Otolaryngology: Open Access
ISSN: 2161-119X

Mini Review Open Access

Head and Neck Pathology: Recent Advances in Head and Neck Tumor Diagnosis and Pathogenesis

Sohail Awan*

Department of Otolaryngology, King Edward Medical University, Pakistan

Abstract

A sizable and diverse set of tumours with a very varied prognosis make up head and neck malignancies, These tumours include lesions that develop from other anatomical sites, such as the salivary glands, the mucosa of respiratory sinuses, the thyroid, skin, and orbit, in addition to those that originate from the squamous epithelial lining of the oral cavity, oropharynx, nasopharynx, hypopharynx, and larynx. The clinic-pathological chapter occasionally includes information on brain tumours as well. Manuscripts that touch on some of the most recent advancements in the pathophysiology and diagnosis of head and neck malignancies are featured in this issue. The first article reviews our knowledge of how oral squamous cell cancer develops and explores potential chemoprevention measures. As was mentioned above, smoking can have an impact on the entire upper aero digestive tract, which can limit the identification of head and neck cancer. Spectroscopy is used to identify malignant squamous mucosa; debate.

Smoking-related cancers, such as head and neck squamous cell carcinoma, are becoming less common as the prevalence of smoking declines in various regions of the world. Unfortunately, this drop has coincided with an increase in oropharyngeal squamous cell carcinomas linked to high-risk human papillomavirus (HPV) infection in many parts of the world. In this issue, you will demonstrate through the use of a case-control study that oropharyngeal squamous cell carcinomas that develop in non-smokers who do not abuse alcohol are more likely to be linked to HPV infection than those that develop in smokers who do and that the tumours linked to HPV have a better prognosis. Review the different molecular, in situ hybridization, and immunohistochemistry test that can be used to prove HPV infection.

The pathology of Sinonasal intestinal-type adenocarcinomas, which are occasionally linked to significant exposure to wood dust, is still unknown. Reviewing a sizable collection of these tumours and looking for potential precursor lesions for these uncommon malignancies are the goals of the final article in this issue.

Keywords: Head and neck; Risk factors; Cancer; Molecular genetics; Sarcomasoft-tissue neoplasms

Introduction

The term "head and neck cancer" (HNC) refers to tumours of the aero alimentary tract, including the lip, oral cavity, nasal cavity, Para nasal sinuses, pharynx, larynx, oropharynx, hypo pharynx, salivary glands, and local lymph nodes. It is the sixth most prevalent cancer in the world. Squamous cell carcinomas (HNSCCs), which develop from the mucosal lining in these areas, account for 90% of all HNCs. While 30-50% of these have been linked to the human papillomavirus (HPV), with type 16 being the most frequently found type in HNSCC, 80-90% of these have been linked to chronic alcohol and tobacco use [1]. There is a large geographic variation in the incidence of this disease, with South-East Asia, the Pacific regions, Latin America, and some parts of Central and Eastern Europe showing higher incidences than other regions. For example, HNSCC is the most prevalent cancer type in India, making up 40% of all malignancies. With a poor quality of life for survivors, the 5-year survival rate of smoking-related HNSCC is still 30–50%. HNSCC may not exhibit clinical signs in the early stages of the disease, hence it is typically not discovered. Therefore, techniques for early identification and diagnosis of lesions with a high potential for malignancy, preventative measures, and novel therapeutic techniques are essential for enhancing treatment outcomes and patients' quality of life [2].

Small, single-stranded RNA molecules known as miRs were initially identified in 1993. They have been proven to affect Caenorhabditis elegans worm larval development by controlling translation through an antisense RNA-RNA interaction. The miRBAse database showed 1600 Homo sapiens miRs in June 2013. Despite being non-coding, they are thought to affect the expression of a large number of protein-coding genes in the human genome. Organ development, cell differentiation, proliferation, apoptosis, and stress responses are all influenced by miRs, which are connected to the translation and degradation of mRNA. One miR can affect the mRNA transcripts of many different genes, but several miRs can target the same mRNA, implicating them in the development of tumours by altering the cellular levels of particular oncogenes or tumour suppressor genes [3].

