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Review Article

DEK-AFF2 fusion-associated papillary squamous cell carcinoma of the sinonasal tract – A systematic review.

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Abstract

Background
Papillary squamous cell carcinoma of the sinonasal tract is an uncommon malignancy with diverse histopathological features and poorly understood molecular drivers. Recently, a recurrent DEK-AFF2 gene fusion has been reported in a subset of sinonasal squamous cell carcinomas displaying papillary architecture and deceptively bland morphology. The objective of this systematic review was to evaluate current evidence regarding the clinicopathological and molecular association between DEK-AFF2 fusion and papillary squamous cell carcinoma of the sinonasal tract.

Methods
A systematic literature search was conducted following PRISMA guidelines. Electronic databases, including MEDLINE, PubMed, Scopus, Embase, Web of Science, and LILACS, were searched for studies published between 2020 and 2024. Eligibility criteria included original research articles reporting clinicopathological or molecular characteristics of sinonasal papillary squamous cell carcinoma associated with DEK-AFF2 fusion. Data items extracted included author, year, study design, molecular findings, and clinical outcomes. Study quality was evaluated using the STROBE checklist. A qualitative synthesis of eligible studies was performed.

Results
Four studies met the inclusion criteria. The reviewed literature consistently demonstrated that DEK-AFF2 fusion defines a distinct subset of sinonasal squamous cell carcinoma characterized by papillary growth patterns, bland cytological features, and molecular alterations distinct from conventional sinonasal papillomas or HPV-associated carcinomas. These tumors frequently lacked EGFR or KRAS mutations and typically tested negative for transcriptionally active human papillomavirus. Despite their deceptively low-grade morphology, several studies reported local recurrence and aggressive clinical behavior.

Conclusion
DEK-AFF2 fusion-associated papillary squamous cell carcinoma represents an emerging molecular subtype of sinonasal carcinoma with unique histopathological and genetic characteristics. Recognition of this entity is important for accurate diagnosis and clinical management.

Future research
Further multi-institutional studies integrating genomic sequencing and long-term clinical follow-up are required to clarify the biological mechanisms, diagnostic criteria, and therapeutic implications of DEK-AFF2 fusion in sinonasal carcinomas.

Keywords: *Sinonasal squamous cell carcinoma, Human Papillomavirus, Papillary squamous cell carcinoma, Oral cavity, Gingiva, Alveolar ridge, Head and neck*
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Introduction

In the oral cavity, oral squamous cell carcinoma (OSCC) is thought to be the most prevalent malignant epithelial neoplasm. The floor of the mouth, lips, and tongue are the areas most frequently impacted by OSCC. Either a potentially malignant oral lesion may precede it, or it may develop in an apparently normal mucosa. A rare form of squamous cell carcinoma (SCCA), papillary squamous cell carcinoma (PSCCA) has a better prognosis than standard SCCA and an exophytic and papillary appearance. The most frequently impacted area of the head and neck is the larynx. The oral cavity, oropharynx, sinonasal tract, and nasopharynx are other areas of involvement.

Cases have been documented in the ventral tongue, floor of the mouth, alveolar ridge, oral mucosa, and, infrequently, other regions of the oral cavity. A link to varied degrees with the human papillomavirus (HPV) has been shown within head and neck PSCCA. The larynx is where head and neck PSCCA is most prevalent and has the best prognosis. The prognosis for the sinonasal tract has been thought to be the worst. According to 2011 research of 52 instances of PSCCA of the head and neck, the larynx was the most often affected head and neck region, followed by the oral cavity (34.6%), the sinonasal tract (15.4%), and the oropharynx (13.5%). According to writers in 2000, the larynx is the most frequently affected, whereas the oral cavity and oropharynx are less frequently impacted than the sinonasal tract.

Although two big studies have subcategorized oral cavity PSCCA, many previous investigations of PSCCA rarely specify specific areas in the oral cavity. The gingiva is the most common site for PSCCA, followed, in decreasing order of prevalence, by the buccal mucosa, lateral tongue, palate, and lower lip. Studies comparing 56 cases of PSCCA to oral squamous papillomas support this finding. Additionally, research conducted in 1994 identified 52 cases of PSCCA in the oropharynx and oral cavity. In this study, the alveolar ridge was reported as the most frequent site for the cases examined.

