ORIGINAL RESEARCH

Mapping of Mucin Signature: Uncoding the Landscape of Verrucous Carcinoma and Oral Squamous Cell Carcinoma

Marina B Joseph[1], Veena Naik[2], Punnya V Angadi[3]

Abstract

Introduction : Verrucous carcinoma (VC) is a low-grade variant of oral squamous cell carcinoma (OSCC) with distinct biological behaviour. Altered mucin expression plays a key role in tumor progression, yet comparative mucin profiling between VC and different histological grades of OSCC remains largely unexplored.

Aim: To evaluate and compare the distribution, intensity, and pattern of acidic and neutral mucins in VC and various histological grades of OSCC using Alcian Blue–Periodic Acid Schiff (AB-PAS) staining.

Materials and Methods: A total of 30 formalin-fixed, paraffin-embedded tissue blocks were retrieved, consisting of 15 VC and 5 cases each of well-differentiated squamous cell carcinoma (WDSCC), moderately differentiated squamous cell carcinoma (MDSCC), and poorly differentiated squamous cell carcinoma (PDSCC). Sections were stained using the AB-PAS technique to differentiate acidic and neutral mucins. Intratumoral and peritumoral regions were assessed for mucin distribution (focal/diffuse), staining intensity (0, +, ++), and predominant coloration. Descriptive statistical analysis was used to evaluate staining patterns across groups.

Results: VC showed focal intratumoral acidic mucin with strong focal neutral mucin, while peritumoral regions demonstrated diffuse, intense neutral mucin. WDSCC presented patterns similar to VC but with increased neutral mucin intensity. In MDSCC and PDSCC, acidic mucin became more diffuse intratumorally, whereas neutral mucin remained focal with strong peritumoral expression. Overall, acidic mucin intensity and diffusion increased with poorer tumor differentiation, while neutral mucin consistently exhibited strong peritumoral presence across all groups.

Conclusion: Distinct mucin signatures exist across VC and OSCC grades. Acidic mucins correlate with tumor aggressiveness, whereas neutral mucins reflect a stable peritumoral tissue response. Mucin profiling may aid in understanding tumor biology and provide supportive diagnostic value.

Keywords: Verrucous carcinoma, Oral squamous cell carcinoma, Mucin, AB-PAS, Tumor microenvironment.

Introduction

Oral cancer, specifically oral squamous cell carcinoma (OSCC), represents the most prevalent form of head and neck cancer, impacting areas such as the lips, tongue, and buccal mucosa. The incidence of OSCC in India is particularly alarming, accounting for approximately 40% of all oral cancer cases. This high prevalence is a significant public health concern, as OSCC remains one of the leading causes of cancer-related morbidity and mortality in the country[1, 2].

Verrucous carcinoma (VC) is a distinct, low-grade variant of OSCC. Unlike the aggressive nature of typical OSCC, VC is characterized by low mitotic activity and slow growth. This results in a phenotype that is less invasive and less aggressive than OSCC. Although VC generally grows slowly and maintains a well-differentiated morphology, it may infiltrate adjacent tissues in the later stages of the disease. Notably, VC does not metastasize to distant organs, reducing the risk of

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spread to other body sites.[3]

On the other hand, OSCC involves the abnormal proliferation of dysplastic squamous cells within the epithelium and connective tissue. These cells exhibit characteristic features of malignancy, such as a loss of normal tissue architecture, irregular cell shapes, increased nuclear atypia etc. OSCC has a much more aggressive behaviour compared to VC and is prone to both local invasion and metastasis. This difference in biological behaviour between VC and OSCC is essential for understanding the clinical management and prognosis of these carcino-

Table 1: Intensity and distribution of acidic mucin and neutral mucin in 15 cases of VC. Where as F is focal distribution and D is diffuse distribution. (+ denoted mild intensity, ++ denoted moderate intensity and +++denotes intense intensity)

