

Research Article

Study of Somatic EGFR Mutations in Squamous Cell Carcinomas of Head and Neck

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Article Info

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Abstract

Background and Aim: Head and neck squamous cell carcinomas (HNSCC) originate from the epithelial lining of the upper aerodigestive tract. It accounts for over 90 % of the malignancies found in the head and neck region. One of the vital receptor that controls key signaling pathways for cell proliferation and survival is epidermal growth factor receptor (EGFR). The present study aimed to study the somatic EGFR mutations in squamous cell carcinomas of head and neck.

Materials and methods: A Cross-sectional descriptive study was carried out on all cases of biopsy proven squamous cell carcinomas of head and neck presenting to a tertiary care hospital over a period of two years (July 2024- July 2026). Mutational analysis was done on 30 samples. The tissue samples were stored in saline at -40°C until analysis. Clinical data, including age, sex, tobacco/smoking history, duration of symptoms, and tumor site, were recorded. Histopathological evaluation was done which included TNM staging (AJCC 7th edition), tumor grading (Broders' criteria), lymphovascular invasion, perineural invasion, and extranodal extension.

Results: The study included 30 patients with head and neck squamous cell carcinoma (HNSCC) with a mean age of 58.2 ± 8.38 years; most patients were 51–60 years old (46.7%) and male (83.3%). The tongue (33.3%) was the most common tumor site, followed by the buccal mucosa (20%), and 50% of patients had a history of tobacco chewing. Most tumors were Stage I (36.7%) and well-differentiated (Grade I, 76.7%). Somatic EGFR mutations were detected in 2 patients (6.7%), both involving the tongue, while 93.3% of cases showed no detectable mutation. No significant association was observed between EGFR mutation status and age, tumor site, TNM stage, or tumor grade ($p > 0.05$).

Conclusion: Low frequency of somatic EGFR mutations (6.7%) in HNSCC was found with no significant association between EGFR mutation status and clinicopathological parameters such as TNM stage and tumor grade.

Keywords: Somatic EGFR Mutations, Squamous Cell Carcinomas, Head and Neck

Introduction

Squamous cell carcinoma is the most prevalent malignancy in the head and neck region. It is linked to a high incidence of morbidity and mortality.[1] The molecular heterogeneity associated with this entity, which involves multiple carcinogenesis pathways, was recently demonstrated in genomic studies.[2] The development of head and neck squamous cell carcinoma (HNSCC) is influenced by a variety of risk factors, including tobacco, alcohol, and viruses like the human papillomavirus (HPV).[3,4] Carcinogenesis is characterized by the self-sufficiency of growth signals. The transmembrane and cytoplasmic tyrosine kinase domain of the epidermal growth factor receptor (EGFR) are responsible for the regulation of downstream signaling pathways that are involved in cell division and survival. Various steps in carcinogenesis, such as cell division, cell migration, and angiogenesis, are regulated by downstream signaling pathways, including Mitogen-Activated Protein Kinase (MAPK), phosphatidylinositol-3-kinase (PI3K)-AKT, and Signal Transducer and Activator of Transcription (STAT).[5]

The EGFR protein was discovered to be overexpressed in approximately 84% of HNSCC.[6] This overexpression of EGFR may be precipitated by oncogenic viruses and epigenetic modifications. The EGFR Tyrosine Kinase (TK) domain mutations have been identified on chromosome 7, specifically in exons 18 to 21. These consist of a single base deletion, insertions, or substitutions. Solid organ tumors, such as non-small cell lung carcinoma, have been the subject of research regarding EGFR mutations. Patients with these mutations have demonstrated a favorable response to targeted therapy, which includes EGFR-tyrosine kinase inhibitors (TKIs) such as Gefitinib[7,8].

Studies on EGFR mutations in cases of HNSCC are limited. Therefore, a study of the presence of mutations in EGFR-TK in our population may improve therapies for these patients. Hence, the

present study was designed to investigate the presence of somatic EGFR mutations in head and neck squamous cell carcinomas.

Materials and Methods

After obtaining clearance from institutional ethical committee, Cross sectional descriptive study was carried out on all cases of biopsy proven squamous cell carcinomas of head and neck presenting to a tertiary care hospital over a period of two years (July 2024- July 2026). Mutational analysis was done on 30 samples.

