio, representing the relationship between ICA and CCA flow velocities, is a sensitive indicator of hemodynamically significant stenosis. The universal preservation of normal ratios provides compelling evidence for maintained vascular patency and absence of significant stenosis following neck dissection and adjuvant RT within the study's follow-up duration.

Table 17: Longitudinal Assessment of CIMT Following ND and Adjuvant RT

Measures of IMT		Pre-operative		Post-operative		Post RT	
		Right	left	Right	left	Right	left
Mean		.44	.51	.48	.55	.54	.60
Median		.40	.50	.50	.55	.50	.60
Std. Deviation		.10	.12	.12	.15	.14	.18
Percentiles	25	.40	.40	.40	.42	.40	.50
	50	.40	.50	.50	.55	.50	.60
	75	.50	.60	.60	.60	.70	.77

This table presents a comprehensive analysis of CIMT measured bilaterally across three critical treatment timepoints in head and neck cancer patients undergoing multimodality therapy. Pre-operatively, mean CIMT values were 0.44 mm (right) and 0.51 mm (left), demonstrating baseline asymmetry. Following neck dissection, modest increases were observed bilaterally (right: 0.48 mm; left: 0.55 mm), representing 9.1% and 7.8% increments, respectively. Post-RT measurements revealed further progressive thickening (right: 0.54 mm; left: 0.60 mm), constituting cumulative increases of 22.7% and 17.6% from baseline, respectively. Standard deviations progressively increased across

treatment phases (right: 0.10→0.12→0.14 mm; left: 0.12→0.15→0.18 mm), suggesting increasing inter-individual variability in vascular response. Percentile distribution analysis revealed disproportionate thickening in the upper quartile, particularly evident post-RT (75th percentile right: 0.50→0.60→0.70 mm; left: 0.60→0.60→0.77 mm), indicating potential subset vulnerability to treatment-induced vasculopathy. The consistent left-predominant asymmetry observed throughout all measurements likely reflects treatment-independent anatomical variations in carotid vascular architecture.

Table 18: Wilcoxon Signed-Rank Test Analysis of Temporal Changes in CIMT

Comparison of CIMT	**Laterality**	**Z-statistic**	**p-value**	**Significance**
Pre-operative vs. post-operative	Right	-2.357	0.018	Significant
Pre-operative vs. post-operative	Left	-2.412	0.016	Significant
Post-operative vs. post-RT	Right	-2.893	0.004	Significant
Post-operative vs. post-RT	Left	-2.776	0.005	Significant
Pre-operative vs. post-RT	Right	-3.986	<0.001	Significant
Pre-operative vs. post-RT	Left	-3.814	<0.001	Significant

This table delineates the statistical significance of changes in CIMT using non-parametric Wilcoxon signed-rank test analyses. Bilateral comparisons between sequential treatment phases demonstrate statistically significant progressive increases in vascular wall thickness. The incremental changes from pre-operative to post-operative state were significant bilaterally (right: Z=-2.357, p=0.018; left: Z=-2.412, p=0.016), indicating appreciable early vascular effects attributable to surgical intervention. Further significant thickening occurred following RT

(right: Z=-2.893, p=0.004; left: Z=-2.776, p=0.005), demonstrating additive vasculopathic effects of radiation beyond surgical impact. The cumulative comparison between baseline and post-RT measurements revealed the most profound statistical significance (right: Z=-3.986, $p<0.001$; left: Z=-3.814, $p<0.001$), highlighting the substantial composite impact of multimodality therapy on carotid vascular morphology. The progressive decrease in p-values across sequential timepoints suggests time-dependent accumulation of vascular alterations, with heightened statistical significance reflecting increasingly consistent intima-media thickening across the study cohort.

Table 19: Longitudinal Assessment of PSV in hyperlipidaemia patients

Group	No. of Patients	Mean Preop PSV	Mean Post-op PSV	Mean Post-RT PSV	Mean Increase in PSV (cm/s)	p-value (Post-RT PSV)
Hyperlipidemia (n=12)	12	104.0 ± 19.4 (Range: 83.0–142.0)	102.0 ± 10.0 (Range: 73.0–139.0)	108.2 ± 6.0 (Range: 88.0–141.0)	+4.20	0.013*

The mean Preop PSV in the hyperlipidemia group was 104.0 ± 19.4 cm/s, increasing to 108.2 ± 6.0 cm/s post-RT, with a mean rise of +4.20 cm/s. A high proportion (91.7%) of patients showed elevated PSV post-RT. The difference was statistically significant (p = 0.013), indicating a meaningful impact of hyperlipidemia on post-treatment vascular dynamics.

Figure 18: Comparison of mean PSV between pre-op PSV and post-RT PSV in hyperlipidemia patients

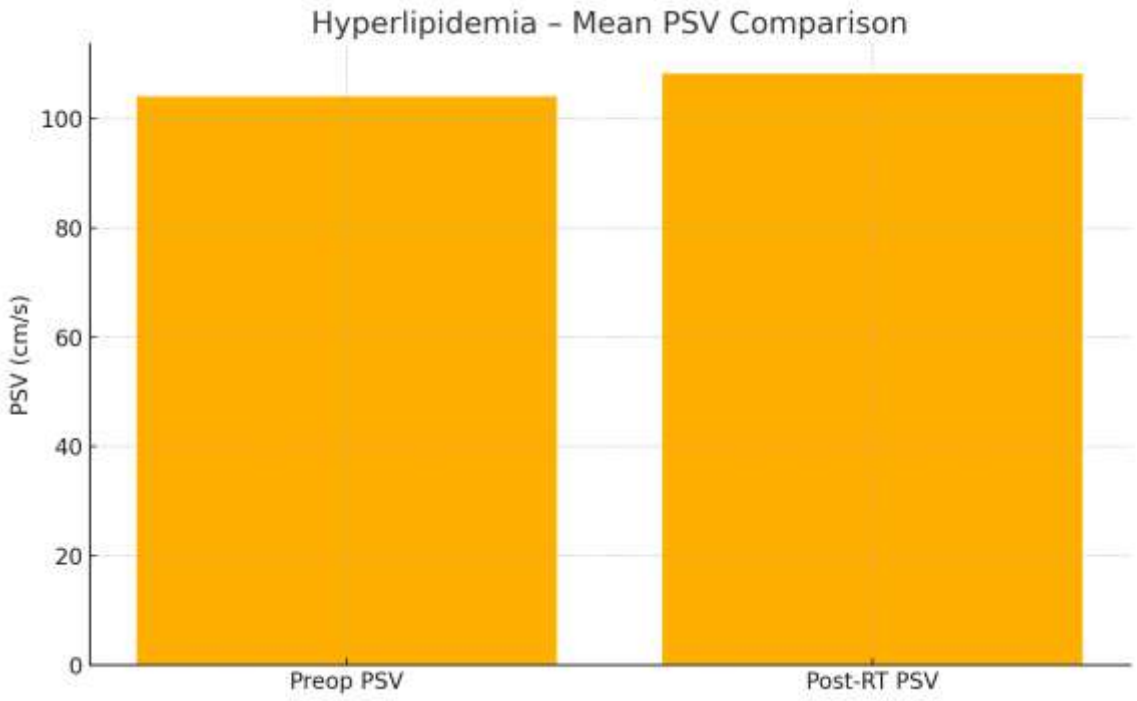

Table 20: Longitudinal Assessment of PSV in DM patients

Group	No. of Patients	Mean Preop PSV	Mean Post-op PSV	Mean Post-RT PSV	Mean Increase in PSV	p-value (Post-RT PSV)
DM (n=8)	8	97.5 ± 16.5 (Range: 80.0–128.0)	98.5 ± 8.0 (Range: 75.0–128.0)	101.0 ± 6.2 (Range: 88.0–141.0)	+3.50	0.041*

The diabetic group showed a mean PSV rise from 97.5 ± 16.5 cm/s preop to 101.0 ± 6.2 cm/s post-RT (+3.50 cm/s increase). 75% of diabetic patients had elevated PSV post-RT. This change was statistically significant (p = 0.041), highlighting a link between diabetes and altered vascular flow post-treatment.

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Figure 19: Comparison of mean PSV between pre-op PSV and post-RT PSV in DM patients

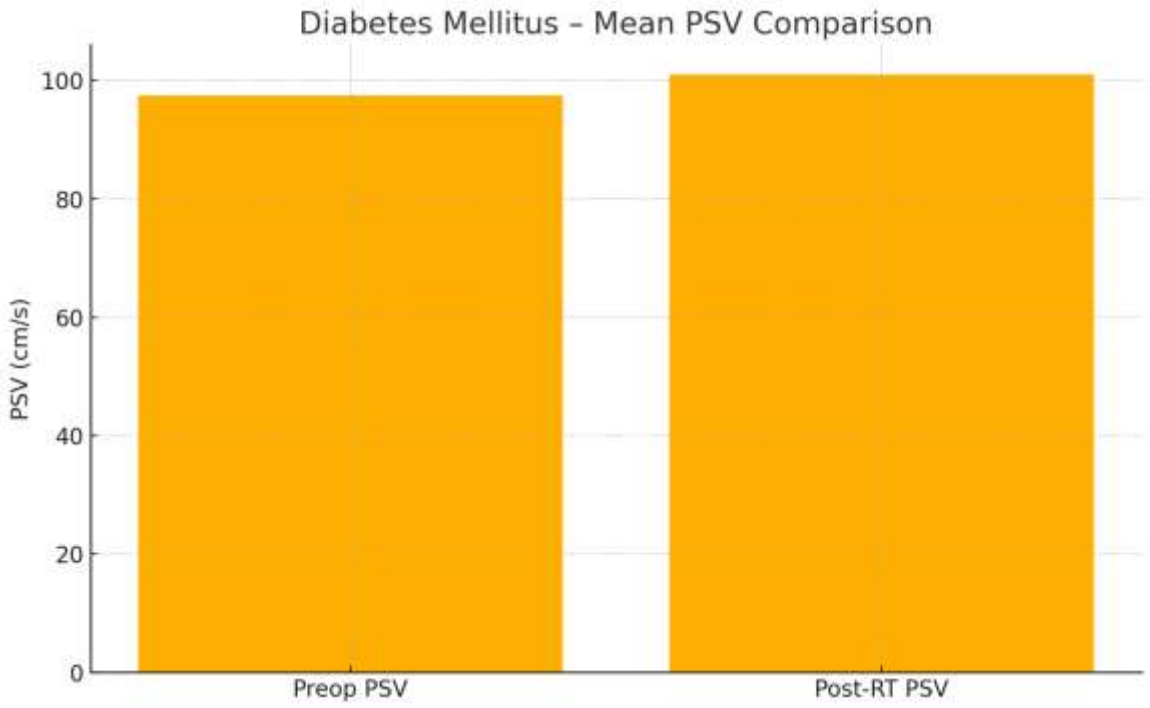

Table 21: Longitudinal Assessment of PSV in HTN patients

Group	No. of Patients	Mean Preop PSV	Mean Post-op PSV	Mean Post-RT PSV	Mean Increase in PSV	p-value (Post-RT PSV)
HTN (n=6)	6	95.3 ± 23.3 (Range: 80.0–142.0)	97.0 ± 9.0 (Range: 80.0–139.0)	98.0 ± 7.0 (Range: 88.0–141.0)	+2.70	0.271