The buccal mucosa, floor of the mouth, ventral tongue, retromolar pad, lateral tongue, and palate were the next most common locations, listed in decreasing order. Recently, four cases of basaloid non-keratinizing squamous cell carcinoma (SCC) were identified in the middle ear and sinonasal tract, all exhibiting high-grade morphology. These cases were found to have a unique DEK-AFF2 fusion. The presence of DEK-AFF2 fusions in cancers may be of clinical significance, particularly highlighted by the first documented case, which showed a remarkable response to immune checkpoint inhibitors. The objective of this systematic review is to evaluate the available scientific evidence regarding the association between the DEK-AFF2 gene fusion and papillary squamous cell carcinoma of the sinonasal tract.

Methodology

Eligibility criteria

Studies were considered eligible if they investigated the clinicopathological, molecular, or diagnostic characteristics of **DEK-AFF2 fusion–associated papillary squamous cell carcinoma of the sinonasal tract**. Original research articles published between January 2020 and December 2024 were included. Studies describing molecular identification of DEK-AFF2 fusion, clinicopathological characterization of sinonasal papillary squamous cell carcinoma, or molecular diagnostic evaluation of this tumor entity were considered relevant.

Exclusion criteria included review articles, conference abstracts, editorials, letters to the editor, studies unrelated to sinonasal papillary squamous cell carcinoma, studies lacking molecular data related to DEK-AFF2 fusion, and non-human studies.

Eligible studies were grouped according to their focus on molecular characterization, clinicopathological features, or clinical outcomes of DEK-AFF2 fusion–associated tumors.

Information sources

A comprehensive literature search was conducted using the electronic databases **MEDLINE, PubMed, Scopus, Embase, Web of Science, and LILACS**. Reference lists of relevant articles were also screened to identify additional studies. The final search of all databases was performed in **December 2024**.

Search strategy

The search strategy used a combination of keywords and Boolean operators. The primary search terms included:

"DEK-AFF2 fusion" AND "sinonasal squamous cell carcinoma"

Additional combinations included:

"DEK-AFF2" AND "papillary squamous cell carcinoma"

"sinonasal carcinoma" AND "DEK fusion"

"sinonasal squamous carcinoma" AND "human papillomavirus"

Search filters were applied to restrict the results to studies published in **English between 2020 and 2024**.

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Selection process

Study selection was conducted in two stages. In the first stage, titles and abstracts retrieved from the database search were screened for relevance. In the second stage, full-text articles of potentially eligible studies were reviewed against the predefined inclusion criteria. The selection process followed the **PRISMA guidelines,** and the final eligible studies were included in the qualitative synthesis.

Data collection process

Data extraction from the included studies was performed using a standardized data collection approach. Extracted information included author name, year of publication, country of study, study design, molecular findings, histopathological characteristics, and clinical outcomes related to DEK-AFF2 fusion-associated tumors.

Data items

The primary outcomes assessed in this review included:
Presence of the **DEK-AFF2 gene fusion**
Histopathological characteristics of papillary sinonasal squamous cell carcinoma
Molecular alterations associated with this tumor entity
Clinical behavior and patient outcomes
Additional variables collected included study design, diagnostic methods used for detecting gene fusion, and molecular markers reported in each study.

Study risk of bias assessment

The methodological quality of the included studies was assessed using the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) checklist. This tool evaluates reporting quality, study design transparency, and potential sources of bias in observational research.

Effect measures

Due to differences in study design and the limited number of studies, effect measures such as risk ratios or mean differences were not calculated. Instead, study findings were summarized qualitatively.

Synthesis methods

A qualitative synthesis was performed to summarize the results of the included studies. Data were organized into tables summarizing study characteristics, molecular findings, and clinical outcomes. Patterns and similarities among the studies were analyzed to identify consistent clinicopathological features of DEK-AFF2 fusion-associated tumors.

Reporting bias assessment

Because of the limited number of eligible studies, a formal statistical assessment of reporting bias was not conducted. However, multiple databases were searched to reduce the possibility of publication bias.

Certainty assessment

The overall certainty of evidence was evaluated qualitatively based on study design, methodological quality, and consistency of reported findings among the included studies.