Inclusion criteria

All consecutive cases of Head and Neck region-diagnosed as squamous cell carcinoma (HNSCC) and in which resection specimens of primary tumor were received at Department of Pathology, tertiary care hospital, starting from July 2024 upto July 2026 were compiled.

Exclusion Criteria

- Cases in which pre-operative radiotherapy and chemotherapy was given to the patient, were excluded from the study.
- Secondary metastatic tumours to the head and neck were not included.

Methodology

Fresh tumor tissue was collected from the resection specimens of 33 patients with head and neck squamous cell carcinoma (HNSCC) between July 2024 and July 2026. The tissue samples were stored in saline at -40°C until analysis. Clinical data, including age, sex, tobacco/smoking history, duration of symptoms and tumor site were recorded. Histopathological evaluation included TNM staging (AJCC 7th edition), tumor grading (Broders' criteria), lymphovascular invasion, perineural invasion, and extranodal extension. All data were entered into an Excel database. DNA was extracted from the fresh tissue samples, and its quality was assessed using NanoDrop spectrophotometry. Three samples were excluded

due to poor DNA quality, leaving 30 samples for further molecular analysis.

DNA isolation

Genomic DNA was isolated by using the Pure Link Genomic DNA Kit as per the kit protocol (Appendix C) and quantified using the nanodrop. DNA concentration of greater than 10 ng microlitre and A 260/280 ratio greater than 1.8 was considered adequate. (A 260/280 ratio less than 1.8 indicated more protein content)

The assay required approximately 80 ng of DNA for each sample per run (10ng/reaction). Lower concentrations were adjusted with the water volume during reaction setup. The DNA quantities of each sample were recorded.

The EGFR Mutation Analysis Assay

The EGFR Mutation Analysis Kit (EntroGen, Inc.) was used to detect somatic mutations in EGFR exons 18, 19, 20, and 21. The assay employed mutant-specific PCR amplification with fluorescent probe detection to identify common EGFR mutations, including T790M, exon 19 deletions, L858R, L861Q, S768I, G719X and exon 20 insertions. Each reaction included mutation-specific primers and probes along with an endogenous control to assess DNA quality and reagent performance. PCR reactions were prepared according to the manufacturer's instructions in a 30 μ L reaction volume and performed on the Roche LightCycler® 480 (Software version 1.5). The amplification data were subsequently analyzed to determine the EGFR mutation status.

Data Analysis

Quantitative data is presented with the help of Mean and Standard deviation. Comparison among the study groups was done with the help of unpaired t test as per results of normality test. Qualitative data was presented with the help of frequency and percentage table. Association among the study groups is assessed with the help

of Fisher test, student 't' test and Chi-Square test. 'p' value less than 0.05 was taken as significant

Results

The study included 30 patients with head and neck squamous cell carcinoma (HNSCC), with a mean age of 58.2 ± 8.38 years. Most patients belonged to the 51–60 years age group (46.7%), followed by the 61–70 years age group (36.7%). A marked male predominance was observed, with 83.3% males and 16.7% females.

The tongue was the most common tumor site (33.3%) followed by the buccal mucosa (20%), while other sites such as the mandible, maxilla, floor of the mouth, gingivobuccal sulcus, glottis, larynx, lip, oropharynx, supraglottis, tonsillar fossa, and tonsillar pillar were less frequently involved. Regarding tobacco exposure, 50% of patients had a history of tobacco chewing, 26.7% were smokers, 3.3% reported both smoking and tobacco chewing, whereas 20% had no history of tobacco use.

Most tumors were diagnosed at Stage I (36.7%), and the majority were well-differentiated (Grade I, 76.7%), followed by moderately differentiated (16.7%) and poorly differentiated tumors (6.7%). Somatic EGFR mutations were detected in only 2 patients (6.7%), while 93.3% of cases showed no detectable EGFR mutation, indicating a low prevalence of EGFR mutations in this study population (Table 1).

In our study, both the patients with somatic EGFR mutation were in the age group of 51–60 years. There was no significant association of Somatic EGFR Mutation and age of patients as per Student t-test ($p > 0.05$) (Table 2).

The results of the present study stated that both the somatic EGFR mutations were located in the tongue. There was no significant association of Somatic EGFR Mutation and site of tumour in patients as per Chi-Square test ($p > 0.05$) (Table 3)

In present study, 1 (3.3%) patient with somatic EGFR mutation had T2 stage while 1 (3.3%) patient had T4 stage. There was no significant association of Somatic EGFR Mutation and TNM

Staging of patients as per Chi-Square test ($p>0.05$) (Table 4).