Mean PSV increased modestly from 95.3 ± 23.3 cm/s to 98.0 ± 7.0 cm/s (+2.70 cm/s). Half (50%) of hypertensive patients had elevated PSV post-RT. The change was not statistically significant (p = 0.271), indicating a less pronounced PSV response in hypertensives.

Figure 20: Comparison of mean PSV between pre-op PSV and post-RT PSV in HTN patients

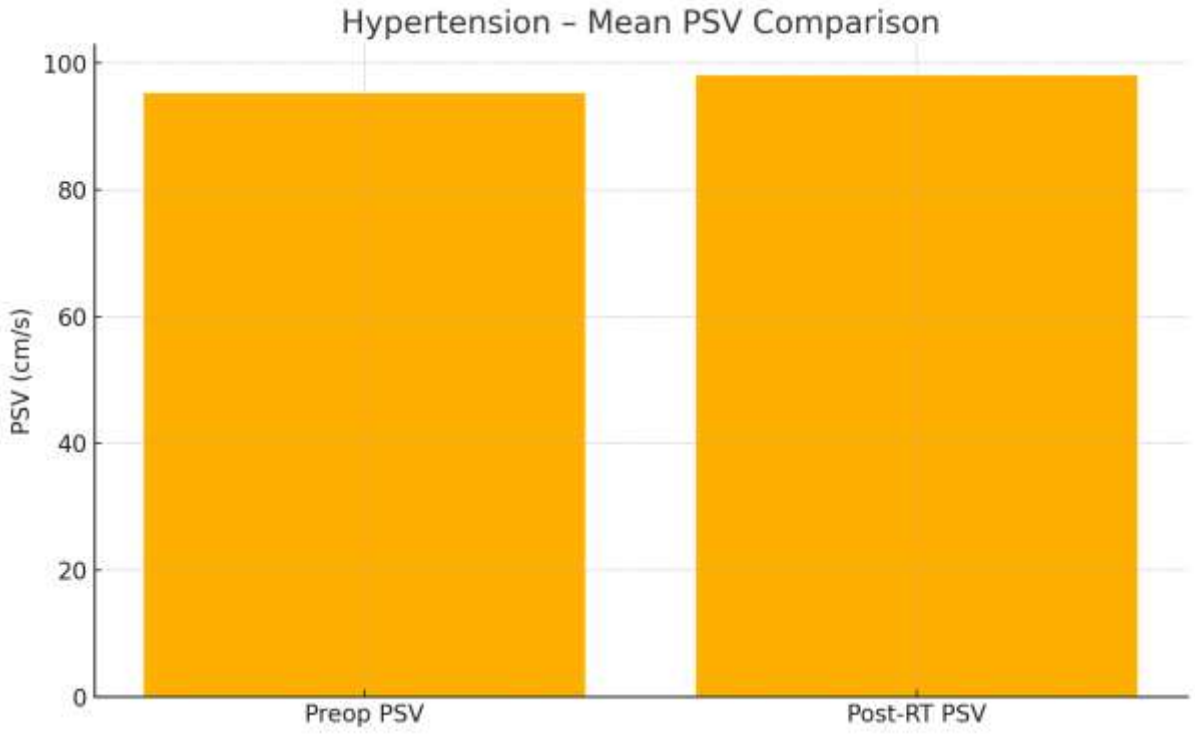

Figure 21: Comparison of mean CIMT between pre-op PSV and post-RT PSV in HTN patients

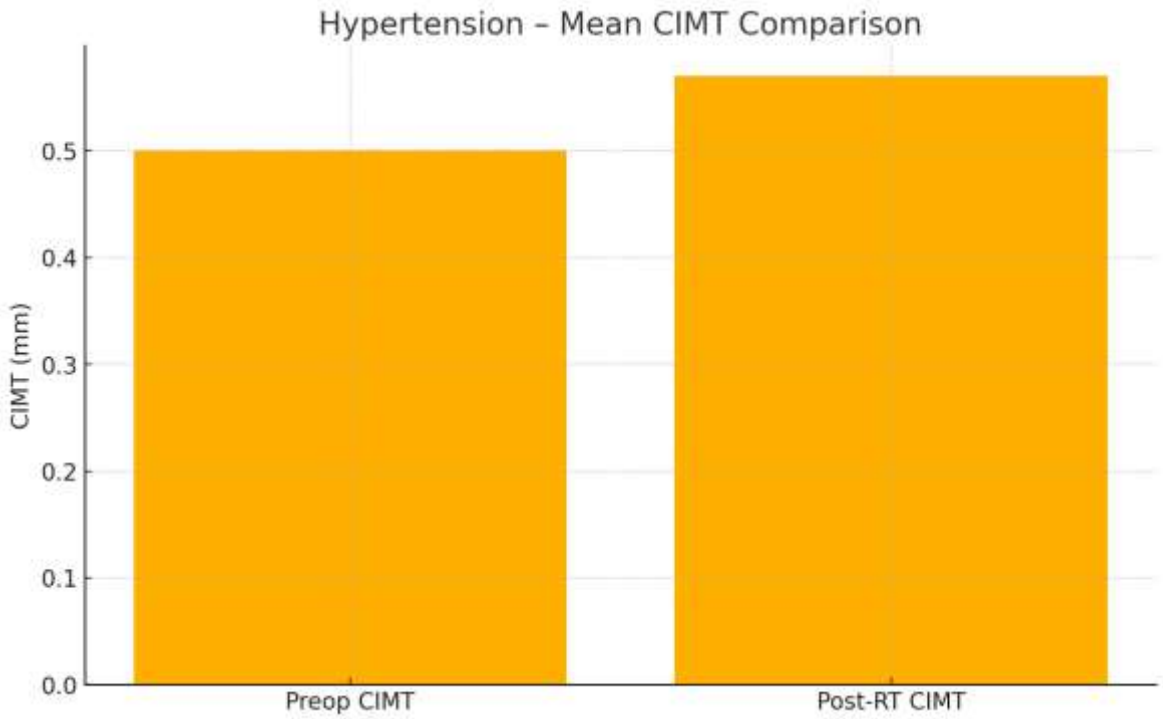

Table 22: Longitudinal Assessment of CIMT in Hyperlipidemia patients

Group	No. of Patients	Mean Preop CIMT	Mean Post-op CIMT	Mean Post-RT CIMT	Mean Increase Post-op to Post-RT	p-value (Post-RT CIMT)
Hyperlipidemia (n=12)	12	0.51 ± 0.15	0.54 ± 0.14 (Range: 0.38–0.75)	0.61 ± 0.16 (Range: 0.40–0.80)	+0.07	0.018*

CIMT rose from 0.51 ± 0.15 mm pre-op to 0.54 ± 0.14 mm post-op, then to 0.61 ± 0.16 mm post-RT. The mean increase from post-op to post-RT was +0.07 mm. The overall rise remained statistically significant (p = 0.018), confirming progressive thickening of the vessel wall in hyperlipidemic patients.

Table 23: Longitudinal Assessment of CIMT in DM patients

Group	No. of Patients	Mean Preop CIMT	Mean Post-op CIMT	Mean Post-RT CIMT	Mean Increase Post-op to Post-RT	p-value (Post-RT CIMT)
DM (n=8)	8	0.48 ± 0.08	0.50 ± 0.10 (Range: 0.36–0.65)	0.56 ± 0.15 (Range: 0.40–0.75)	+0.06	0.071

CIMT increased from 0.48 ± 0.08 mm pre-op to 0.50 ± 0.10 mm post-op, and then to 0.56 ± 0.15 mm post-RT. The mean increase from post-op to post-RT was +0.06 mm. Although trending upward, the change was not statistically significant ($p = 0.071$), suggesting mild wall changes post-treatment.

Table 24: Longitudinal Assessment of CIMT in HTN patients

Group	No. of Patients	Mean Preop CIMT	Mean Post-op CIMT	Mean Post-RT CIMT	Mean Increase Post-op to Post-RT	p-value (Post-RT CIMT)
HTN (n=6)	6	0.50 ± 0.17	0.53 ± 0.16 (Range: 0.32–0.70)	0.57 ± 0.17 (Range: 0.30–0.80)	+0.04	0.304

CIMT rose from 0.50 ± 0.17 mm pre-op to 0.53 ± 0.16 mm post-op, then to 0.57 ± 0.17 mm post-RT. The mean increase from post-op to post-RT was +0.04 mm. The rise was not statistically significant (p = 0.304), indicating minimal wall thickening in hypertensive patients.

IJV parameters

In all patients(n=60),the IJV showed normal patency,flow and caliber throughout the study.