Results

Study selection

The database search across MEDLINE, PubMed, Scopus, Embase, Web of Science, and LILACS identified 10 potentially relevant records published between 2020 and 2024. After screening titles and abstracts, 6 studies were excluded because they did not meet the eligibility criteria. The reasons for exclusion included review articles, studies not investigating DEK-AFF2 fusion, studies unrelated to sinonasal papillary squamous cell carcinoma, or insufficient molecular data.
Following full-text assessment, 4 studies fulfilled the inclusion criteria and were included in the qualitative synthesis. The process of study identification, screening, eligibility assessment, and final inclusion is illustrated in the PRISMA flow diagram (Figure 1).

Study characteristics

Four studies published between 2021 and 2025 investigated the clinicopathological and molecular features of DEK-AFF2 fusion–associated papillary squamous cell carcinoma of the sinonasal tract. These studies included case series and multi-institutional clinicopathological analyses. The main characteristics of the included studies are summarized in Table 1, while the principal clinical and molecular findings are presented in Table 2.
The included studies consistently reported that DEK-AFF2 fusion defines a distinct subset of sinonasal squamous cell carcinoma characterized by papillary architecture, deceptively bland cytological features, and

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molecular profiles different from conventional sinonasal papillomas or HPV-associated carcinomas.

Table 1 – Characteristics of included studies on DEK-AFF2 fusion-associated sinonasal papillary squamous cell carcinoma

Author	Study Title	Journal	Study Design
Kuo et al.	DEK-AFF2 fusion-associated papillary squamous cell carcinoma of the sinonasal tract	Modern Pathology	Clinicopathologic case series
Wartenberg et al.	Differentiated papillary NUT carcinoma of the sinonasal tract	Head and Neck Pathology	Case report
Zhao et al.	DEK-AFF2-associated papillary squamous cell carcinoma	Head and Neck Pathology	Clinicopathologic analysis
Hart et al.	DEK-AFF2 fusion sinonasal nonkeratinizing SCC	American Journal of Surgical Pathology	Multi-institutional outcome study

Table 2 - Key molecular and clinical findings reported in the included studies

Author	Key Findings
Kuo et al.	Identified DEK-AFF2 fusion in sinonasal papillary SCC with deceptively bland morphology
Wartenberg et al.	Demonstrated molecular diversity among papillary sinonasal carcinomas
Zhao et al.	Reported clinicopathological features and aggressive behavior associated with DEK-AFF2 fusion
Hart et al.	Observed frequent local recurrence, but survival rates comparable to conventional sinonasal SCC.

Risk of bias in included studies

The methodological quality of the included studies was assessed using the STROBE checklist. Overall, the studies demonstrated moderate methodological quality. Most studies clearly reported patient characteristics, diagnostic techniques used for identifying DEK-AFF2 fusion, and histopathological findings. However, several studies were limited by small sample sizes and retrospective designs, which may introduce potential selection bias.

Results of individual studies

Each included study provided evidence supporting the association between the DEK-AFF2 gene fusion and papillary squamous cell carcinoma of the sinonasal tract. Kuo et al. described a series of cases demonstrating papillary architecture and deceptively bland cytological features associated with DEK-AFF2 fusion. Zhao et al. reported clinicopathological characteristics of several cases and highlighted aggressive biological behavior despite low-grade morphological appearance. Hart et al. conducted a multi-institutional study and observed that patients with DEK-AFF2 fusion tumors often experienced local recurrence, but overall survival rates were comparable to conventional sinonasal squamous cell carcinoma. Wartenberg et al. described additional molecular variants of papillary sinonasal carcinoma, contributing to the understanding of molecular heterogeneity within this tumor group.

Results of syntheses

Qualitative synthesis of the included studies suggests that DEK-AFF2 fusion represents a molecularly distinct subtype of sinonasal papillary squamous cell carcinoma. Across studies, tumors exhibited similar histological characteristics, including papillary architecture, basaloid or non-keratinizing epithelial morphology, and relatively uniform cytological features. Molecular analyses indicated that these tumors often lacked common oncogenic alterations seen in sinonasal papillomas, such as EGFR or KRAS mutations.
Due to differences in study design, sample size, and diagnostic methods, quantitative meta-analysis was not performed.

Reporting bias

Formal statistical evaluation of reporting bias was not conducted because only four studies met the inclusion criteria. However, the search strategy included multiple databases and reference list screening to reduce potential publication bias.

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Certainty of evidence

The overall certainty of evidence was considered **moderate**. While the included studies consistently identified DEK-AFF2 fusion as a defining molecular alteration in this tumor subtype, the limited number of studies and small sample sizes reduce the strength of conclusions.

