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Helicobacter pylori- A potential biomarker in Oral Squamous Cell Carcinoma

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Abstract

Background: oral squamous cell carcinoma (OSCC) is the sixth most common cancer worldwide and is one of the leading causes of death. It is mainly known to be caused by tobacco in various forms & also due to viral, fungal infection & poor oral hygiene etc. Poor oral hygiene is often a neglected etiological factor in carcinogenesis which leads to colonization of pathogenic bacteria including H. pylori in oral cavity & found to be associated with gingivitis & periodontitis. 50% of world population harbors H. pylori in gastric mucosa & proven to cause peptic ulcer, gastric carcinoma, etc.

Therefore, early detection and eradication of H. pylori in high-risk patients is suggested. Various staining techniques like modified Giemsa, Warthin-Starry, Gimenez, Genta and immunohistochemical staining with H. pylori antibody are employed for it. Immunohistochemistry is the agreed "gold standard" for histological identification, being a highly sensitive and specific staining method.

Aim: To detect H. pylori in various grades of OSCC by using immunohistochemical staining

Objective

- To explore association of H pylori in etiopathogenesis OSCC
- To study the morphology of H. pylori.
- To study the distribution pattern of H. pylori in OSCC

Materials and method: Histopathologically diagnosed cases of OSCC will be selected from the archive of the department. 4 µm thick sections of paraffin embedded tissues of these cases will be taken & subjected for immunohistochemical stain. Sections will be observed under the microscope for presence of H Pylori.

Result: The presence of H. pylori was significantly found in histopathologically diagnosed cases of OSCC

Keywords: OSCC, H Pylori, Immunohistochemical.

Introduction

Oral squamous cell carcinoma (OSCC) is the sixth most common cancer worldwide and is one of the leading causes of death especially in developing countries like India. More than 90% of all oral cancers are squamous cell carcinoma. It is mainly known to be caused by tobacco in its various forms.[1] Poor oral hygiene is often a neglected etiological factor of oral carcinogenesis[2].

Human microflora of oral cavity can harbor variety of microorganisms including Helicobacter pylori (H. Pylori.)[3]. H. pylori is a microaerophilic, gram-negative, spiral organism which was first isolated by Marshall and Warren from human gastric biopsy specimens in 1983[4]. 50% of world population harbors H. pylori in gastric mucosa & its presence has been universally associated with chronic gastritis, duodenal ulcers & gastric carcinoma.[5, 6] H. pylori has evolved the ability to colonize the highly acidic environment found within the stomach by metabolizing urea to ammonia via urease, which generates a neutral environment enveloping the bacterium which enables them to survive in acidic environment.[7]

H. pylori produces a potent cytotoxin VacA (Vacuolating cytotoxin A) which causes progressive vacuolization and gastric injury. It induces various effects on gastric epithelial cells along with other cells. It causes vacuolization forming membrane anion- selective channel and pore formation and disruption of endosomal and lysosomal activity in host cells. VacA has immunomodulatory actions on T cells and other immune cells, possibly contributing to the chronic infections seen with the organisms. H. pylori virulence factors including VacA and CagA (cytotoxin-associated gene A), along with host genetic and environmental factors, constitute a complex network to regulate chronic gastric injury and inflammations, which is involved in a multistep process leading to gastric carcinogenesis.[8] Hence, H. pylori has been designated as a type I or definite carcinogen by the World Health Organization through its association with the development of gastric adenocarcinoma.

There is increasing evidence for role of the oral cavity as reservoir of H. pylori and new methods of detection are continuing to support this.[9] Different studies have revealed that H. pylori can also be isolated from the dental plaque (supragingival and subgingival plaque), dorsum of the tongue, salivary secretions, etc.[2] Poor oral hygiene also creates acidic environment in the oral cavity and harbors H Pylori.[10] Fernando et al., (2009) has proved that there is evidence of increased H pylori infection with tobacco, betel nut, alcohol consumption and poor oral hyigene[11]. It seems likely that the presence of H. pylori might be a risk factor for the developing oral lesions, ulcers and cancers.[2, 12] Dayama A. et al (2011) conducted a pilot case control study which supports the association of H. pylori with oral cancers.[13]

Therefore, early detection and eradication of H. pylori in high-risk patients is suggested.[14] There are various means for identification of H. pylori like Polymerase Chain Reaction (PCR) culture, urease breath test, etc.[15] The histological identification of H pylori infection is now a widely used means of diagnosis. Various staining techniques like modified Giemsa, Warthin-Starry, Gimenez, Genta and immunohistochemical staining with H. pylori antibody are employed for it. Immunohistochemistry is the agreed "gold standard" for histological identification, being a highly sensitive and specific staining method.[16]

Literature reveals variability in relation to the presence and association of H. pylori in etiopathogenesis of Oral squamous cell carcinoma. This study is designed to explore the possible association between H. pylori & oral squamous cell carcinoma by detecting the presence of it in histological sections of oral squamous cell carcinoma using immunohistochemical technique.