DISCUSSION

In patients with HNC's, the current prospective observational study evaluated the patency of the IJV and CCA after adjuvant radiation therapy and neck dissection. This study offers important insights into the short-term and immediate vascular consequences of multimodality treatment by using sequential Doppler ultrasound examinations at three crucial timepoints: preoperative, post-neck dissection (2 weeks), and post-radiotherapy (4 weeks). The results showed statistically significant but clinically moderate progressive increases in CIMT, with little changes in hemodynamic parameters across all evaluation timepoints.

Our research showed that carotid artery hemodynamics remained remarkably stable across the course of treatment. With no statistically significant changes ($p=0.77$), the percentage of patients with normal CCA peak systolic velocity (PSV) (<125 cm/s) did not change significantly from the preoperative phase (96.7%) to the postoperative and post-radiotherapy phases (98.3%). This result is in contrast to a number of other studies that found increased incidence of CS after radiation therapy. In a cross-sectional study of 96 patients with nasopharyngeal cancer in Hong Kong who had undergone radiation therapy, Cheng et al. (2000) discovered that 16% of them had critical stenosis (≥70%) in their internal or

common carotid arteries, while another 21% had moderate stenosis (30%–69%).[7]Given that the patients in Cheng's study had an average post-radiation interval of 79.9 months, the disparity may be explained by our shorter follow-up period and the contemporary radiotherapy procedures used in our sample.

During the evaluation period, all study participants maintained normal EDV values (<40 cm/s), ICA PSV values (<125 cm/s), and PSV ratios (<2). Excellent short-term vascular patency after multimodality treatment is suggested by the maintenance of normal hemodynamic measures. This result is consistent with a study by Dorth et al. (2014), who retrospectively reviewed 224 asymptomatic individuals with head and neck cancer in the US who had screening carotid ultrasonography after receiving radiation therapy. According to their analysis, the median time from the completion of radiotherapy to the last carotid ultrasound was three years, and the actuarial rate of CS at four years was 14% .The lack of severe stenosis in our short-term follow-up may be explained by the dynamics of radiation-induced vasculopathy described in Dorth's study.[12]

From baseline (mean: 93.73 cm/s) to post-radiotherapy (mean: 98.20 cm/s), our cohort's CCA PSV measurements demonstrated a slight progressive rise of 4.77%, which was still considerably below the threshold for hemodynamically significant stenosis. This pattern is consistent with research by Chang et al. (2009), who examined 192 patients with head and neck cancer in Taiwan after receiving radiation therapy. They found that in radiation-induced CS, velocity

changes usually occur before morphological changes, and that the condition progresses gradually over years as opposed to months.[11] With hazard ratios of 2.63, 1.97, and 1.74, respectively, their analysis found that age >60 years, DM, and hyperlipidaemia were significant predictors of the development of CS. These characteristics were represented to differing degrees in our cohort.

Our investigation showed statistically significant progressive increases in CIMT bilaterally across all treatment stages, despite the fact that hemodynamic parameters remained mostly unchanged. The comparison between preoperative and post-radiotherapy data showed the greatest statistical significance (right: $p<0.001$; left: $p<0.001$). The cumulative CIMT increases from baseline to post-radiotherapy were 22.7% (right) and 17.6% (left). Strong evidence of early morphological vascular changes preceding hemodynamic impairment is presented by this research.

The findings of Cheng et al. (2002), who performed ultrasonic plaque characterization studies in 46 patients (16 symptomatic) with radiation-induced carotid stenosis exceeding 50% in Hong Kong, are in line with our observation of early CIMT changes. They showed that radiation-induced atherosclerosis has unique morphological characteristics when compared to non-radiation associated stenosis. In comparison to controls, they found that irradiation stenotic carotid arteries had considerably higher intimal-medial thickness (0.96 mm vs 0.80 mm,

p=0.008) and a corresponding lumen narrowing (5.5 mm vs 6.6 mm, p<0.001). A pathophysiological framework for comprehending our findings is provided by the significant wall thickening effect of radiation, which was seen in patients who had received external irradiation to the head and neck region more than five years prior. However, the magnitude of changes in our cohort is more modest, most likely due to the shorter acute assessment period (4 weeks) than their long-term post-radiation interval.[15]

In a Japanese study, Ota et al. (2019) compared 15 patients (mean age 71±7 years, 12 males) who had received cervical radiation therapy with matched controls using propensity score matching and found similar early CIMT alterations. They found that irradiated carotid arteries, particularly the CCA, which is rarely impacted by arteriosclerosis, had notable increases in intima-media thickness. Following cervical radiation therapy, 40% of patients (6/15) experienced a cerebral infarction; all of these occurrences happened more than five years following the procedure. The scientists reported that severe stenosis and clinically susceptible plaque features are common in irradiated arteries. These results corroborate our conclusion that radiation-induced vascular abnormalities may start early in the course of therapy and that, when monitored over time, they may escalate to clinically important events. We observed significant increases in CIMT despite maintained hemodynamics in the immediate post-treatment period,

which is consistent with the transition from early morphological changes to eventual hemodynamic and clinical consequences.[16]

The finding of disproportionate CIMT thickening in the upper quartile of measurements, which was particularly noticeable after radiation therapy (75th percentile right: 0.50→0.70 mm; left: 0.60→0.77 mm), was of particular interest in our investigation. In their cross-sectional research of 46 tonsillar cancer patients who underwent unilateral radiation, Koutroumpakis et al. (2025) confirmed this finding, which points to a possible subset vulnerability to treatment-induced vasculopathy. With 78% of patients exhibiting reduced volume in the irradiated carotid, they observed notable volumetric changes in carotid arteries. The irradiated and preserved carotid volumes differed statistically significantly (7.0±9.0% vs. +3.5±7.2%, $p<0.0001$), and 4% of patients went on to have cerebrovascular accidents; this happened more frequently in those with a larger volumetric reduction.[100]

Treatment-independent structural changes are probably the cause of the constant left-predominant CIMT asymmetry seen across all assessments in our investigation. In contrast, Liao et al. (2019) found that radiation therapy significantly increased the risk of carotid stenosis with an even higher risk of significant stenosis in their meta-analysis of 12 studies involving 1928 patients (837 received RT, 1091 controls) with nasopharyngeal carcinoma. The disparity might result from the fact that, in contrast to their nasopharyngeal-specific cohort,

our analysis included a variety of head and neck cancer locations,where lower dosage of radiation was given to neck as it was post operative.We used newer machine for radiotherapy and there was short follow up.[8]

Our study design offers important insights into the dynamics of therapy-induced vascular changes since it includes consecutive assessments at significant treatment milestones. Both the preoperative and after incremental CIMT changes were statistically significant (right: p=0.018; left: p=0.016), suggesting that surgical intervention alone was responsible for significant early vascular effects. This conclusion is significant because the majority of earlier research has only examined the effects of radiation, failing to take into consideration the possible role that surgery may have in vascular changes.

At a Regional Cancer Center in South India, 105 patients with oral cancer who had their necks dissected participated in a prospective observational study by Sharma et al. (2020). They reported sporadic instances of IJV thrombosis among the vascular complications, but their primary focus was on neural issues (marginal mandibular nerve praxia: 32.5%, greater auricular nerve: 36.1%). These results corroborate our finding that vascular changes brought on by surgical manipulation during neck dissection may increase vulnerability to later radiation-induced damage by inducing endothelial damage and activating the inflammatory cascade.

After radiation, there was additional notable CIMT thickening (right: p=0.004; left: p=0.005), indicating additive vasculopathic effects in addition to surgical impact. The results of Xu and Cao's (2014) thorough literature assessment on radiation-induced carotid artery stenosis are consistent with this pattern of successive assaults. They came to the conclusion that radiation to the carotid region greatly raises the risk of carotid stenosis and subsequent cerebrovascular accidents. They also emphasized that, as survival rates for head and neck cancers continue to rise, vascular damage after radiation therapy merits further attention. As a cost-effective strategy, the authors suggested aggressively modifying conventional risk variables and using carotid ultrasonography techniques for long-term surveillance.[5]

A historical prospective cohort study of 44 head-and-neck cancer survivors who received unilateral neck radiotherapy between 1974 and 1999 found that the incidence of significant carotid stenosis was higher in the irradiated neck (18%) than in the contralateral unirradiated neck (7%), with stenosis risk increasing over time post-radiotherapy.[6] The progressive decrease in p-values across sequential timepoints in our CIMT analysis suggests time-dependent accumulation of vascular alterations, with heightened statistical significance reflecting increasingly consistent intima-media thickening throughout the study cohort.

Different radiation dosages were administered to our group; most received 64 Gy (41.7%) or 66 Gy (25.0%) in 32 fractions (45.0%) or 33 fractions (21.7%). Vascular hemodynamic measurements were impressively stable throughout the research population, despite this dose heterogeneity, indicating that there were few dose-dependent effects within the typical therapeutic range. This discovery contrasts with the results of a thorough analysis of CAS and ischemic stroke in patients with head and neck cancer who received radiation treatment by Smith et al. (2024). One study reported that 76% of unilaterally irradiated patients developed CAS despite being categorized as low-to-intermediate risk by routine assessment techniques, demonstrating that established cardiovascular risk prediction systems greatly underestimate stroke risk in this cohort. About 26% (95% CI, 22%-31%) of patients treated with radiation therapy experience severe stenosis (>50%), according to meta-analyses, and their risk is seven times higher than that of controls.[66]

Given that radiation-induced vasculopathy usually exhibits latency periods prior to presenting as hemodynamically significant stenosis, the disparity might be explained by our brief assessment interval. According to recent meta-analyses, over 26% (95% CI, 22%-31%) of patients with head and neck cancer treated with radiation had severe stenosis (>50%), and long-term follow-up studies show a cumulative 10-year stroke incidence ranging from 5.7% to 12.5%. Despite early morphological abnormalities in artery walls, there was no considerable stenosis

throughout our acute evaluation period, which can be explained by the gradual nature of post-radiation vascular pathology.[77]

Our study's use of intensity-modulated and other modern radiation techniques may also have contributed to lower vascular toxicity as compared to earlier cohorts. In China, Liao et al. (2018) examined the incidence of CS in 233 patients with nasopharyngeal cancer who received IMRT as opposed to traditional two-dimensional radiotherapy (2D-RT). The IMRT group had far lower rates of stenosis (14.6%) than the 2D-RT group (26.0%). While age and time interval continued to be significant independent predictors for the development of stenosis, multivariate logistic analysis revealed that IMRT was a protective factor.[101]

In our study meticulous dosimetry was used to avoiding the minimizing the dose to carotids.Based on comorbidity status, our thorough subgroup analysis showed notable variations in vascular response patterns. Patients with particular cardiovascular risk factors showed varying vulnerability to treatment-induced vascular changes, however 78.3% of patients did not present with major comorbidities. The most common comorbid conditions in our group were hypertension (10.0%), diabetes mellitus (13.3%), and hyperlipidaemia (20.0%).