RNA polymerase II (RNA Pol II) initiates the transcription of miRs, generating primary miRs (pri-miRs), which are then transformed into precursor miRs (pre-miRs) by Drosha, an RNAase III endonuclease, and DiGeorge syndrome critical area gene 8. (DGCR8). Exportin 5 transports pre-miRs (70–100 nt long) to the cytoplasm, where they are broken down by the RNAase III enzyme Dicer and its partner TRBP to create double-stranded (ds) RNA, which is about 22 nt long. The mature miR and the complementary strand (miR*) make up this dsRNA. Although miRs* are typically destroyed, they occasionally function. By base-pairing to partially complementary areas, mature miRs can control the translation of protein-coding mRNAs. It can identify certain sequences, and by degrading the target mRNA and

***Corresponding author:** Sohail Awan, Department of Otolaryngology, King Edward Medical University, Pakistan; Tel: 927518453932; E-mail: Sohail.awan81@gmail.com

Received: 28-Sep-2022, Manuscript No: ocr-22-76691, **Editor Assigned:** 01-Oct-2022, Pre QC No: ocr-22-76691(PQ), **Reviewed:** 15-Oct-2022, QC No: ocr-22-76691, **Revised:** 22-Oct-2022, Manuscript No: ocr-22-76691(R), **Published:** 31-Oct-2022, DOI: 10.4172/2161-119X.1000489

Citation: Awan S (2022) Head and Neck Pathology: Recent Advances in Head and Neck Tumor Diagnosis and Pathogenesis. Otolaryngol (Sunnyvale) 12: 489.

suppressing its translation, it uses the proteins Argonaute 2 (Ago2), TRBP, and RNA-inducing silencing complex (RISC; a multiprotein complex). Additionally, it has been discovered that miRs interfere with RNA binding activities (decoy activity) without the need for RISC [4].

Cellular factors, such as c-Myc (an oncogenic protein that induces the expression of oncomirs), p53 (a tumour suppressor protein that induces the expression of tumour suppressor miRs), and E2F, or flaws in the miR biogenesis machinery, control miR expression at the transcriptional and posttranscriptional levels (Drosha, DGCR8, exportin 5, Dicer, TRBP, and Ago2). Single nucleotide polymorphisms (SNPs) in pri- and pre-miRs as well as in miR biogenesis pathway genes, loss or gain of chromosomal material since miR encoding genes are frequently located in fragile regions, altered DNA methylation, or histone deacetylase inhibition that results in reduced or lost expression are all possible causes of changes in miR expression in cancer (OSCC), or the dysregulation of important miR biogenesis-related genes [5].

Despite the fact that 31.4% of potentially malignant oral lesions progress to OSCC, clinical and histological characteristics are not helpful in the early identification of HNSCC and have limited promise as predictors of transformation. A diagnostic problem is posed by the fact that up to 50% of HNSCCs may develop from mucosa that seems to be clinically normal. Epithelial dysplasia and potentially malignant oral lesions (the most prevalent of which are leukoplakia and erythroplakia) are statistically more likely to develop into cancer, but the actual mechanisms by which this occurs are still poorly understood, and it is not always the case that a dysplastic lesion will turn malignant [6].

Therefore, the prognosis is poor upon clinical diagnosis of HNSCC and disease staging is frequently advanced. The histopathological analysis of tissue biopsies can be interpreted in a wide variety of ways because it is subjective. Similar to this, it is impossible to determine which dysplastic lesions are more likely to develop into cancer over time using concrete, verified criteria. The presence of dysplasia can only be used to suggest that an oral lesion may have a higher probability of developing into a malignant transformation given the state of scientific knowledge at this time. The death rate for individuals with HNSCC is still high, and current treatment modalities are still linked to a number of negative side effects and a poor quality of life. This is true despite the significant advancements in diagnostic technology and therapeutics over the past three decades [7].

Methods and Materials

This investigation was prospective and cross-sectional. Between April 2015 and December 2017, a total of 18 individuals with HNCs who experienced shoulder discomfort and dysfunction following SND were included. Each patient's diagnosis, localization, and stage of HNCs were established based on their medical history. In order to conduct this study, we gathered patients with HNCs who had had SND, were in stable health, had skin that had fully healed, and did not have any metastases to the neck or shoulder. The institutional review board gave their clearance to this study. All participants provided their written informed permission after being enrolled [8].