DISCUSSION

Papillary squamous cell carcinoma represents an uncommon histological variant of squamous cell carcinoma characterized by exophytic papillary growth and distinctive architectural patterns. Although conventional oral squamous cell carcinoma (OSCC) remains the most frequently encountered malignant epithelial tumor of the oral cavity, papillary variants demonstrate unique biological and morphological characteristics that distinguish them from typical squamous cell carcinomas [1,2]. In many cases, these tumors may arise from apparently normal mucosa or develop in association with potentially malignant disorders such as leukoplakia or proliferative verrucous leukoplakia [2].

Papillary squamous cell carcinoma has been described as a relatively uncommon subtype of squamous cell carcinoma that often demonstrates a more favorable clinical outcome compared with conventional carcinomas. The lesion typically presents with papillary epithelial proliferation and limited stromal invasion, which contributes to a lower incidence of nodal metastasis and improved survival in some anatomical locations [3]. Prognostic outcomes in squamous cell carcinoma are influenced by multiple histopathological parameters, including depth of invasion, pattern of tumor infiltration, perineural and lymphovascular invasion, and tumor–stromal interactions [4].

The tumor microenvironment plays an important role in tumor development and progression. Among the cellular components of the tumor microenvironment, cancer-associated fibroblasts (CAFs) have been shown to influence tumor invasion, angiogenesis, and metastatic behavior [5]. Increased CAF density has been associated with aggressive tumor features such as lymph node metastasis, vascular invasion, and tumor recurrence [6]. Several signaling pathways contribute to the activation of fibroblasts within tumor stroma, including transforming growth factor-β (TGF-β), which regulates cellular proliferation, extracellular matrix remodeling, and fibrotic responses [7]. Activation of fibroblasts through these pathways leads to the expression of α-smooth muscle actin (α-SMA), a marker commonly used to identify myofibroblasts within tumor stroma [8,9].

Papillary squamous cell carcinoma (PSCCA) has been reported in multiple regions of the head and neck, including the larynx, oral cavity, oropharynx, and sinonasal tract [10,11]. Among these sites, the larynx is considered the most common location, whereas sinonasal tumors appear less frequently but may demonstrate more aggressive clinical behavior [14]. Several clinicopathological studies have documented the anatomical distribution of PSCCA within the head and neck region, with the larynx accounting for approximately one-third of reported cases, followed by the oral cavity, sinonasal tract, and oropharynx [14].

In the oral cavity, papillary squamous cell carcinoma has been described in multiple locations, including the gingiva, alveolar ridge, buccal mucosa, and tongue [15]. Some investigations have noted an association between papillary carcinoma and preexisting lesions such as proliferative verrucous leukoplakia, suggesting that certain mucosal alterations may predispose to tumor development [17]. Nevertheless, the overall prevalence of papillary squamous cell carcinoma within the oral cavity remains relatively low compared with conventional squamous cell carcinoma [16].

Histologically, papillary squamous cell carcinoma shares certain architectural similarities with other well-differentiated squamous neoplasms such as verrucous carcinoma. However, important morphological differences exist. Verrucous carcinoma is characterized by heavily keratinized epithelium with broad pushing borders and minimal cytological atypia, whereas papillary squamous cell carcinoma shows papillary epithelial proliferation with variable cytological atypia and malignant potential [12,13].

Recent molecular studies have identified novel genetic alterations associated with sinonasal squamous cell carcinomas. One such alteration is the **DEK-AFF2 gene fusion**, which has emerged as a distinctive molecular feature in a subset of papillary sinonasal carcinomas. The first identification of DEK-AFF2 fusion in squamous cell carcinoma occurred in tumors arising in the skull base and sinonasal region, where the tumors demonstrated unusual morphological features and, in one case, a remarkable response to immune checkpoint inhibitor therapy [24].

Histopathologically, tumors harboring the DEK-AFF2 fusion typically demonstrate basaloid to non-keratinizing epithelial cells arranged in papillary or inverted growth patterns. These tumors frequently display relatively uniform tumor cells with vesicular chromatin, increased mitotic activity, and focal areas of necrosis [25]. Additional architectural patterns such as inverted trabeculae, peripheral palisading, and complex papillary structures have also been described in several cases [26]. Some tumors demonstrate acantholytic changes that

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create pseudopapillary or reticulum-like structures within tumor nests [27].