Both patients with somatic EGFR mutation had Grade 1 tumor. There was no significant

association of Somatic EGFR Mutation and Grade of patients as per Chi-Square test ($p>0.05$) (Table5).

Table 1: Demographic profile of study participants

Age	N	%
31-40	1	3.3%
41-50	3	10%
51-60	14	46.7%
61-70	11	36.7%
71-80	1	3.3%
Total	30	100%
Mean $\pm$ SD	58.2 $\pm$ 8.38	
Sex		
Male	25	83.3%
Female	5	16.7%
Total	30	100%
Site		
Tongue	10	33.3%
Buccal Mucosa	6	20%
Mandible	3	10%
Maxilla	2	6.7%
Floor of mouth	1	3.3%
Gingivobuccal sulcus	1	3.3%
Glottis	1	3.3%
Larynx	1	3.3%
Lip	1	3.3%
Oropharynx	1	3.3%
Supraglottis	1	3.3%
Tonsillar fossa	1	3.3%
Tonsillar pillar	1	3.3%
Tobacco Intake		
Tobacco chewing	15	50%
Smoking	8	26.7%
Smoking and Tobacco chewing	1	3.3%
No smoking/tobacco chewing	6	20%
TNM Staging		
0	1	3.3%
1	11	36.7%
2	4	13.3%
3	3	10%
Grade		
1	23	76.7%
2	5	16.7%
3	2	6.7%
Somatic EGFR Mutation		
Present	2	6.7%
Absent	28	93.3%

Table 2: Association of Somatic EGFR Mutation and Age of patients

Age	Somatic EGFR Mutation				Total		p Value
	Present		Absent				
	N	%	N	%	N	%	
31-40	0	-	1	3.3%	1	3.3%	>0.05
41-50	0	-	3	10%	3	10%	
51-60	2	6.7%	12	40%	14	46.7%	
61-70	0	-	11	36.7%	11	36.7%	
71-80	0	-	1	3.3%	1	3.3%	
Total	2	6.7%	28	93.3%	30	100%	
Mean ± SD	53.5 ± 2.12		58.6 ± 8.57		58.2 ± 8.38		

Table 3: Association of Somatic EGFR Mutation and Site of Tumour in patients

Site	Somatic EGFR Mutation				Total		p Value
	Present		Absent				
	N	%	N	%	N	%	
Tongue	2	6.7%	8	26.7%	10	33.3%	>0.05
Buccal Mucosa	0	-	6	20%	6	20%	
Mandible	0	-	3	10%	3	10%	
Maxilla	0	-	2	6.7%	2	6.7%	
Floor of mouth	0	-	1	3.3%	1	3.3%	
Gingivobuccal sulcus	0	-	1	3.3%	1	3.3%	
Glottis	0	-	1	3.3%	1	3.3%	
Larynx	0	-	1	3.3%	1	3.3%	
Lip	0	-	1	3.3%	1	3.3%	
Oropharynx	0	-	1	3.3%	1	3.3%	
Supraglottis	0	-	1	3.3%	1	3.3%	
Tonsillar fossa	0	-	1	3.3%	1	3.3%	
Tonsillar pillar	0	-	1	3.3%	1	3.3%	
Total	2	6.7%	28	93.3%	30	100%	

Table 4: Association of Somatic EGFR Mutation and TNM Staging of patients

TNM Staging	Somatic EGFR Mutation				Total		p Value
	Present		Absent				
	N	%	N	%	N	%	
0	0	-	1	3.3%	1	3.3%	>0.05
1	0	-	11	36.7%	11	36.7%	
2	1	3.3%	3	10%	4	13.3%	
3	0	-	3	10%	3	10%	
4	1	3.3%	10	33.4%	11	36.7%	
Total	2	6.7%	28	93.3%	30	100%	

Table 5: Association of Somatic EGFR Mutation and Grade of patients

Grade	Somatic EGFR Mutation				Total		p Value
	Present		Absent				
	N	%	N	%	N	%	
1	2	6.7%	21	70%	23	76.7%	>0.05
2	0	-	5	16.7%	5	16.7%	
3	0	-	2	6.7%	2	6.7%	
Total	2	6.7%	28	93.3%	30	100%	

DISCUSSION

The EGFR gene encodes a receptor protein that dimerizes upon ligand binding, activating tyrosine kinase activity and receptor phosphorylation. The kinase activity of EGFR can be increased by mutations to EGFR, inducing the hyperactivation of downstream pro-survival signaling pathways.