VC(Number of cases =15)	ALCIAN BLUE (ACIDIC MUCIN)		PERIODIC ACID-SCHIFF (NEUTRAL MUCIN)	
	Distribution	Intensity	Distribution	Intensity
Intratumoral	F=12 (80%) D=3 (20%)	+ = 15(100%)	F= 9(60%) D=6(40%)	+ = 2(13%) ++= 13(87%)
Peritumoral	F= 12 (80%) D=3 (20%)	+ =13(87%) ++= 2 (13%)	F=1(7%) D=14(13%)	+ = 3(20%) ++= 12(80%)

Table 2: Intensity and distribution of acidic mucin and neutral mucin in 5 cases of WDSCC. Whereas F is focal distribution and D is diffuse distribution. (+ denoted mild intensity, ++ denoted moderate intensity and +++denotes intense intensity)

WDSCC(Number of cases =5)	ALCIAN BLUE (ACIDIC MUCIN)		PERIODIC ACID-SCHIFF (NEUTRAL MUCIN)	
	Distribution	Intensity	Distribution	Intensity
Intratumoral	F=3 (60%) D=2 (40%)	+ = 3(60%) ++=2(40%)	F= 3(60%) D=2(40%)	++= 5(100%)
Peritumoral	F= 5 (100%)	+ =5(100%)	D=5(100%)	++= 5(100%)

Table 3: Intensity and distribution of acidic mucin and neutral mucin in 5 cases of MDSCC. Whereas F is focal distribution and D is diffuse distribution. (+ denoted mild intensity, ++ denoted moderate intensity and +++denotes intense intensity)

MDSCC (Number of cases =5)	ALCIAN BLUE (ACIDIC MUCIN)		PERIODIC ACID-SCHIFF (NEUTRAL MUCIN)	
	Distribution	Intensity	Distribution	Intensity
Intratumoral	F=2 (40%) D=3 (60%)	+ = 2(40%) + += 3(60%)	F= 4 (80%) D=1 (20%)	+=1 (20%) ++= 4(80%)
Peritumoral	F= 4 (80%) D=1 (20%)	+ =5(100%)	F=1 (20%) D=4(80%)	+=1 (20%) ++= 4(80%)

Table 4: Intensity and distribution of acidic mucin and neutral mucin in 5 cases of PDSCC. Whereas F is focal distribution and D is diffuse distribution. (+ denoted mild intensity, ++ denoted moderate intensity and +++denotes intense intensity)

PDSCC(Number of cases =5)	ALCIAN BLUE (ACIDIC MUCIN)		PERIODIC ACID-SCHIFF (NEUTRAL MUCIN)	
	Distribution	Intensity	Distribution	Intensity
Intratumoral	D=5(100%)	+ = 5(100%)	F= 5(100%)	+=3(60%) ++= 2(40%)
Peritumoral	F= 4 (80%) D=1 (20%)	+ =5(100%)	D= 5(100%)	+=1 (20%) ++= 4(80%)

mas[4, 5].

The behaviour of any carcinoma is influenced not only by the genetic makeup of the tumour cells themselves but also by the microenvironment in which they reside. This concept, known as the tumour microenvironment, emphasizes the crucial role of surrounding tissue components, including stromal cells, blood vessels, immune cells, and extracellular matrix components, in shaping the behaviour of the tumour. The interaction between tumour cells and their microenvironment can dictate tumour growth, proliferation, invasion, and metastasis.[1,6]

The tumour microenvironment provides not only the physical scaffold for tumour cells to grow but also influences critical processes such as angiogenesis (formation of new blood vessels), immune evasion, and metastatic spread. As cancer cells interact with their microenvironment, they can manipulate surrounding tissues to support their growth and resistance to treatment[1, 6].