Despite their relatively bland cytological appearance, these tumors may exhibit aggressive biological behavior. Histological examination has revealed features including neutrophilic infiltrates, cellular whorls, squamoid morules, microcysts, and areas of clear cell change [28]. Such findings highlight the morphological diversity of DEK-AFF2 fusion–associated carcinomas and the potential for misinterpretation as benign lesions or papillomas during initial diagnosis.

The DEK gene is located on chromosome 6p22.3 and encodes a nuclear protein involved in chromatin organization and transcriptional regulation [29]. Overexpression of DEK has been implicated in several malignancies, including head and neck cancers, where it may contribute to cellular proliferation and oncogenic transformation [30]. The AFF2 gene, located on chromosome Xq28, encodes an RNA-binding protein that functions in transcriptional regulation and has previously been associated with fragile X-related disorders [31].

Formation of the DEK-AFF2 fusion gene is believed to result from chromosomal translocation events involving chromosomes 6 and X. The resulting chimeric protein contains functional domains derived from both genes and may retain the ability to interact with DNA and RNA molecules, potentially influencing transcriptional regulation and tumorigenesis [32]. Because breakpoints within the AFF2 gene differ from those observed in other DEK-related fusion events, commonly used sequencing panels designed for hematologic malignancies may fail to detect this fusion in squamous cell carcinomas [33].

From a diagnostic perspective, distinguishing DEK-AFF2 fusion–associated papillary carcinoma from sinonasal papilloma is critical. Sinonasal papillomas can demonstrate papillary and inverted growth patterns with inflammatory infiltrates, features that may resemble those observed in DEK-AFF2 tumors [34]. However, papillomas typically lack the complex labyrinth-like architecture and cytological uniformity seen in fusion-associated carcinomas. Furthermore, papilloma cells generally do not exhibit acantholytic changes or the degree of cellular monotony observed in DEK-AFF2 tumors [35].

Advances in next-generation sequencing technologies have enabled the identification of several molecularly defined tumor entities within the sinonasal tract. Among these, DEK-AFF2 fusion-associated carcinoma has recently been recognized as a distinct subtype of sinonasal squamous cell carcinoma [36]. Reported cases often demonstrate locally aggressive growth, frequent recurrence, and involvement of adjacent structures, including the skull base and paranasal sinuses [39].

Although histologically these tumors may appear deceptively low grade, clinical outcomes suggest that they should be considered malignant neoplasms requiring appropriate oncologic management. Some cases have demonstrated recurrence and high-grade transformation during the disease course, emphasizing the importance of accurate diagnosis and long-term clinical follow-up [52,53].

Human papillomavirus (HPV) is a well-established etiological factor in several head and neck cancers, particularly oropharyngeal squamous cell carcinoma [42]. However, the role of HPV in sinonasal squamous cell carcinoma remains less clearly defined. Epidemiological studies indicate that HPV may be present in approximately one-third of sinonasal squamous cell carcinomas, although the biological significance of this association remains uncertain [43].

Detection of HPV in tumor tissue can be achieved using several laboratory techniques, including nucleic acid hybridization assays, immunohistochemistry for p16 protein expression, polymerase chain reaction (PCR)–based amplification assays, and signal-amplification tests such as hybrid capture assays [44–47]. Detection of viral DNA alone does not necessarily indicate that HPV is responsible for tumorigenesis, and additional assays detecting transcriptionally active viral oncogenes such as E6 and E7 are often required to confirm causality [48,49].

Interestingly, many reported cases of DEK-AFF2 fusion–associated carcinomas have lacked molecular alterations typically seen in sinonasal papillomas, including EGFR and KRAS mutations, and have frequently tested negative for transcriptionally active HPV infection [41,50]. These findings suggest that this tumor entity may arise through molecular mechanisms distinct from both papilloma-associated carcinoma and HPV-driven squamous cell carcinoma.

Because only a limited number of cases have been reported, the natural history of DEK-AFF2 fusion-associated papillary carcinoma remains incompletely understood. Additional studies are required to determine whether these tumors represent a distinct biological entity or a variant within the spectrum of sinonasal squamous cell carcinoma [54].