In the present study, majority of the patients (46.7%) were in the age group of 51-60 years followed by 36.7% in the age group of 61-70 years, 10% in the age group of 41-50 years and 3.3% in the age groups of 31-40 years and 71-80 years. The mean age of the patients was 58.2 ± 8.38 years. There was male preponderance (83.3%) while female patients constituted 16.7% of the study group. This is similar to a prospective clinical study of somatic EGFR mutations in HNSCC cases by Smilek P et al.[9]

The most common site of tumor in our study was Tongue (33.3%) followed by Buccal Mucosa (20%), Mandible (10%) and Maxilla (6.7%). In the study by Smilek P et al [9], majority of tumors were located in the oral cavity and oropharynx (72.4%) followed by hypopharynx (17.3%) and larynx (10.3%).

It was observed in the present study that the TNM stage was T0 in 1 (3.3%) patient while it was T1 and T2 in 11 (36.7%) and 4 (13.3%) patients respectively. The TNM stage was T3 and T4 in 3 (10%) patients and 11 (36.7%) patients respectively. Majority of the patients in our study (76.7%) had Grade 1 tumor while 5 (16.7%) and 2 (6.7%) patients had Grade 2 and Grade 3 tumor respectively. The study of Smilek P et al found all patients had advanced disease (clinical stage III and IV). Almost 60% of patients had grade 2 tumor, 31% grade 3 and 10% grade 1.[9]

In our study 1 (3.3%) patient was presented with lympho-vascular invasion. Perineural invasion (PNI) and Extra-nodal Extension was found in 1 (3.3%) and 2 (6.7%) patients. Epidermal Growth Factor Receptor (EGFR) Mutations were found in 2 (6.7%) patients. 1 (3.3) patient had single mutation (S768I) while the other patient had double mutations (S768I, T790M). Nagalakshmi K et al in a hospital-based case-control study, assessed the EGFR mutations in head and neck. HNSCC, 81.39% of cases were found to harbor four EGFR mutations (G2155C, G2176A, C2188G, and G2471A). Two of these mutations had been reported previously in other malignancies, while the other two were identified as novel mutations in HNSCC.[10] In the present study, one (3.3%) patient with somatic EGFR mutation had T2 stage while 1 (3.3%) patient had T4 stage. There was no significant association of Somatic EGFR Mutation and TNM Staging of patients as per Chi-Square test ($p > 0.05$).

The study of Smilek P et al reported no significant difference between different clinical characteristics (age, clinical stage, grade, toxicity) and EGFR v III mutation with the exception of gender.[9]

Both patients with somatic EGFR mutation in our study had Grade 1 tumor. There was no significant association of Somatic EGFR Mutation and Grade of patients as per Chi-Square test ($p > 0.05$). Similar observations were noted by Smilek P et al⁹ and Nagalakshmi K et al.[10]

Smilek P et al [9] prospective clinical study reported patients who achieved a complete response had significantly higher expression of the EGFR gene than patients with a partial response or without a response. It also found patients associated with exon 19 mutations to have a worse outcome.

Among *EGFR*-mutated HNSCCs [11], the majority of TKD mutations were located in exon 19 followed by exon 20, exon 21 and exon 18. Of all *EGFR* mutations, missense mutations in exons 18-21 occurred in 73%, followed by deletions in exon 19 and insertion mutations in exon 20. In our study 1 patient had single mutation S768I while the other patient had double mutations S768I, T790M. The missense mutation T790M in exon 20, which has a known association with acquired resistance to EGFR TKIs in fifty percent NSCLC, was found in 7.5% of all *EGFR* mutations in HNSCC as per the study by Perisanidis. In-frame deletions in exon 19, which accounts for 45% of all EGFR mutations and is responsive to TKIs in NSCLC was seen in 22% of all mutated cases in HNSCC. 3% of all *EGFR*

mutations in NSCLC is frequently associated with resistance to EGFR TKIs, were observed in 5% of all *EGFR* mutations in HNSCC. All together it is clear that HNSCC and NSCLC have substantial differences between distribution of mutations within exons 18-21 and these must be taken together, to understand that substantial differences exist between HNSCC and NSCLC.