The Role of Mucins in Oral Cancer and Tumour Microenvironment

Mucins, which are high-molecular-weight glycoproteins, are produced predominantly by the epithelial cells lining various body cavities and structures. These mucins play vital roles in protecting and lubricating the epithelial surfaces of

Verrucous carcinoma

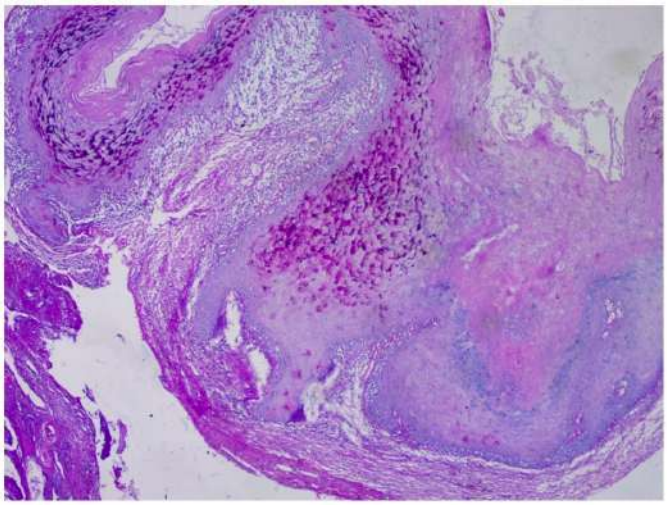

Fig. 1: AB-PAS stained section of verrucous carcinoma at 4X magnification showing focal acidic mucin positivity and diffuse neutral mucin expression in the stromal connective tissue.

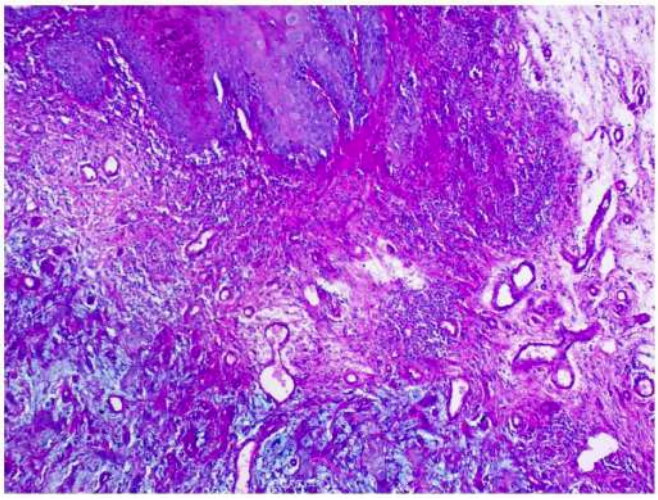

Fig. 2: AB-PAS stained section of verrucous carcinoma at 10X magnification demonstrating mild acidic mucin and intense neutral mucin distribution in the peritumoral region.

Well Differentiated Squmous Cell Carcinoma

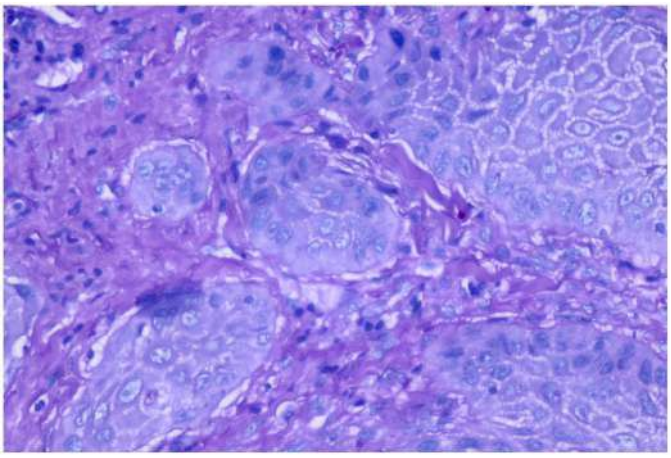

Fig. 3: AB-PAS stained section of well-differentiated squamous cell carcinoma (WDSCC) at 40X magnification showing focal intratumoral distribution of acidic mucin along with intense neutral mucin positivity within tumour islands and surrounding connective tissue stroma.