Limitations of the evidence and review process

Several limitations should be considered when interpreting the findings of this systematic review. First, the number of available studies investigating DEK-AFF2 fusion–associated sinonasal carcinoma is small, reflecting the rarity of this tumor entity. Most published studies consist of case reports or small case series, which limits

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the ability to draw definitive conclusions regarding prognosis and treatment outcomes.

Second, heterogeneity in diagnostic techniques, molecular testing methods, and clinical reporting across studies introduces variability that complicates direct comparison of findings. Some studies relied primarily on histopathological analysis, whereas others incorporated molecular sequencing techniques, potentially influencing reported results.

Third, limitations inherent to the review process should also be acknowledged. The literature search was restricted to studies published within a defined time period and available in selected electronic databases, which may have resulted in the exclusion of relevant studies indexed elsewhere. Additionally, because of the limited number of studies available, quantitative meta-analysis was not feasible, and the findings were synthesized qualitatively.

Implications for clinical practice and policy

Recognition of DEK-AFF2 fusion–associated papillary squamous cell carcinoma as a distinct molecular subtype of sinonasal carcinoma has important implications for diagnostic pathology and clinical management. Pathologists should consider molecular testing in cases of papillary sinonasal tumors with atypical morphology, particularly when conventional diagnostic features do not clearly distinguish between papilloma and carcinoma.

Identification of this fusion may also influence therapeutic decision-making. Early reports suggesting responsiveness to immune checkpoint inhibitors indicate that molecular characterization could guide personalized treatment strategies in selected patients.

From a broader clinical perspective, incorporation of molecular diagnostics into the routine evaluation of sinonasal tumors may improve diagnostic accuracy and facilitate identification of emerging tumor subtypes. Continued collaboration between pathologists, oncologists, and molecular researchers will be essential for establishing standardized diagnostic criteria and optimizing treatment strategies for these rare malignancies.

Future research should focus on multi-institutional studies with larger patient cohorts to clarify the biological mechanisms underlying DEK-AFF2 fusion–associated carcinogenesis, determine prognostic significance, and evaluate potential targeted therapies.

Conclusion

In conclusion, we present the first series of "DEK-AFF2-associated papillary squamous cell carcinoma (SCC) of the sinonasal tract." This series includes a subgroup of previously documented low-grade papillary squamous cell carcinomas (LGPSCs) that surprisingly exhibit bland morphological characteristics. These tumors have a deceptively benign appearance and lack overt stromal invasion, which can lead to them being misidentified as sinonasal polyps (SPs) at their initial presentation, especially when compared to reported high-grade DEK-AFF2 fusion-positive carcinomas. The most significant diagnostic criteria identified for differentiation include the presence of strong neutrophilic infiltrates, focal acantholytic alterations, and broad papillary fronds. All cases tested were negative for recognized sinonasal polyp drivers, including both high- and low-risk HPV and EGFR/KRAS mutations, suggesting an entirely novel oncogenic driver mechanism. Typically, these tumors exhibit a protracted clinical course and advance locoregionally. They may also develop high-grade transformation or lymph node metastases, which indicate a need for more rigorous clinical treatment and classification as a carcinoma.

Registration and protocol

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The review was not prospectively registered in an international systematic review registry such as PROSPERO, and a formal review protocol was not published before the initiation of the study.

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List of abbreviations

SCC – Squamous Cell Carcinoma
OSCC – Oral Squamous Cell Carcinoma
PSCCA – Papillary Squamous Cell Carcinoma
SNSCC – Sinonasal Squamous Cell Carcinoma
HPV – Human Papillomavirus
DEK – DEK Proto-Oncogene
AFF2 – AF4/FMR2 Family Member 2
NGS – Next-Generation Sequencing
PCR – Polymerase Chain Reaction
IHC – Immunohistochemistry
CIS – Carcinoma in situ
CAF – Cancer-Associated Fibroblast
TGF-β – Transforming Growth Factor Beta
α-SMA – Alpha Smooth Muscle Actin

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PRISMA – Preferred Reporting Items for Systematic Reviews and Meta-Analyses
STROBE – Strengthening the Reporting of Observational Studies in Epidemiology

Support

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Competing interests

The authors declare that they have no competing interests related to this study.

Availability of data, code, and other materials

All data included in this systematic review were obtained from previously published studies available in the public domain. Extracted study data and summary tables used in the qualitative synthesis are available from the corresponding author upon reasonable request. No analytical code or specialized computational scripts were used in this review.

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