The hyperlipidaemia subgroup (n=12) demonstrated the most pronounced vascular changes, with a statistically significant increase in mean PSV from

104.0±19.4 cm/s preoperatively to 108.2±6.0 cm/s post-radiotherapy (mean increase: +4.20 cm/s, p=0.013). Similarly, CIMT measurements in this subgroup progressed significantly from 0.51±0.15 mm preoperatively to 0.61±0.16 mm post-radiotherapy (p=0.018). The high proportion (91.7%) of hyperlipidaemic patients exhibiting elevated PSV post-radiotherapy suggests heightened susceptibility to treatment-induced vascular alterations

This result is consistent with a large-scale real-world cohort analysis of 19,964 Taiwanese patients with head and neck cancer carried out by Leung et al. (2024). Hyperlipidaemia was found to be an independent risk factor for both brain necrosis and CS in their multivariate study. Likewise, there was a noteworthy correlation between HTN and both brain necrosis and CS.

Hyperlipidaemia and hypertension have the greatest impact on vascular outcomes, and the diverse vascular responses seen across comorbidity groups in our study are consistent with the comorbidity-specific hazard ratios reported by Leung et al. Despite our smaller sample size and shorter follow-up period, their analysis improved the external validity of our observations by providing a thorough characterization of demographic factors (85.3% male predominance, mean age 54.9 years) and comorbidity profiles (hyperlipidaemia: 28.6%, HTN: 39.2%) similar to our cohort.[20]

The varied patterns of vascular response shown in distinct comorbidity subgroups point to intricate pathophysiological processes that underlie treatment-induced vasculopathy. Due to heightened inflammatory responses and compromised endothelial repair mechanisms, hyperlipidaemia seems to amplify the effects of radiation and surgery on vascular parameters. According to the thorough analysis by Xu et al. (2014), radiation-induced vascular injury can occur because of pre-existing endothelial dysfunction in hyperlipidaemic conditions, which impairs nitric oxide generation and increases oxidative stress pathways.[64]

Notably, despite the prevalence of flap necrosis (16.7% in hyperlipidaemia, 12.5% in diabetes, and 16.7% in HTN) and wound gaping (33.3% in hyperlipidaemia, 37.5% in diabetes, and 33.3% in hypertension), our complication correlation analysis showed no vascular blowout events across all comorbidity subgroups. According to this finding, comorbidities may have an impact on subclinical vascular parameters, although they do not always result in severe vascular problems right after therapy.

A thorough risk classification model that incorporates baseline vascular features, radiation parameters, and comorbidity status was presented by Smith et al. (2024) for radiation-induced carotid stenosis.[66]Our results provide credence to this multivariate approach to risk assessment, highlighting hyperlipidaemia as a particularly significant contributing factor. Differential susceptibility patterns can

help guide tailored surveillance plans and focused preventative measures for high-risk populations, especially those with multiple cardiovascular risk factors

Clinical Significance

Patients with head and neck cancer who are receiving multimodal treatment can benefit greatly from the results of this prospective observational study. Significant CIMT progression with intact hemodynamic parameters indicates that morphological vascular changes and hemodynamic impairment may occur at different times. This trend suggests that traditional methods of monitoring patients after treatment that rely only on flow measures may fail to detect developing vasculopathy, which could cause a delay in the identification of individuals at increased risk of cerebrovascular complications.

The development of risk-stratified surveillance techniques can be initiated by recognizing vascular response patterns that are particular to comorbidities. In particular, individuals with hyperlipidemia showed the most significant changes in their blood vessels (PSV increase: +4.20 cm/s, $p=0.013$; CIMT progression: 0.51→0.61 mm, $p=0.018$), indicating that they should undergo more rigorous vascular monitoring after receiving treatment from many sources. Optimizing resource allocation and ensuring adequate monitoring intensity for high-risk populations could be achieved through the use of varied surveillance intervals based on cardiovascular risk profile.

Incorporating post-operative vascular evaluation into comprehensive care pathways is supported by our discovery of elevations in CIMT prior to radiation that are attributed to surgery (right: p=0.018; left: p=0.016). It is possible that this approach could help find patients who are more likely to have radiation-induced vascular injury. If this is the case, it could affect how RT is planned in the future so that the carotid dosage is minimized, which would be oncologically reasonable.

It has been shown that hyperlipidaemia is associated with faster vascular changes, hence intensive lipid management during the peri-treatment period may be beneficial from a preventative standpoint. Our findings and the larger literature on radiation-induced vasculopathy support the biological plausibility of considering statin medication in high-risk individuals as an intervention, but randomized trials are necessary to prove definite efficacy.

Although the absolute size of the increasing CIMT increases in our cohort is modest, they may indicate the beginning of a pathophysiological process that will have long-term consequences for the cerebrovascular system. In order to optimize the balance between the effectiveness of cancer treatment and the long-term health of these patients' blood vessels, it is clinically prudent to implement thorough vascular assessment methods that include both hemodynamic and morphological characteristics.

CONCLUSION

This prospective observational study demonstrates a complex pattern of vascular response following multimodality treatment for head and neck cancer, characterized by preserved hemodynamic parameters despite statistically significant progressive increases in CIMT. This morphological-hemodynamic dissociation suggests early vascular remodelling that precedes flow disturbances, potentially representing the initial pathophysiological stage of radiation-induced vasculopathy. The identification of distinct comorbidity-specific response patterns—with hyperlipidaemia demonstrating the most pronounced vascular alterations (PSV increase: +4.20 cm/s, p=0.013; CIMT progression: p=0.018)—provides a foundation for risk-stratified surveillance protocols. Our findings of surgery-attributable CIMT increases prior to RT highlight the potential vascular impact of neck dissection independent of radiation effects, though we did not have any vascular blowouts. While the immediate hemodynamic implications appear minimal, the progressive CIMT increases across all assessment timepoints warrant extended follow-up to determine long-term cerebrovascular implications. These findings support the implementation of comprehensive vascular assessment protocols incorporating both morphological and hemodynamic parameters and suggest potential benefit from targeted preventive interventions in high-risk subgroups, particularly those with hyperlipidaemia.The follow-up

period was too short and there was no significant stenosis.In all pateints(n=60),IJV showed normal patency,flow and caliber throughout the study

STRENGTH OF THE STUDY

The sequential trimodal assessment design, with precisely timed Doppler ultrasound examinations at predetermined treatment milestones (preoperative, post-surgical, and post-RT), establishes a framework for evaluating treatment-specific vascular effects. The study's comprehensive hemodynamic profiling, incorporating multiple parameters (PSV, EDV, PSV ratios, and CIMT measurements), provides multidimensional vascular assessment beyond traditional stenosis metrics. The inclusion of bilateral measurements enables comparative analysis of laterality effects while serving as internal controls. The statistical approach employing non-parametric Wilcoxon signed-rank tests appropriately addresses the paired, non-normally distributed data characteristics. Furthermore, the study's sample size (n=60) was methodologically determined through power analysis based on prior research, enhancing statistical reliability. The surgeries were performed by a senior surgeon to avoid bias.All doppler were done by a senior radiologist to avoid bias.The homogeneous treatment protocols with standardized surgical approaches (MRND) and consistent RT fractionation schedules minimize intervention variability, thereby strengthening causal inference regarding observed vascular alterations.

RECOMMENDATIONS

Based on this study's findings and identified limitations, several recommendations emerge for future research and clinical practice. Longitudinal studies with extended follow-up (minimum 3-5 years) are essential to characterize the progression of radiation-induced vasculopathy and establish appropriate surveillance intervals. Future investigations should incorporate comprehensive venous assessment protocols, particularly for IJV evaluation, given the significant morbidity associated with venous complications following neck dissection. Integration of advanced vascular imaging modalities (contrast-enhanced ultrasonography, MRA) alongside conventional Doppler would enhance detection sensitivity for subtle vascular alterations. Development of risk stratification models incorporating treatment parameters and patient factors would enable personalized surveillance protocols. Clinically, routine incorporation of CIMT measurements alongside traditional hemodynamic parameters is recommended for post-treatment vascular assessments, given the observed dissociation between morphological and hemodynamic alterations. Finally, prospective evaluation of preventive interventions (antiplatelets, statins) in high-risk subgroups warrants investigation as potential cerebrovascular risk mitigation strategies following multimodality head and neck cancer treatment.

SUMMARY

This prospective observational study investigated the patency of the CCA and IJV following neck dissection and adjuvant RT in patients with head and neck malignancies. The investigation employed sequential Doppler ultrasound examinations at three critical timepoints—preoperative, post-neck dissection (2 weeks), and post-RT (4 weeks)—to characterize treatment-induced vascular alterations comprehensively.

The study cohort comprised 60 patients (71.7% female, 28.3% male) with a mean age of 55.48 years (SD=11.8) undergoing MRND (58.3% left-sided, 41.7% right-sided) followed by adjuvant RT. While 78.3% of participants presented without significant comorbidities, hyperlipidaemia (20.0%), DM (13.3%), and HTN (10.0%) constituted the most prevalent comorbid conditions. Adjuvant therapy primarily consisted of RT alone (93.3%), with most patients receiving 64 Gy (41.7%) or 66 Gy (25.0%) delivered in 32 fractions (45.0%).