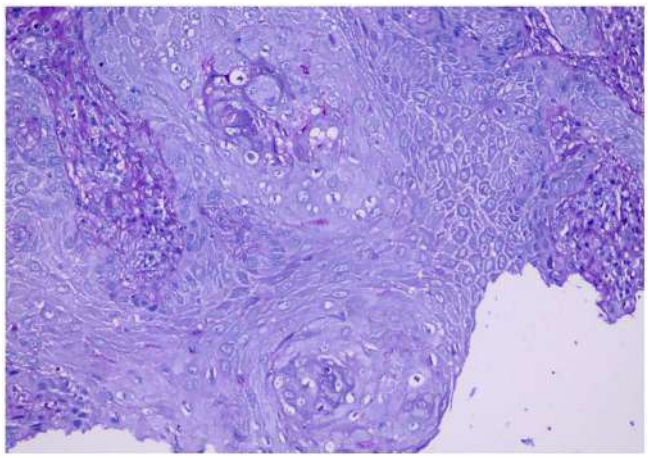

Fig. 4: AB-PAS stained section of well-differentiated squamous cell carcinoma (WDSCC) at 10X magnification demonstrating focal acidic

organs such as the gastrointestinal tract, respiratory system, and oral cavity. However, under pathological conditions such as cancer, the production of mucins can become dysregulated. In the context of OSCC, dysregulated mucin production has been identified as a crucial factor linking inflammation to cancer development. Inflammatory processes often precede and accompany the development of cancer, and mucins are thought to play a significant role in this interplay.

The abnormal or excessive production of mucins in the tumour microenvironment contributes to several aspects of cancer progression. It can facilitate the development of a protective barrier around tumour cells, allowing them to evade immune surveillance. This barrier may also serve to enhance tumour cell migration and metastasis by altering the extracellular matrix and promoting invasiveness. Furthermore, mucins have been shown to interact with growth factors

Moderately Differentiated Squmous Cell Carcinoma

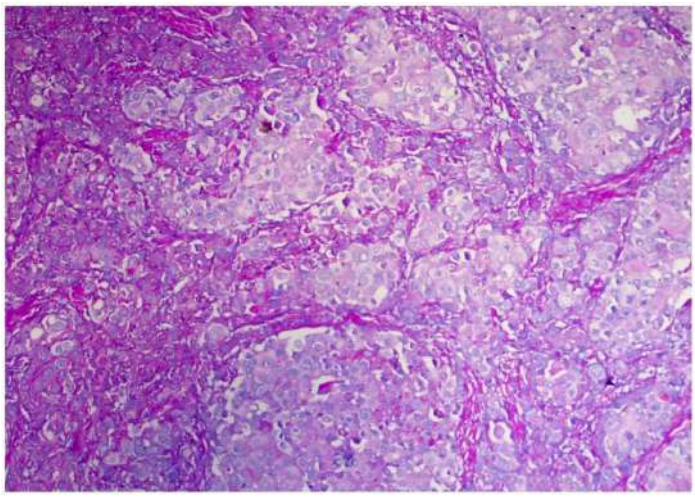

Fig. 5: AB-PAS stained section of moderately differentiated squamous cell carcinoma (MDSCC) at 40X magnification showing diffuse intratumoral acidic mucin expression with focal areas of neutral mucin positivity within the tumour nests