Aim And Objectives

Aim: The aim of the present study was to detect the presence of H. pylori in histopathological sections of Oral Squamous Cell Carcinoma by using immunohistochemical staining method.

Objective

- To find the presence of H. pylori in Oral Squamous Cell Carcinoma.
- To explore the possible association of H pylori in etiopathogenesis Oral Squamous Cell Carcinoma
- To study the morphological form of H. pylori.
- To study the type of distribution (whether isolated or in groups) of H. pylori in Oral Squamous Cell Carcinoma

Materials And Method

Histopathologically diagnosed cases of oral squamous cell carcinoma will be selected from the archive of the department (It is an in-vitro study & patient's identity will not be revealed). 4 µm thick sections of paraffin embedded tissues of these cases will be taken & subjected for immunohistochemical stain (H. Pylori antibody marker)

The sections stained with H. pylori antibody were observed under oil immersion objective(100x) of Research microscope (DM 1000 LED) with Computer assisted image analyzer (Leica Application Suit V-4.1) to study following parameters;

- Presence or absence of H. pylori.
- Morphological pattern of H. pylori.
- Distribution oh H. pylori

Results & Discussion

Oral squamous cell carcinoma is the sixth most common cancer worldwide and is one of the leading causes of death especially in developing countries like India. More than 90% of all oral cancers are squamous cell carcinoma and its incidence has increased in recent years. Surgery is the preferred treatment of OSCC. Despite great progress in chemotherapy, radiotherapy, and targeted therapy in the last three decades, the prognosis of OSCC is poor due to aggressive local invasion and metastasis, leading to recurrence. Thus, OSCC is still a challenging disease to treat in the field of head and neck cancer.[16] It is mainly known to be caused by tobacco in its various forms.[1] Other possible risk factor include poor oral hygiene.[17] Poor oral hygiene harbors many microorganisms in the oral cavity and which are known to cause oral cancer.[3, 18]

Human microflora of oral cavity can harbor variety of microorganism including Helicobacter pylori (H. Pylori.)[3]. H. pylori is a microaerophilic, gram-negative, spiral organism which was first isolated by Marshall and Warren from human gastric biopsy specimens in 1983[4]. 50% of world population harbors H. pylori in gastric mucosa & its presence has been universally associated with chronic

gastritis, duodenal ulcers & gastric carcinoma.[5, 6] The ward Helicobacter was derived from the ancient Greek ward which means spiral or coil and pylori means "of the pylorus" or "pyloric valve".

It is 3 micrometres long with a diameter of about 0.5 micrometres. It has 2-6 flagella that confers mobility.

Both in vivo and in vitro H. pylori can be found in coccoid and spiral form. H. pylori can change their shape from spiral to coccoid in some stressful conditions such as exposure to alkaline pH, increased temperature, nutrient starvation, exposure to antibiotics, etc. it has been also proposed that this transformation of bacterium to coccoid form is temporary in order to survive in stressful or unfavourable conditions and thus it can revert back to its original form i.e. spiral.[19] 50% of world population harbors H. pylori in gastric mucosa & its presence has been universally associated with chronic gastritis, duodenal ulcers & gastric carcinoma.[5] H. pylori has evolved the ability to colonize the highly acidic environment found within the stomach by metabolizing urea to ammonia via urease, which generates a neutral environment enveloping the bacterium which enables them to survive in acidic environment. An acidic environment promotes the survival of H. pylori in the presence of urea.[7]