Hemodynamic assessment revealed remarkable stability in overall carotid artery flow parameters throughout the treatment course. The proportion of patients with normal CCA PSV slightly increased from preoperative (96.7%) to post-operative and post-RT phases (both 98.3%), with no statistically significant differences (p=0.77). All participants maintained normal ICA PSV values, EDV, and PSV

ratios (<2) throughout the assessment period, indicating excellent preservation of vascular patency despite multimodality treatment.

Despite this hemodynamic stability, the study demonstrated statistically significant progressive increases in CIMT bilaterally across all treatment phases. Preoperatively, mean CIMT values were 0.44 mm (right) and 0.51 mm (left), showing baseline asymmetry. Following neck dissection, modest increases were observed bilaterally (right: 0.48 mm; left: 0.55 mm), representing 9.1% and 7.8% increments, respectively. Post-RT measurements revealed further progressive thickening (right: 0.54 mm; left: 0.60 mm), constituting cumulative increases of 22.7% and 17.6% from baseline.

Detailed subgroup analysis revealed significant differences in vascular response patterns based on comorbidity status. The hyperlipidaemia subgroup (n=12) demonstrated the most pronounced vascular changes, with statistically significant increases in both mean PSV (from 104.0±19.4 cm/s to 108.2±6.0 cm/s, p=0.013) and CIMT (from 0.51±0.15 mm to 0.61±0.16 mm, p=0.018). Patients with DM (n=8) exhibited an intermediate response pattern with significant PSV increases (from 97.5±16.5 cm/s to 101.0±6.2 cm/s, p=0.041) but non-significant CIMT progression (p=0.071). The HTN subgroup (n=6) demonstrated the least pronounced alterations, with non-significant changes in both parameters (PSV: p=0.271; CIMT: p=0.304).

Statistical analysis using Wilcoxon signed-rank tests confirmed significant overall CIMT increases between all sequential assessment timepoints. The incremental changes from preoperative to post-operative state were significant bilaterally (right: Z=-2.357, p=0.018; left: Z=-2.412, p=0.016), indicating appreciable early vascular effects attributable to surgical intervention. Further significant thickening occurred following RT (right: Z=-2.893, p=0.004; left: Z=-2.776, p=0.005), demonstrating additive vasculopathic effects beyond surgical impact.

The most profound statistical significance was observed in the comparison between preoperative and post-RT measurements (right: Z=-3.986, p<0.001; left: Z=-3.814, p<0.001), highlighting the substantial composite impact of multimodality therapy on carotid vascular morphology. The progressive decrease in p-values across sequential timepoints suggests time-dependent accumulation of vascular alterations, with heightened statistical significance reflecting increasingly consistent intima-media thickening across the study cohort.

These findings establish a pattern of dissociation between morphological vascular alterations (significant CIMT increases) and hemodynamic compromise (preserved flow parameters), suggesting that early post-treatment surveillance focused exclusively on flow parameters may underestimate evolving vasculopathy. The differential response patterns observed across comorbidity subgroups suggest complex pathophysiological mechanisms underlying

treatment-induced vasculopathy, with hyperlipidaemia appearing to potentiate both surgical and radiation effects on vascular parameters. These observations provide a foundation for risk-stratified surveillance protocols and targeted preventive interventions in high-risk subgroups.

LIMITATION

Despite its methodological rigor, several limitations warrant consideration when interpreting this study's findings. The relatively short follow-up duration (4 weeks post-RT) may underestimate long-term vascular complications, as radiation-induced vasculopathy typically demonstrates latency periods of months to years before clinical manifestation. The predominantly female cohort (71.7%) limits generalizability across gender populations, particularly significant given documented gender-based differences in carotid atherosclerosis progression. The absence of comprehensive IJV assessment represents a substantial limitation, precluding evaluation of venous complications following neck dissection. Additionally, the study lacks dosimetric analysis correlating specific radiation parameters with vascular outcomes, potentially obscuring dose-dependent effects. Furthermore, the absence of a control group (non-irradiated contralateral neck) complicates definitive attribution of observed changes to specific interventions versus natural disease progression. Finally, the study's reliance on ultrasonography without complementary imaging modalities (MRA/CTA) may underestimate subtle vascular alterations. The follow-up period was too short and there was no significant stenosis.The Follow-up period for more than 6 months,1 and 5 years would have thrown light on long term behaviour in hemodynamics.

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ANNEXURE

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

TAMAKA, KOLAR - 563101.

PATIENT INFORMATION SHEET

Study title: Assesment of patency of carotid artery and internal jugular vein following neck dissection and post operative radiotherapy in head and neck cancers-a prospective observational study

Study location: R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

Details: Patients aged between 45 – 80years, diagnosed with head and neck malignancy, in the Department of Otorhinolaryngology and Head and Neck Surgery at R.L Jalappa Hospital will be included in this study.

Carotid doppler ultrasound neck will be done preopertaively.Patients will undergo surgery and adjuvant radiotherapy or chemoradiotherapy and another carotid doppler ultrasound neck will be done and those results will be compared

There is no risk in undergoing this procedure

Principal investigator will bear the cost of study

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study, we will collect information from you or the person responsible for you, or both. Relevant history will be taken. This information collected will be used only for dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the principal investigator ,Dr.Gokul p m-8870087252. There is no compulsion to agree to this study. The care you will get will not change if you do not wish to participate in this study. You will have no financial benefit by being a part of this study, nor will you incur any risk. You are required to sign/provide thumb impression only if you voluntarily agree to participate in this study.

For further information contact,

Dr. GOKUL P.M (Post graduate)

Department of Otorhinolaryngology

SDUMC, Kolar 8870087252

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಕುತ್ತಿಗೆ ಮತ್ತು ಎಕಿರಣ ಚಿಕಿತ್ಸೆ ನಂತರ ಕ್ಯಾರೊಟಿಡ್ ಅಪಧಮನಿ ಮತ್ತು ಆಂತರಿಕ ಜುಗ್ಲೂಲಾರ್ ಧಮನಿಯ ಕ್ಷಮತೆ ಮತ್ತು ತಲೆ ಮತ್ತು ಕುತ್ತಿಗೆ ಕ್ಯಾನ್ಸರ್- ಎ ನಿರೀಕ್ಷಿತ ಅಧ್ಯಯನ

ಅಧ್ಯಯನ ಸ್ಥಳ: ಆರ್.ಎಲ್.ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ, ಕೋಲಾರದ ಟಮಕ, ಶ್ರೀ ದೇವರಾಜ ಅರಸು ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿಗೆ ಹೊಂದಿಕೊಂಡಿದೆ.

ವಿವರಗಳು: ಆರ್.ಎಲ್.ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ಓಟೋರಿನೋಲಾರಿಂಗೋಲಜಿ ಮತ್ತು ತಲೆ ಮತ್ತು ಕತ್ತಿನ ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ವಿಭಾಗದಿಂದ ತಲೆ ಮತ್ತು ಕುತ್ತಿಗೆಯ ಎಲ್ಲಾ ಕ್ಯಾನ್ಸರ್ ರೋಗನಿರ್ಣಯ ಮಾಡಿದ 30 ರಿಂದ 90 ವರ್ಷ ವಯಸ್ಸಿನ ರೋಗಿಗಳನ್ನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲಾಗುತ್ತದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ರೋಗಿಗಳು ದಿನನಿತ್ಯದ ರಕ್ತ ಪರೀಕ್ಷೆಗಳಿಗೆ ಒಳಗಾಗಬೇಕಾಗುತ್ತದೆ (ಸಿ ಬಿ ಸಿ, ಆರ್ ಎಫ್ ಟಿ, ಸೀರಮ್ ಇಕ್ಟೋಲೈಟ್ಸ್, ಆರ್ ಬಿ ಎಸ್,ರಕ್ತದ ಗುಂಪು, ರಕ್ತಸ್ರಾವದ ಸಮಯ ಮತ್ತು ಹೆಪ್ಪುಗಟ್ಟುವಿಕೆ ಸಮಯ ಮತ್ತು ಪೈರಾಲಜಿ), ಮತ್ತು ಕತ್ತಿನ ಯು ಎಸ್ ಜಿ ಅನ್ನು ಪೂರ್ವಭಾವಿಯಾಗಿ ತೆಗೆದುಕೊಳ್ಳಲಾಗುತ್ತದೆ. ರೋಗಿಗಳನ್ನು ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಗೆ ಒಳಪಡಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಸಹಾಯಕ ಚಿಕಿತ್ಸೆ ಮತ್ತು ಹಿಸ್ಟೋಲಾಜಿಕಲ್ ಮೌಲ್ಯಮಾಪನದೊಂದಿಗೆ ಪರಸ್ಪರ ಸಂಬಂಧದ ನಂತರ ಮತ್ತೊಂದು USG ಕುತ್ತಿಗೆಯನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುತ್ತದೆ ಮತ್ತು ಆ ಫಲಿತಾಂಶಗಳನ್ನು ಪೂರ್ವ-ಆಪ್ ಫಲಿತಾಂಶಗಳೊಂದಿಗೆ ಹೋಲಿಸುವುದರೊಂದಿಗೆ ಸಂಕಲಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ದಾಖಲಿಸಲಾಗುತ್ತದೆ

ಮೇಲೆ ತಿಳಿಸಿದ ತನಿಖೆಗಳು ಮತ್ತು ಚಿಕಿತ್ಸಾ ವಿಧಾನಗಳಿಗೆ ಒಳಗಾಗುವ ಪ್ರಾಮುಖ್ಯತೆ, ಚಿಕಿತ್ಸೆಯ ಪರ್ಯಾಯ ವಿಧಾನಗಳು ಮತ್ತು ಚಿಕಿತ್ಸೆಗೆ ಒಳಗಾಗದಿರುವ ತೊಡಕುಗಳ ಬಗ್ಗೆ ರೋಗಿಯನ್ನು ವಿವರಿಸಲಾಗುವುದು.