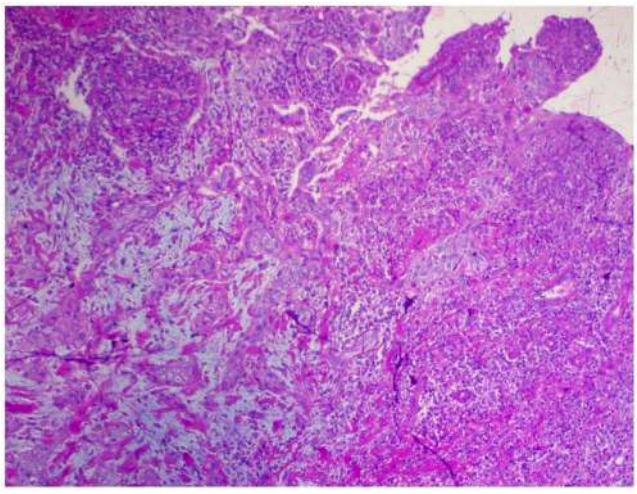

Fig. 6: AB-PAS stained section of moderately differentiated squamous cell carcinoma (MDSCC) at 10X magnification demonstrating diffuse acidic mucin distribution and intense neutral mucin positivity in the surrounding connective tissue stroma.

Poorly Differentiated Squmous Cell Carcinoma.

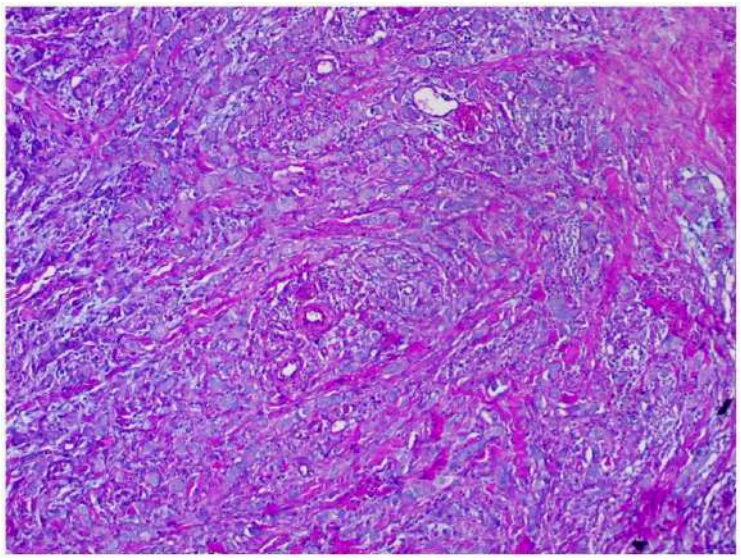

Fig. 7: AB-PAS stained section of poorly differentiated squamous cell carcinoma (PDSCC) at 10X magnification demonstrating diffuse acidic mucin distribution and intense neutral mucin positivity in the surrounding connective tissue stroma.

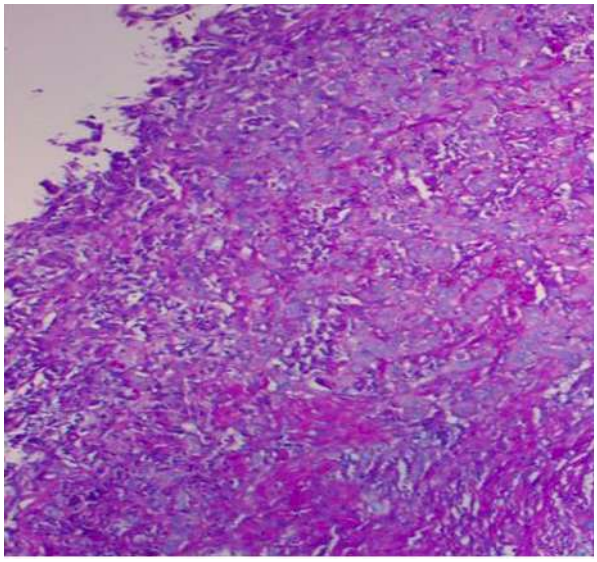

Fig. 8: AB-PAS-stained section of poorly differentiated squamous cell carcinoma (PDSCC) at 40X magnification demonstrating diffuse acidic mucin distribution with intense neutral mucin staining in the peritumoral connective tissue stroma.

and cytokines in the microenvironment, thereby supporting tumour cell survival and proliferation in the face of therapeutic interventions[7].