The role of H. pylori in the pathogenesis of peptic and duodenal ulcers, gastric adenocarcinoma and low-grade B-cell mucosa associated lymphoid tissue lymphoma has already been proven.[20,21] Two mechanisms have been suggested for H. pylori pathogenesis. First, H. pylori interacts with surface epithelial cells, developing direct cell damage or producing pro-inflammatory mediators. Secondly, H. pylori reaches the underlying mucosa to stimulate an immune response, leading to the release of different cytokines and oxygen radicals that transform the chronic gastritis into gastroduodenal ulcers and gastric carcinoma.[2] In this way H. pylori can act as a direct mutagen. Interaction of intercellular signaling molecules and H. pylori CagA may predispose cells to accumulate multiple genetic and epigenetic changes that promote multistep carcinogenesis.[22]

H. pylori is not a normal commensal of oral cavity however, poor oral hygiene creates acidic environment and may harbor it.[10]

Different studies have revealed that H. pylori can be isolated from the oral cavity, dental plaque supragingival and subgingival plaque), dorsum of the tongue and salivary secretions[2] and supports the association of H. pylori in different oral lesions like periodontitis, premalignant and malignant conditions. In addition to this, Kgomo M. et al. (2016), also found that H. pylori infection is associated with an increased risk of developing Squamous cell carcinoma of the oesophagus.[24] The relation between H. pylori and gastric tumor pathogenesis has been well described. It is seems likely that it could also act in the same way in progression of oral and oropharyngeal carcinoma.[12]

Therefore, early detection and eradication of H. pylori in high-risk patients is suggested. There are various means for identification of H. pylori like Polymerase Chain Reaction (PCR).[12] The histological identification of H pylori infection is now a widely used means of diagnosis. Various staining techniques like modified Giemsa, Warthin-Starry, Gimenez, Genta and Immunos H. pylori antainig tibody are employed for it. Immunohistochemistry is the agreed "gold standard" for histological identification, being a highly sensitive and specific staining method.[16] In the present study, firstly, due to IHC specificity for H. Pylori detection and secondly, due to the decision to show the location of H. pylori inside the tissue as well as its invasion to the lamina propria, IHC was employed to detect H. pylori .

The first objective of present study was to check the presence of H. pylori in the histological sections of all the three study groups. Results showed (Table 1) that, out of 36 cases of OSCC, 28 cases (77.8%) were reactive for H. pylori and 8 cases (22.2%) were reactive for H. pylori. This suggests that the presence of H. pylori differs significantly between group 1 and group 3. Thus, the results of present study showed that there is significant association between H. pylori and OSCC. The exact role of H. pylori in the pathogenesis of OSCC is not known. However it could possibly act in following way;

Poor oral hyigene and other environmental factors like tobacco and betel nut chewing creates acidic environment in the oral cavity and may harbor H. pylori. H. pylori interacts with surface epithelial cells leading to the development of superficial mucositis by producing different extracellular products like VacA and CagA which progresses to chronic mucositis. These toxins reaches underlying oral mucosa to stimulate an immune response leading to release of different cytokines and oxygen radicals that transforms chronic mucositis to atrophic mucositis leading to the development of metaplasia and dysplasia which ultimately may lead to OSCC.[18]

The second objective was to study the morphological form of H. pylori in the histological sections of all the 3 study groups. Results of present study showed (table 2) showed that, out of 36 cases of OSCC, 26 had curvilinear morphology and 2 cases had helicle morphological form. None of the sample from this group showed coccoid form of H. pylori. This might be because, all the cases of OSCC were clinically manifested as ulcerated lesion and which have generated acidic environment favorable for maintaining the curvilinear form of H. pylori. Whereas out of 12 cases of group 2, 7 cases (58.3%) had curvilinear form and 5 cases (41.7%) had helicle form.

Mulugeta L et al, stated that H. pylori had poor motility in lag phase, became highly motile in mid-exponential phase and lost motility in the decline phase of growth. In the mid/late-exponential phase of growth bacteria had helical or curvilinear morphologies and multiple polar flagella, typical of H. pylori in the gastric mucus layer. In the decline phase of growth bacteria shed flagella, and had precoccoidal or coccoidal morphologies. These findings support the view that helical and coccoidal H. pylori are in different phases of growth with different roles in gastric colonization.[22]

As in the present study 72.2% cases of OSCC had curvilinear morphological form of H. pylori, it suggests that they are in mid/late exponential phase of growth and as none of the case showed coccoid form of H. pylori that means H. pylori present in all the cases of OSCC are not in the decline phase of their growth whereas they are in active phase of growth. Thus lending credibility to the theory that H. pylori plays active role in the pathogenesis of OSCC.