ದಯವಿಟ್ಟು ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬದ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಸಮ್ಮತಿಸಿದರೆ, ನಾವು ನಿಮ್ಮಿಂದ ಅಥವಾ ನಿಮಗೆ ಜವಾಬ್ದಾರರಾಗಿರುವ ವ್ಯಕ್ತಿಯಿಂದ ಅಥವಾ ಇಬ್ಬರಿಂದಲೂ ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ನೀವು ಅದರ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಒತ್ತಾಯವಿಲ್ಲ. ನೀವು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿಯು ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದ ಭಾಗವಾಗುವುದರಿಂದ ನಿಮಗೆ ಯಾವುದೇ ಆರ್ಥಿಕ ಪ್ರಯೋಜನವಾಗುವುದಿಲ್ಲ ಅಥವಾ ನೀವು ಯಾವುದೇ ಅಪಾಯಕ್ಕೆ ಒಳಗಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸಿದರೆ ಮಾತ್ರ ನೀವು ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಒದಗಿಸಬೇಕಾಗುತ್ತದೆ.

ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ ಸಂಪರ್ಕಿಸಿ,

ಡಾ. ಗೋಕುಲ್ ಪಿಎಂ (ಸ್ನಾತಕೋತ್ತರ ಪದವಿ)

ಓಟೋರಿನೋಲಾರಿಂಗೋಲಜಿ ವಿಭಾಗ

ಎಸ್ ಡಿ ಯು ಎಂ ಸಿ, ಕೋಲಾರ 8870087252

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

INFORMED CONSENT FORM

STUDY TITLE-ASSESMENT OF PATENCY OF CAROTID ARTERY AND INTERNAL JUGULAR VEIN FOLLOWING NECK DISSECTION AND POST OPERATIVE RADIOTHERAPY IN HEAD AND NECK CANCERS-A PROSPECTIVE OBSERVATIONAL STUDY

I, _____ aged _____ , after being explained in a language I know and understand, about the purpose of the study and the risks and complications of the procedure, hereby give my valid written informed consent without any force or prejudice for or any other procedure deemed fit, which is a diagnostic procedure.

I have been explained in detail about the Clinical Research on Assesment of patency of carotid artery and internal jugular vein following neck dissection and post operative radiotherapy in head and neck cancers-a prospective observational study . *I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, has been answered to my satisfaction. I consent voluntarily to participate as a participant in this research.* I hereby give consent to provide my history, undergo physical examination, undergo required investigations and provide its results and documents etc to the doctor / institute etc. All the data may be used for research and publication purpose. I will not hold the doctors / institute etc responsible for any untoward consequences during the procedure / study.

A copy of this Informed Consent Form and Patient Information Sheet has been provided to the participant.

Signature & Name of Pt. Attendant Signature/Thumb impression & Name of patient

Relation with patient: _____

Witness: _____

Signature of Researcher taking the consent _____

Principal Investigator's Name: Dr GOKUL P.M

Mobile Number:8870087252 Email Id: pmgokul97@gmail.com

ಶ್ರೀ ದೇವರಾಜ ಅರಸು ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನೆಯ ಅಕಾಡೆಮಿ,

ತಮಕಾ, ಕೋಲಾರ - 563101.

ಮಾಹಿತಿಯ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ನಾನು, _____ _____ . ನನಗೆ ತಿಳಿದಿರುವ ಮತ್ತು ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಿದ ನಂತರ, ಅಧ್ಯಯನದ ಉದ್ದೇಶ ಮತ್ತು ಕಾರ್ಯವಿಧಾನದ ಅಪಾಯಗಳು ಮತ್ತು ತೊಡಕುಗಳ ಬಗ್ಗೆ. ಈ ಮೂಲಕ ಯಾವುದೇ ಬಲ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ಅಥವಾ ಯಾವುದೇ ಇತರ ಕಾರ್ಯವಿಧಾನಕ್ಕೆ ನನ್ನ ಮಾನ್ಯ ಲಿಖಿತ ತಿಳುವಳಿಕೆಯನ್ನು ನೀಡುತ್ತೇನೆ. ಫಿಟ್, ಇದು ಡಯಾಗ್ನೋಸ್ಟಿಕ್ & / ಅಥವಾ ಚಿಕಿತ್ಸಕ ವಿಧಾನ / ಬಯಾಪ್ಸಿ / ಟ್ರಾನ್ಸ್‌ಫ್ಯೂಷನ್ / ಆಪರೇಷನ್ ಅನ್ನು ಯಾವುದೇ ಅರಿವಳಿಕೆ ಅಡಿಯಲ್ಲಿ ಫಿಟ್ ಎಂದು ಪರಿಗಣಿಸಲಾಗಿದೆ. ಕಾರ್ಯವಿಧಾನದಲ್ಲಿ ಒಳಗೊಂಡಿರುವ ಸ್ವಭಾವ ಮತ್ತು ಅಪಾಯಗಳು (ಶಸ್ತ್ರಚಿಕಿತ್ಸೆ ಮತ್ತು ಅರಿವಳಿಕೆ) ನನ್ನ ತೃಪ್ತಿಗೆ ನನಗೆ ವಿವರಿಸಲಾಗಿದೆ.

ಕುತ್ತಿಗೆ ಮತ್ತು ವಿಕಿರಣ ಚಿಕಿತ್ಸೆ ನಂತರ ಕ್ಯಾರೊಟಿಡ್ ಅಪಧಮನಿ ಮತ್ತು ಆಂತರಿಕ ಜುಗ್ಲೂಲಾರ್ ಧಮನಿಯ ಕ್ಷಮತೆ ಮತ್ತು ತಲೆ ಮತ್ತು ಕುತ್ತಿಗೆ ಕ್ಯಾನ್ಸರ್- ಎ ನಿರೀಕ್ಷಿತ ಅಧ್ಯಯನ ಕುರಿತು ಕ್ಲಿನಿಕಲ್ ಸಂಶೋಧನೆಯ ಕುರಿತು ನನಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ - ಒಂದು ನಿರೀಕ್ಷಿತ ವೀಕ್ಷಣಾ ಅಧ್ಯಯನ.. ನಾನು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಲು ರೋಗಿಯ ಮಾಹಿತಿಯ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ . ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವವನಾಗಿ ಭಾಗವಹಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸುತ್ತೇನೆ. ನನ್ನ ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ಅಗತ್ಯವಿರುವ ತನಿಖೆಗಳು ಮತ್ತು ಶಸ್ತ್ರಚಿಕಿತ್ಸಾ ವಿಧಾನಗಳಿಗೆ ಒಳಗಾಗಲು ನಾನು ಈ ಮೂಲಕ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳು ಇತ್ಯಾದಿಗಳನ್ನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳಿಗೆ ಒದಗಿಸುತ್ತೇನೆ. ಶೈಕ್ಷಣಿಕ ಮತ್ತು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಕಾರ್ಯಾಚರಣೆ / ಕಾರ್ಯವಿಧಾನ ಇತ್ಯಾದಿಗಳನ್ನು ವೀಡಿಯೊ ಗ್ರಾಫ್ ಮಾಡಬಹುದು ಅಥವಾ ಛಾಯಾಚಿತ್ರ. ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಬಳಸಬಹುದು.

ಈ ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆ ನಮೂನೆಯ ಪ್ರತಿಯನ್ನು ಮತ್ತು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಭಾಗವಹಿಸುವವರಿಗೆ ಒದಗಿಸಲಾಗಿದೆ.

ಪಂ.ನ ಸಹಿ ಮತ್ತು ಹೆಸರು. ಪರಿಚಾರಕ ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತು ಮತ್ತು ರೋಗಿಯ ಹೆಸರು

ರೋಗಿಯೊಂದಿಗೆ ಸಂಬಂಧ: _____

ಸಾಕ್ಷಿ: _____

ಸಮ್ಮತಿಯನ್ನು ತೆಗೆದುಕೊಳ್ಳುವ ಸಂಶೋಧಕರ ಹೆಸರನ್ನು ಮುದ್ರಿಸಿ _____

ಒಪ್ಪಿಗೆಯನ್ನು ತೆಗೆದುಕೊಳ್ಳುವ ಸಂಶೋಧಕರ ಸಹಿ _____

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು: ಡಾ ಗೋಕುಲ್ ಪಿ ಎಂ

ಮೊಬೈಲ್ ಸಂಖ್ಯೆ:8870087252 ಇಮೇಲ್ ಐಡಿ: pmgokul97@gmail.com

PROFORMA

NAME
AGE
GENDER
ADDRESS
UHID NUMBER
COMPLAINTS
HISTORY OF PRESENTING ILLNESS DURATION OF SYMPTOMS OCCUPATION AND HABITS OTHER COMPLAINTS
PRE EXISTING SYTEMIC ILLNESS DIABETES ☐ THYROID DISORDER ☐ TUBERCULOSIS ☐ ANAEMIA ☐ HYPERTENSION ☐ OTHERS, SPECIFY ☐
DIAGNOSIS -
SURGERY PERFORMED:
DATE OF SURGERY:

INVESTIGATION MODALITY

CAROTID DOPPLER ULTRASOUND-PREOPERATTIVE

- Both common carotid arteries -
- Both Internal jugular vein-
- Intimo-medial thickness measures in common carotid artery:
- Right- ~ mm.
  Left- ~ mm
- Intimo medial thickness measures in internal jugular vein-
  Right- mm
  Left- mm
  - Both ijv show normal waveforms
  - IJV-patency-
  - Caliber-
  - Flow-

RIGHT			**LEFT**		
CCA- PSV (cm/s)			CCA- PSV (cm/s)		
ICA (cm/s)	PSV		ICA (cm/s)	PSV	
	EDV			EDV	
ICA/CCA (PSV ratio)			ICA/CCA (PSV ratio)		