Discussion

It's crucial to think about how this patient's fracture developed, why it led to non-union and pseudarthrosis, and how it was treated. Taller neck injuries typically occur as a result of high-energy trauma, which causes the foot and talus to hyperdorsiflex, according to the usual process. As a result, the anterior lip of the tibial plafond is impinged upon by the talus's neck, and additional forced dorsiflexion beyond this point causes failure at the higher neck due to a bending moment about the anterior tibial plafond, which serves as a fulcrum. Taller dorsiflexion could not occur in this case, indicating a distinct difference in the mechanism. There are various mechanisms that could have caused this fracture. First, the impact may have resulted in a bending moment on the tibiotalar fusion due to the midfoot and forefoot dorsiflexion. Axial compression that travels through the calcaneus is the other option. Since the calcaneus did not shatter, the stress may have been transmitted as shear to the higher neck by way of the calcaneus' anterior process or posterior subtler facet [9].

Once ankle movement was eliminated, the taller neck was subjected to its maximum amount of stress, resulting in six taller neck fractures over the course of nine testing. Another intriguing study examined the impact of Achilles strain on the mechanism of fracture in healthy ankles. Both an external force and internal muscle tension applied through the Achilles tendon during preimpact bracing can apply axial loading to the leg. Despite the fact that calcaneus fractures were the most frequent overall fracture, they discovered that increasing Achilles strain increased the risk of pilon fractures [10]. As a result of the patient's displaced injury in their case, open reduction and internal fixation were performed. In this instance, there were 24 years between ankle fusion and the patient's injury, giving the patient more than enough time to load and rebuild the bone. Additionally, there were no indications of osteoporosis on his photos. A stress riser had been removed from this patient's joint five years prior to the injury owing to metalwork prominence, therefore neither metalwork nor a stress riser were present at the time of the injury. This patient was at a significant risk of having a non-union since the likely diagnosis of an undisplaced fracture at the time of the event was overlooked [11].

The fracture was not properly immobilised, to start. Second, the blood flow to the fracture would probably be hampered by iatrogenic microvascular damage from the surgery and the vasculopathy of diabetes. Thirdly, there would be additional movement at the fracture site due to the fused ankle joint. Fourth, despite the presence of the normal protective sense, there may have been some minor diabetic neuropathy (notice the higher HbA1C). These variables are probably what caused the painful non-union or pseudarthrosis to form. Three screws were used to treat this case in order to compress both the higher neck and the subtler joint. In the presence of prior surgeries and scars, using a single lateral approach lowered the incidence of wound complications. Since there was no immature fusion to be preserved, the authors were spared the technical issue that Kwon and Myerson had in their situation [12].

Conclusions

It has not yet been documented that a higher neck fracture can occur years following tibiotalar fusion. The axial compression of the calcaneus, which results in shear force across the taller neck, is the most likely cause. This is a completely distinct process from the typical higher neck fracture brought on by excessive talus dorsiflexion. Due to preferred mobility at the fracture site and the already impaired blood supply to the talus following surgery, the fracture has a significant probability of failing to heal. Therefore, it is crucial to suspect this fracture in any patient who presents with foot and ankle injuries and a unified tibiotalar fusion.

After neck dissection, spinal accessory nerve (SAN) damage is a prevalent complication in patients with head and neck malignancy (HNCs). In the past, radical neck dissection has been the go-to surgical procedure for patients with HNCs that have metastasized to the neck

lymph nodes. However, this method resulted in a total SAN injury, which is connected to a lot of ipsilateral shoulder pain and dysfunction and may have a bad effect on one's quality of life. Due to the potential topographical tumour subsites-lymph node relationship, the nerve-sparing technique of selective neck dissection (SND) was created in order to preserve the SAN, a more selective operation, and it has since grown in popularity among patients with HNCs who have no or limited lymph node metastasis (N0) (N1). Nevertheless, after SND, the accessory nerve was observed to have been injured in up to 67% of patients.

Conflict of Interest

None

Acknowledgement

None

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