Taking into consideration the changes occurring in connective tissue mucins, the present study aims to evaluate the alterations in mucins in VC and various histological grades of OSCC. Additionally, it seeks to assess the staining intensity and distribution patterns of glycoproteins, acidic mucins, and neutral mucins using different histochemical stains.

Materials and Methods

In the present study, a total of 30 formalin-fixed paraffin-embedded (FFPE) tissue blocks were obtained from the archives of the department for histopathological analysis. These tissue samples were divided equally into two groups: A total of 15 cases of VC and 15 cases of oral OSCC, comprising 5 cases each of well-differentiated (WDSCC), moderately differentiated (MDSCC), and poorly differentiated (PDSCC) grades, were included in the study. These cases were carefully selected based on diagnostic accuracy to ensure uniformity in the study population. Sections of 4 μm thickness were prepared from each selected tissue block, and the sections were stained using the Alcian Blue-Periodic Acid Schiff (AB-PAS) technique. The staining procedure was carried out according to the method described by Bancroft and Gamble. AB-PAS is a well-established histochemical method used to detect and differentiate acidic and neutral mucins in tissue samples.

The staining procedure followed a standard protocol to maintain consistency across all samples. Initially, deparaffinization of the slides were done, which involved the removal of paraffin wax from the tissue to ensure optimal staining. This was followed by hydration through a series of graded alcohols to rehydrate the tissue. The slides were then washed for 10 min, and periodic acid was applied for 15 minutes followed by a rinse with distilled water. The sections were then stained with Alcian Blue for 20 minutes to highlight acidic mucins, after which they were washed with water for 2 minutes to remove excess stain. Subsequently, the sections were treated with Schiff reagent for 30 minutes, which reacts with aldehyde groups to identify neutral mucins and glycoproteins. The slides were rinsed again. A counterstain was applied using haematoxylin to provide contrast and better visualization of tissue structures. A final water wash ensured the removal of any remaining reagents. Bluing was performed using lithium carbonate to enhance the nuclear staining, and finally, the sections were dried and mounted using DPX, a synthetic resin medium, for microscopic examination.

Following the staining process, the stained sections were systematically examined under a light microscope. The primary objective of the evaluation was to assess the presence and distribution of mucins in the VC and OSCC tissues. The assessment of mucin distribution and staining intensity was carried out based on previously established histochemical evaluation criteria described in earlier by Sahni A et, al. Mucin was evaluated in both the intratumoral (within the tumour mass) and peritumoral (surrounding the tumour) regions to determine variations in mucin expression between different anatomical tumour zones. The mucin distribution was classified as either focal, where mucin was limited to specific areas, or diffuse, where mucin was more uniformly spread throughout the tissue.

Furthermore, the predominant colour exhibited by the mucins following AB-PAS staining was recorded. This colorimetric difference helps in distinguishing between acidic and neutral mucins. The intensity of mucin staining was graded on a semi-quantitative scale ranging from 0 to 2. A score of 0 indicated the complete absence of mucin staining, a score of 1 (+) signified minimal or weak staining, and a score of 2 (++) represented moderate to strong staining, indicating higher mucin content or greater affinity of the mucins for the stains used.

The stained sections were independently evaluated by two experienced oral pathologists who were blinded to the histopathological diagnosis. Assessment of mucin distribution and staining intensity was performed according to the predefined scoring criteria. To determine the reliability of observations, interobserver and intraobserver agreement were assessed using Cohen's kappa statistics. We got a kappa value of inter-observer agreement as 0.79 and for intra-observer agreement as 0.82 which is suggestive of good to excellent agreement.

All collected data were compiled and subjected to descriptive statistical analysis. Frequencies and percentages were calculated for various staining patterns, mucin distribution, and staining intensities observed in both groups. This statistical evaluation aimed to identify trends and differences in mucin expression between verrucous carcinoma and oral squamous cell carcinoma, thereby contributing to a better understanding of the histopathological behaviour and diagnostic value of mucin in these distinct but related oral malignancies.