INVESTIGATION MODALITY

CAROTID DOPPLER ULTRASOUND-POSTOPERATTIVE

DATE OF SURGERY-

- Both common carotid arteries -
- Both Internal jugular vein-
- Intimo-medial thickness measures in common carotid artery:
- Right- ~ mm.
  Left- ~ mm
- Intimo medial thickness measures in internal jugular vein-
  Right- mm
  Left- mm
  - Both ijv show normal waveforms
  - IJV-patency-
  - Caliber-
  - Flow-

RIGHT			**LEFT**		
CCA- PSV (cm/s)			CCA- PSV (cm/s)		
	PSV			PSV	

		EDV		ICA (cm/s)	EDV		
	ICA/CCA (PSV ratio)			ICA/CCA (PSV ratio)			

INVESTIGATION MODALITY

CAROTID DOPPLER ULTRASOUND-POST RADIOTHERAPY

DATE OF RADIATION –

DOSE OF RADIATION-

FRACTIONS OF RADIATION RECEIVED-

- Both common carotid arteries -
- Both Internal jugular vein-
- Intimo-medial thickness measures in common carotid artery:
- Right- ~ mm.
  Left- ~ mm
- Intimo medial thickness measures in internal jugular vein-
  Right- mm
  Left- mm
  - Both ijv show normal waveforms
  - IJV-patency-
  - Caliber-
  - Flow-

RIGHT			**LEFT**		
CCA- PSV (cm/s)			CCA- PSV (cm/s)		
ICA (cm/s)	PSV		ICA (cm/s)	PSV	
	EDV			EDV	
ICA/CCA (PSV ratio)			ICA/CCA (PSV ratio)		

MASTER CHART

NUMBER	AGE (YEARS)	GENDER	CHIEF COMPLAINTS	COMORBIDITIES	DIAGNOSIS	SURGERY PERFORMED	ADJUVANT TREATMENT	DOSAGE OF RT	NO OF FRACTION	preop				POSTOP				POST RT				IMT(PREOP)		IMT(postop)		IMT(POST RT)	
										CCA-PSV	ICA-PSV	ICA-EDV	PSV RATIO	CCA-PSV	ICA-PSV	ICA-EDV	PSV RATIO	CCA-PSV	ICA PSV	ICA EDV	PSV RATIO	RIGHT	LEFT	RIGHT	LEFT	RIGHT	LEFT
1	65	FEMALE	ULCER IN THE INNER ASPECT OF LEFT CHEEK	NIL	squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	64Gy	32	98	78	29	0.8	98	78	29	0.8	103	78	24	0.7	0.5	0.6	0.6	0.5	0.6	0.5
2	58	FEMALE	ULCER IN INNER ASPECT OF RIGHT CHEEK	NIL	squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	60Gy	30	82	57	29	0.6	90	72	29	0.8	94	62	28	0.6	0.4	0.5	0.5	0.6	0.5	0.6
3	50	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	66Gy	32	89	60.7	27	0.8	97	68	27	0.7	95	68	27	0.7	0.6	0.4	0.5	0.4	0.4	0.5
4	70	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	56Gy	28	92	62	19	0.6	93	66	19	0.6	99	71	23	0.7	0.6	0.4	0.7	0.6	0.8	0.9
5	52	FEMALE	ULCER IN INNER ASPECT OF RIGHT CHEEK	HTNx5 YEARS	squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	60Gy	30	91	67	23	0.6	89	68	21	0.7	93	71	22	0.7	0.4	0.5	0.6	0.5	0.6	0.5
6	60	FEMALE	ULCER IN INNER ASPECT OF RIGHT CHEEK	NIL	squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	64Gy	32	88	78	19	0.8	91	82	21	0.9	98	79	23	0.8	0.6	0.8	0.5	0.7	0.6	0.8
7	45	MALE	ULCER IN THE RIGHT LATERAL BORDER OF TONGUE	NIL	squamous cell carcinoma RIGHT BUCCAL LATERAL TONGUE	MRND (Right)	RT	66Gy	33	80.6	64	24.7	0.7	85	48.7	13	0.5	89	56	21	0.6	0.7	0.9	0.8	1.4	0.8	1.2
8	45	MALE	ULCER IN INNER ASPECT OF RIGHT CHEEK	hpl	squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	64Gy	32	100	74	19	0.7	73	65	27	0.8	97	77	24	0.7	0.5	0.6	0.6	0.6	0.7	0.8
9	58	FEMALE	ULCER IN INNER ASPECT OF RIGHT CHEEK	hpl	squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	66Gy	33	93	78	24	0.8	96	77	23	0.8	99	78	24	0.7	0.3	0.4	0.3	0.4	0.4	0.4
10	54	FEMALE	ULCER IN INNER ASPECT OF RIGHT CHEEK	hpl	squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	64Gy	32	115	84	27	0.7	109	78	30	0.8	104	77	29	0.7	0.5	0.6	0.5	0.6	0.5	0.6
11	39	FEMALE	ULCER AND PAIN IN INNER ASPECT OF RIGHT CHEEK	NEWLY DIAGNOSED DM	squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	56Gy	28	128	78	17	0.4	117	77	23	0.6	108	81	31	0.6	0.3	0.4	0.3	0.4	0.4	0.4
12	72	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	SQUAMOUS CELL squamous cell carcinoma LEFT LOWER GBS	MRND (Left)	RT	64Gy	32	93	61	16	0.6	89	74	25	0.8	99	76	23	0.7	0.5	0.6	0.6	0.7	0.7	0.8
13	74	FEMALE	ULCER IN INNER APECT OF LEFT CHEEK	NIL	VERUCOUS squamous cell carcinoma LEFT GBS EXTENDING TO HARD PALATE	MRND (Left)	CT+RT	66Gy	33	98	78	29	0.8	96	72	23	0.7	102	77	21	0.7	0.6	0.6	0.6	0.6	0.5	0.6
14	63	FEMALE	ULCER IN INNER ASPECT OF RIGHT CHEEK	hpl	squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	56gy	28	94	69	23	0.7	90	64	21	0.7	99	72	23	0.7	0.4	0.5	0.5	0.6	0.5	0.6
15	83	MALE	ULCER IN LEFT SIDE OF TONGUE	hpl	METASTASIS OF UNKNOWN ORIGIN	MRND RIGHT	RT	56Gy	28	83	68	19	0.8	87	71	19	0.8	93	73	19	0.7	0.3	0.4	0.3	0.4	0.45	0.5
16	61	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	SQAMOUS CELL squamous cell carcinoma LEFT BUCCAL MUCOSA EXTENDING TO RMT	MRND (Left)	RT	64gy	32	100	76	19	0.7	98	77	21	0.7	108	79	19	0.7	0.4	0.5	0.5	0.6	0.7	0.8