Table 6:Prevalence of epidermal growth factor receptor (EGFR) kinase domain mutations in patients with head and neck cancer by country and geographic region

Geographic region	No. of studies	Patients with EGFR mutation	EGFR mutation prevalence 95%(CI)
Overall	53	117/4122	2.8 (2.4-3.4)
Europe	13	9/670	1.3 (0.7-2.6)
Austria	2	3/240	1.3 (0.3-3.9)
Spain	2	0/35	0
Belgium	2	0/76	0
UK	1	0/40	0
Switzerland	1	0/6	0
Czech Republic	1	2/29	6.9 (1.2-24.2)
Hungary	1	0/7	1 0
Greece	1	3/92	3.3 (0.8-9.9)
France	1	0/4	1 0
Italy	1	1/40	2.5 (0.1-14.7)
North America	19	52/1901	2.7 (2.1-3.6)
USA	19	52/190	1 2.7 (2.1-3.6)
Southeast Asia	15	53/1079	4.9 (3.7-6.4)
Republic of Korea	5	41/271	15.1 (11.2-20.1)
Taiwan	3	2/248	0.8 (0.1-3.2)
Singapore	3	2/159	1.3 (0.2-4.9)
Japan	2	5/173	2.9 (1.1-7.0)
China	2	3/228	1.3 (0.3-4.1)
South Asia	4	0/367	0
India	4	0/367	0
South America	1	2/45	4.4 (0.8-16.4)
Australia	1	1/60	1.7 (0.1-10.1)

The present study showed a low frequency of somatic EGFR mutations (6.7%) in HNSCC, with no significant association between EGFR mutation status and clinicopathological parameters such as TNM stage and tumor grade. These findings are consistent with previous studies, suggesting that EGFR mutations are relatively uncommon in HNSCC. However, their detection may still be clinically important for identifying patients who could benefit from targeted EGFR-based therapies.

Limitations

The present study has certain limitations, including a small sample size and a single-center, cross-sectional study design, which may limit the generalizability of the findings. EGFR mutation analysis was restricted to selected mutations in exons 18–21, and three samples were excluded due to poor DNA quality. Furthermore, only two cases showed EGFR mutations, limiting meaningful statistical correlation with clinicopathological parameters. The absence of long-term follow-up also precluded assessment of

the prognostic and therapeutic significance of the detected mutations.

Conclusion

In conclusion, the present study demonstrated a low prevalence of somatic EGFR mutations (6.7%) in patients with head and neck squamous cell carcinoma (HNSCC). The detected mutations included S768I and T790M, while no significant association was observed between EGFR mutation status and clinicopathological parameters such as TNM stage or tumor grade. These findings are consistent with several previous studies reporting the rarity of EGFR mutations in HNSCC and their limited correlation with tumor characteristics. The variation in mutation frequency reported across different populations may be attributed to differences in ethnicity, sample size, tumor site, and molecular techniques used. Although EGFR mutations appear to occur infrequently in HNSCC, their identification remains clinically relevant, as selected mutations may have therapeutic implications for targeted EGFR inhibitor therapy. Larger multicentric studies with comprehensive molecular profiling are warranted to better define the prognostic and predictive significance of EGFR mutations in HNSCC.

Declarations

Ethics Approval: The study was conducted after obtaining approval from the Institutional Ethics Committee of the Military Hospital, Jodhpur, Rajasthan, India. The study was conducted in accordance with the ethical standards applicable to research involving human participants and in accordance with the Declaration of Helsinki.

Informed Consent: Written informed consent was obtained from all participants prior to their inclusion in the study and for the use of clinical and pathological specimens for research purposes.

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Conflict of Interest: The authors declare that they have no conflict of interest.

Author Contributions: All authors contributed to the conception and design of the study, acquisition and interpretation of data, and preparation and critical revision of the manuscript. All authors approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Data Availability Statement: The data generated and/or analyzed during the present study are available from the corresponding author on reasonable request, subject to institutional and ethical restrictions.

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Use of Artificial Intelligence: The authors declare that no generative artificial intelligence tools were used to generate, analyze, or interpret the scientific data or to make scientific conclusions presented in this manuscript. Any language editing was performed by the authors.

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