Results

In VC stained with AB-PAS (Figures 1 and 2), the intratumoral area exhibits a weak, focal presence of acidic mucin and a strong, focal distribution of neutral mucin, whereas the peritumoral region shows a similarly weak, focal presence of acidic mucin but a diffuse and intense distribution of neutral mucin.

In WDSCC stained with AB-PAS (Figures 3 and 4), the intratumoral region shows both acidic and neutral mucins in a focal pattern, with neutral mucin exhibiting greater staining intensity. The peritumoral area displays a weak, focal presence of acidic mucin, accompanied by a diffuse and strongly stained distribution of neutral mucin (Table 2).

In MDSCC stained with AB-PAS (Figures 5 and 6), the intratumoral region demonstrates a diffuse pattern of acidic mucin and a focal distribution of neutral mucin, while the peritumoral area shows a weak presence of acidic mucin along with a diffuse and intense distribution of neutral mucin (Table 3)

PDSCC stained with AB-PAS (Figures 7 and 8) displays a diffuse pattern of acidic mucin and a focal distribution of neutral mucin in the intratumoral region, while the peritumoral areas consistently show a weak presence of acidic mucin along with an intense, diffuse distribution of neutral mucin (Table 4).

Key Differences and Trends:

1. Acidic Mucin:
    1. Found in a focal pattern intratumorally in VC and WDSCC, while diffuse in MDSCC/PDSCC.

2. Peritumoral acidic mucin is generally weak and focal in VC, WDSCC, MDSCC, and PDSCC.

2. Neutral Mucin:
   1. More focal intratumorally with stronger intensity in VC and WDSCC.
   2. In MDSCC/PDSCC, neutral mucin is focal but shows higher intensity in MDSCC.
   3. Peritumoral distribution is intense and diffuse across all types but is consistently more pronounced in VC.

Discussion:

VC is a rare variant of OSCC. In 1948, Lauren V. Ackermann first described this neoplasm of the oral mucous membrane, which is now also known as ''Verrucous carcinoma of Ackermann'' or ''Ackermann's tumor''[8]. VC is distinct in its slow growth and ability to become locally aggressive if not treated appropriately. However, even with local tumors progression, it is intriguing that regional or distant metastasis is rare.[9] OSCC is one of the most common malignancies with poor prognosis.

The extracellular matrix (ECM) which is part of tumour microenvironment play a important role in the tumour progression. The ECM is a dense latticework of collagen and elastic fibres which is embedded in a viscoelastic ground substance and is composed of proteoglycans and glycoproteins. This matrix acts as a scaffold which isolates tissue compartments, mediates cell attachment and influences tissue architecture. Interactions between normal cells and the matrix may be altered in neoplasia and thus may influence tumor proliferation and invasion.[10]

Mucin, one of the main ECM components seems to play a role in the process of tumour progression, invasion and metastasis and also in tumour cell survival and protection against the host immune response. Changes in the expression levels and glycosylation of mucins have been associated with several diseases including carcinomas.[11]

In the present study the histochemical evaluation of mucin distribution and staining patterns in VC and various grades of OSCC that is WDSCC, MDSCC, and PDSCC— reveals significant variations in both acidic and neutral mucins across intertumoral and peritumoral areas. These differences appear to correlate closely with the histological differentiation and biological aggressiveness of each tumour type.

In VC which is a well-differentiated and less aggressive variant, both acidic and neutral mucins are found in a focal distribution in intertumoral area. Notably, acidic mucin staining is weak, whereas neutral mucin is strongly expressed in a localized manner. In the peritumoral region, acidic mucin remains weak and focal, but neutral mucin shows a more diffuse and intense distribution. This pattern suggests a relatively localized tumour behaviour, with limited mucin secretion within the tumour but more extensive mucin deposition in the surrounding connective tissue, possibly as part of a host response or tissue remodelling.