17	53	FEMALE	ULCER IN INNER ASPECT OF RIGHT CHEEK	NIL	SQUAMOUS CELL squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	66gy	33	79	57	21	0.7	83	59	24	0.7	91	63	21	0.6	0.5	0.5	0.4	0.5	0.5	0.5
18	62	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	64gy	32	98	78	29	0.8	92	73	29	0.8	88	71	19	0.8	0.3	0.4	0.3	0.4	0.4	0.4
19	48	FEMALE	ULCER IN INNER ASCPECT OF LEFT CHEEK	NIL	SQAMOUS CELL squamous cell carcinoma OF LEFT BUCCAL MUCOSA	MRND (Left)	RT	66Gy	33	101	76	18	0.7	79	67	27	0.8	97	77	21	0.7	0.5	0.6	0.7	0.6	0.7	0.8
20	53	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	POST OP CASE OF RIGHT BUCCAL MUCOSA WITH SECOND PRIMARY IN LEFT BUCCAL MUCOSA INVOLVING LEFT LOWE GBS	MRND (Left)	CT+RT	56gy	28	108	78	30	0.7	115	84	27	0.7	99	78	29	0.8	0.6	0.6	0.5	0.6	0.6	0.6
21	65	MALE	ULCER INDER THE TONGUE	DM SINCE 4 YEARS	SQAMOUSE CELL FLOOR OF MOUTH	MRND LEFT	RT	64Gy	32	93	78	24	0.8	96	77	23	0.8	99	77	23	0.7	0.4	0.5	0.4	0.5	0.5	0.5
22	74	MALE	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	SQAMOUS CELL squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	64Gy	32	83	68	19	0.8	92	69	23	0.7	96	64	21	0.6	0.4	0.5	0.4	0.5	0.5	0.5
23	65	FEMALE	ULCER IN TIP OF TONGUE	NIL	squamous cell carcinoma TIP OF TONGUE	MRND RIGHT	RT	52Gy	26	90	73	29	0.8	98	78	29	0.8	93	73	22	0.7	0.3	0.4	0.3	0.4	0.3	0.4
24	40	MALE	ULCER IN INNER ASPECT OF RIGHT CHEEK	newly diagnosed hypothyrodism	squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	64Gy	32	97	78	29	0.8	92	73	28	0.8	96	71	24	0.7	0.4	0.4	0.4	0.5	0.5	0.5
25	67	MALE	ULCER IN INNER ASPECT OF RIGHT alveolus	NIL	SQAMOUS CELL squamous cell carcinoma RIGHT UPPER ALVEOLUS	MRND (Right)	RT	66Gy	33	83	69	19	0.8	88	71	23	0.7	88	78	23	0.8	0.4	0.4	0.4	0.4	0.4	0.4
26	38	FEMALE	ULCER IN INNER ASPECT OF RIGHT CHEEK	NIL	SQUAMOUS CELL squamous cell carcinoma RIGHT LOWER GBS	MRND (Right)	RT	64gy	32	95	60	14	0.6	100	74	19	0.7	102	77	21	0.7	0.3	0.4	0.4	0.4	0.5	0.5
27	54	FEMALE	ULCER IN INNER ASPECT OF RIGHT CHEEK	NIL	SQUAMOUS CELL squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	64Gy	32	99	78	28	0.7	92	77	23	0.8	96	69	24	0.7	0.4	0.4	0.4	0.5	0.5	0.5
28	58	MALE	ULCER IN RIGHT SIDE OF TONGUE	DM SINCE 6 YEARS	SQAMOUS CELL squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	56Gy	28	94	69	23	0.7	89	67	21	0.7	96	71	18	0.7	0.4	0.6	0.4	0.6	0.4	0.6
29	68	FEMALE	ULCER IN INNER ASPECT OF RIGHT CHEEK	NIL	SQAMOUS CELL squamous cell carcinoma RIGHT LOWER ALVEOLUS	MRND (Right)	RT	66GY	33	94	74	21	0.7	96	63	19	0.6	103	69	21	0.68	0.5	0.6	0.6	0.6	0.6	0.7
30	52	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	HTNx5 YEARS	POST OP CASE OF LEFT BUCCAL MUCOSA WITH LOCAL RECCURENCE	MRND (Left)	CT+RT	64Gy	32	100	73	19	0.7	95	62	14	0.6	98	71	24	0.7	0.3	0.4	0.4	0.5	0.5	0.6
31	55	MALE	ULCER IN LEFT SIDE OF TONGUE	NIL	SQAMOUS CELL squamous cell carcinoma LEFT LATERAL BORDER OF TONGUE WITH LEUKOPLAKIA OF LEFT BUCCAL MUCOSA	MRND (Left)	RT	64Gy	32	97	78	29	0.8	94	77	21	0.7	98	78	23	0.7	0.5	0.5	0.5	0.5	0.5	0.5
32	34	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	64gy	32	95	66	17	0.6	97	69	23	0.7	107	77	28	0.7	0.4	0.5	0.5	0.6	0.7	0.8
33	58	FEMALE	ULCER IN INNER ASPECT OF CHEEK	htn	SQAMOUS CELL squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	66Gy	33	98	78	29	0.8	98	73	28	0.8	99	79	26	0.7	0.3	0.4	0.3	0.4	0.3	0.4
34	37	MALE	ULCER IN RIGHT SIDE OF TONGUE	NIL	SQAMOUS CELL squamous cell carcinoma RIGHT LATERAL BORDER OF TONGUE	MRND (Right)	RT	64Gy	32	83	59	27	0.6	90	87	26	0.9	99	81	25	0.8	0.4	0.5	0.5	0.6	0.6	0.6
35	60	MALE	ULCER IN LEFT SIDE CHEEK	NIL	squamous cell carcinoma TRANSGLOTTIS	MRND RIGHT	CT+RT	60Gy	30	98	78	29	0.8	101	64	21	0.6	112	72	29	0.6	0.4	0.5	0.6	0.7	0.7	0.8
36	59	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	DMx2 years,BA x2 years	SQAMOUS CELL squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	64Gy	32	80	68	22	0.8	87	66	18	0.7	99	79	28	0.7	0.5	0.6	0.5	0.6	0.7	0.8
37	49	female	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	66Gy	33	115	84	27	0.7	109	78	30	0.7	117	83	29	0.7	0.6	0.7	0.7	0.7	0.8	0.9
38	69	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	SQAMOUS CELL squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	56Gy	28	95	60	14	0.6	79	57	21	0.7	94	68	24	0.7	0.4	0.6	0.6	0.7	0.8	0.8
39	37	male	ULCER IN LEFT SIDE OF TONGUE	HTN	squamous cell carcinoma LEFT LATERAL BORDER OF TONGUE POST 4 CYCLE NACT	MRND (Left)	RT	64gy	32	89	55	18.3	0.6	85	70	14	0.8	92	74	16	0.8	0.5	0.6	0.6	0.6	0.7	0.8
40	41	female	ULCER IN INNER ASPECT OF CHEEK AND PAIN	NIL	squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	66gy	33	80	73	22.3	0.9	101	64	20.4	0.6	96	79	17	0.8	0.3	0.4	0.4	0.4	0.4	0.4

41	56	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK AND PAIN	NIL	SQAMOUS CELL squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	64gy	32	87	57	15	0.6	107	76	14	0.7	99	77	21	0.8	0.4	0.5	0.4	0.5	0.5	0.6
42	40	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	HTN	SQAMOUS CELL squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	66gy	33	94	69	23	0.7	97	73	17	0.7	99	77	21	0.7	0.3	0.4	0.3	0.4	0.3	0.4
43	42	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	squamous cell carcinoma TONGUE AND BUCCAL MUCOSA	MRND LEFT	RT	66gy	32	84	74	25	0.8	93	61	16	0.6	98	68	27	0.7	0.5	0.5	0.5	0.6	0.6	0.6
44	41	male	ULCER IN INNER ASPECT OF LEFT CHEEK	NEWLY DIAGNOSED DM	SQUAMOUS CELL squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	64GY	32	89	77	23	0.8	88	74	23	0.7	89	71	22	0.7	0.4	0.5	0.4	0.5	0.4	0.5
45	42	MALE	WOUND IN INNER ASPECT OF LEFT CHEEK	NIL	SQUAMOUS CELL squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	66GY	33	91	67	21	0.8	90	66	18	0.8	89	71	21	0.8	0.3	0.4	0.3	0.4	0.4	0.4
46	43	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	SQAMOUS CELL squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	56GY	28	87	71	19	0.8	89	76	19	0.8	92	74	19	0.8	0.5	0.5	0.5	0.6	0.6	0.7
47	58	FEMALE	ulcer in inner aspect of left cheek	NIL	SQAMOUS CELL squamous cell carcinoma LEFT LOWER ALVEOLUS	MRND (Left)	RT	64Gy	32	83	68	19	0.8	94	69	23	0.7	96	81	22	0.8	0.4	0.45	0.4	0.4	0.4	0.4
48	54	female	ULCER in left side of neck	nil	POST OP CASE OF RIGHT BUCCAL MUCOSA WITH RECURRENCE	MRND (Right)	RT	60GY	30	107	78	30	0.7	103	77	26	0.7	111	79	23	0.7	0.5	0.6	0.6	0.6	0.7	0.7
49	41	FEMALE	ULCER OVER INNER ASPECT OF RIGHT CHEEK	NIL	SQAMOUS CELL squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	56GY	28	83	61	26	0.7	89	74	24	0.8	92	77	26	0.8	0.5	0.6	0.6	0.6	0.6	0.7
50	52	FEMALE	ULCER IN INNER ASPECT OF RIGHT CHEEK AND PAIN	NIL	POST OP CASE OF squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	64GY	32	91	67	21	0.8	90	66	18	0.8	89	71	22	0.7	0.3	0.4	0.4	0.4	0.3	0.4
51	72	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	HTNx5 YEARS	SQAMOUS CELL squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	60GY	30	80	68	22	0.8	87	66	18	0.7	91	79	24	0.7	0.4	0.5	0.5	0.5	0.5	0.6
52	51	female	ULCER IN INNER ASPECT OF LEFT CHEEK	newly diagnosed HTN	squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	64GY	32	84	74	25	0.8	93	61	18	0.7	97	77	23	0.7	0.5	0.6	0.6	0.7	0.7	0.7
53	54	FEMALE	ULCER IN INNER ASPECT OF LEFT CHEEK	NIL	METASTASIS OF UNKOWN ORIGINNWITH ERTHROPLAKIA OF LEFT BUCCAL MUCOSA	MRND (Left)	RT	64GY	32	90	62	19	0.6	89	66	20	0.7	91	71	21	0.7	0.5	0.6	0.6	0.6	0.6	0.7
54	62	MALE	ULCER IN LEFT CHEEK	NIL	squamous cell carcinoma TRANSGLOTTIS,POST CT+RT WITH RECURRENCE OVER LEFT VOCAL CORD	MRND (Left)	RT	60GY	30	91	77	21	0.8	90	74	20	0.8	92	76	23	0.8	0.3	0.4	0.4	0.5	0.5	0.5
55	52	FEMALE	ULCER OVER INNER ASPECT OF RIGHT CHEEK	nil	SQUAMOUS CELL squamous cell carcinoma RIGHT RETROMOLAR TRIGONE	MRND (Right)	RT	56GY	28	97	67	23	0.6	94	64	22	0.6	96	67	19	0.6	0.4	0.4	0.4	0.5	0.4	0.4
56	56	FEMALE	ULCER INNER ASPECT OF LEFT CHEEK	NEWLY DIAGNOSED DM,hpl	SQUAMOUS CELL squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	60GY	30	101	82	24	0.8	99	83	23	0.8	114	87	22	0.7	0.5	0.6	0.6	0.7	0.7	0.8
57	75	male	ULCER IN LEFT SIDE CHEEK	NEWLY DIAGNOSED HTN,hpl	SQUAMOUS CELL squamous cell carcinoma LEFT PYRIFORM FOSSA	MRND (Left)	RT	66GY	33	142	122	23.8	0.8	139	114	23	0.8	141	116	22	0.8	0.7	0.9	0.6	0.8	0.7	0.9
58	74	MALE	ULCER OVER INNER ASPECT OF LEFT CHEEK	Bronchial asthma and HTN	squamous cell carcinoma LEFT BUCCAL MUCOSA	MRND (Left)	RT	64GY	32	91	76	21	0.8	92	79	19	0.8	89	71	19	0.7	0.3	0.3	0.3	0.4	0.3	0.3
59	74	FEMALE	ULCER OVER INNER ASPECT OF RIGHT CHEEK	K/C/O HTN SINCE 15 YRS	SQAMOUS CELL squamous cell carcinoma RIGHT LOWER GBS	MRND (Right)	RT	60GY	30	84	74	25	0.8	89	76	24	0.8	91	79	26	0.8	0.4	0.5	0.4	0.5	0.5	0.5
60	45	FEMALE	ULCER OVER INNER ASPECT OF RIGHT CHEEK	NIL	squamous cell carcinoma RIGHT BUCCAL MUCOSA	MRND (Right)	RT	56GY	28	99	81	22.6	0.8	96	79	21	0.8	101	86	23	0.8	0.5	0.6	0.6	0.6	0.6	0.7