In WDSCC, the pattern is similar to VC in the intertumoral area, with focal distribution of both mucin types. However, neutral mucin again exhibits greater intensity, reflecting increased mucin production by the tumor cells. The peritumoral region consistently shows weak, focal acidic mucin and diffuse, strong neutral mucin, reinforcing the notion that neutral mucins play a prominent role in the extracellular matrix response around well-differentiated tumours.

A marked shift is observed in MDSCC, where the intratumoral region shows a diffuse and more intense pattern of acidic mucin, coupled with a focal distribution of neutral mucin. This change indicates that as the tumour becomes less differentiated and more aggressive, there is a corresponding increase in acidic mucin secretion throughout the tumour mass. In the peritumoral area, weak acidic mucin and diffuse, intense neutral mucin persist, suggesting that neutral mucins are more prominently involved in peritumoral tissue response regardless of tumour grade, while acidic mucin production is more reflective of tumour aggressiveness.

In PDSCC, which represents the most poorly differentiated and aggressive tumor type, the intratumoral distribution remains diffuse for acidic mucin and focal for neutral mucin, mirroring the pattern seen in MDSCC but with reduced intensity of acidic mucin. This reduction may be attributed to the loss of specialized cellular functions in poorly differentiated tumours. The peritumoral region continues to show weak acidic mucin and intense, diffuse neutral mucin, consistent with observations in other tumour types.

Overall, a clear trend emerges: acidic mucin distribution within the tumour becomes more diffuse and intense as tumour grade worsens, particularly in MDSCC, whereas neutral mucin remains focal intratumorally but consistently diffuse and strong peritoumorally across all tumor types. This suggests that acidic mucins may serve as markers of tumor differentiation and aggressiveness, while neutral mucins likely play a more stable role in the tumor microenvironment and tissue response.

These findings highlight the potential diagnostic and prognostic value of mucin profiling in oral squamous cell carcinomas and may offer insights into tumour behaviour, aiding in the stratification of malignancy and possibly guiding therapeutic approaches.

Both acidic and neutral mucins appear to play a significant role in cancer progression, and a possible transition between different mucin types may occur during tumour progression Transformation in the mucins may be induced by the tumor cells, stromal cells or tumor associated fibroblasts need to be further investigated.

Further studies are required to establish the correlation with clinical details TNM staging, follow-up, habits and history to validate its diagnostic and prognostic purpose.

The findings of the present study are consistent with previous histochemical investigations that demonstrated progressive stromal alterations with increasing grades of OSCC. Sahni et al. reported enhanced stromal mucin deposition in oral epithelial dysplasia and OSCC, suggesting that mucin accumulation reflects tumour-associated extracellular matrix remodelling and may facilitate tumour progression[1]. George et al. observed significant stromal changes across different histological grades of OSCC and proposed that these alterations may contribute to tumour invasion and aggressiveness[12]. Similarly, Kardam et al. demonstrated modifications in stromal architecture accompanying tumour progression and

highlighted the prognostic significance of stromal alterations in OSCC(13) Recent studies evaluating tumour microenvironment components have further emphasized the importance of extracellular matrix remodelling, glycoprotein expression and stromal interactions in promoting tumour invasion, angiogenesis and immune evasion. These observations support the concept that alterations in mucin composition and distribution are not merely secondary phenomena but may actively participate in oral carcinogenesis and tumour progression.

To the best of our knowledge, the literature of acidic mucin histochemistry in VC is not available. Only a limited number of studies have documented the extensive mucin alterations across the varying grades of OSCC. (12-14) However, the reason for such transition in the acidic mucins has not been explained.

Conclusion

This study represents a foundational step toward investigating mucin alterations in VC and OSCC. While modified mucin expression is a well-established marker in gastrointestinal tracts and other mucosal tissues, only limited research has explored this phenomenon in OSCC. Special staining techniques remain a valuable, cost-effective alternative for both diagnostic and prognostic purposes due to their ease of standardization. This research can therefore serve as a critical platform for future investigations into the role of mucin modification in these oral pathologies.

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