The third objective was to study the distribution of H. pylori in histological sections of OSCC i.e. group 1 and compare it with that of H. pylori found in the H. pylori infected gastric mucosa i.e. group 2. The results reveals that all the cases of H. pylori infected gastric mucosa showed distribution of H. pylori in groups whereas, most of the cases of OSCC showed isolated type of distribution of H. pylori. This suggests that the distribution of H. pylori differs significantly between OSCC and H. pylori infected gastric mucosa. ($P < 0.001$)

This might be because the pH of stomach is highly acidic as compared to the pH of oral cavity. In addition to this the high number of H. pylori might be associated with increased pathogenicity of H. pylori and hence considered to be a main etiological factor responsible for various gastric pathologies like gastritis, peptic ulcers and gastric

cancer. In present study most of the cases of OSCC showed isolated type of H. pylori distribution which suggests that H. pylori might not be the prime etiological agent for the pathogenesis of OSCC but it can act as contributing or co-etiological agent in the pathogenesis of OSCC.

Limitations of the study

Infection by H. pylori remains one of the most important scientific phenomenons in the biomedical literature worldwide and represents the most prevalent chronic bacterial disease because it affects more than half of the world's population. Helicobacter pylori consist of a large diversity of strains, and oral cavity is more likely to contain the entire DNA from every strain but at different levels of concentration. As our study have shown the presence of H. pylori in association with OSCC, further molecular studies are required for identifying and isolating the virulent strain of H. pylori.

Conclusion

Oral cancer poses a great health burden to our society. Tobacco and betel nut chewing, smoking, alcohol consumption, prolonged exposure to Sun, HPV infection and family history are the only risk factors that have been proven to be strongly associated with the development of oral cancer. However poor oral hygiene is often a neglected etiological factor which may harbors bacteria like H. pylori.

As the role of H. pylori in the pathogenesis of gastric cancer has been already established and as our study showed significant presence of H. pylori in histological sections of OSCC, it seems likely that presence of H. pylori might be a risk factor for the developing oral lesions like oral cancer. The possible role of H. pylori in the pathogenesis OSCC is as follows.

H. pylori interacts with surface epithelial cells leading to the development of superficial mucositis by producing different extracellular products like VacA and CagA which progresses to chronic mucositis. These toxins reach underlying mucosa of oral cavity to stimulate an immune response leading to release of different cytokines and oxygen radicals that transforms chronic mucositis to atrophic mucositis leading to development of metaplasia and dysplasia which ultimately may lead to OSCC.

Early detection and eradication of H. pylori in the oral cavity, especially in high risk patients such as tobacco users, alcohol consumers, patients with history of gastritis or with oral cancer might prevent its harmful consequences. In addition to this further studies on relationships between the genetic diversity and pathogenicity of H. pylori strains would lead to the development of novel and efficient therapeutic strategies for eradication of this microorganism.

This study should also heighten the awareness in the population regarding oral hygiene, poor oral hygiene harbors harmful microorganisms including been H. pylori, which may have the significant role in the etiopathogenesis of oral cancer.

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Legend Tables and Figure

Table 1: Prevalence of presence of H-Pylori in the histological sections (n=60).

	Group 1 [OSCC]		Group 2 [Positive Control]		Group 3 [Negative Control]		P-values [Inter-Group]		
H-Pylori	N	%	N	%	N	%	Group 1 v Group 2	Group 1 v Group 3	Group 2 v Group 3
Absent	8	22.2	0	0.0	12	100.0	0.522^{NS}	0.001***	0.001***
Present	28	77.8	12	100.0	0	0.0			
Total	36	100.0	12	100.0	12	100.0			

Values are n (% of cases). P-value by Chi-Square test with Post-Hoc Bonferroni's test for multiple group comparisons. P-value<0.05 is considered to be statistically significant. *P-value<0.05, **P-value<0.01, ***P-value<0.001, NS: Statistically Non-Significant.

Table 2: Distribution of morphological pattern of H-Pylori in the histological sections (n=60).

Morphological	Group 1 [OSCC]		Group 2 [Positive Control]		Group 3 [Negative Control]		P-values [Inter-Group]		
Pattern	N	%	N	%	n	%	Group 1 v Group 2	Group 1 v Group 3	Group 2 v Group 3
NA	8	22.2	0	0.0	12	100.0	0.012*	--	--
Curvilinear	26	72.2	7	58.3	0	0.0			
Helicle	2	5.6	5	41.7	0	0.0			
Total	36	100.0	12	100.0	12	100.0			

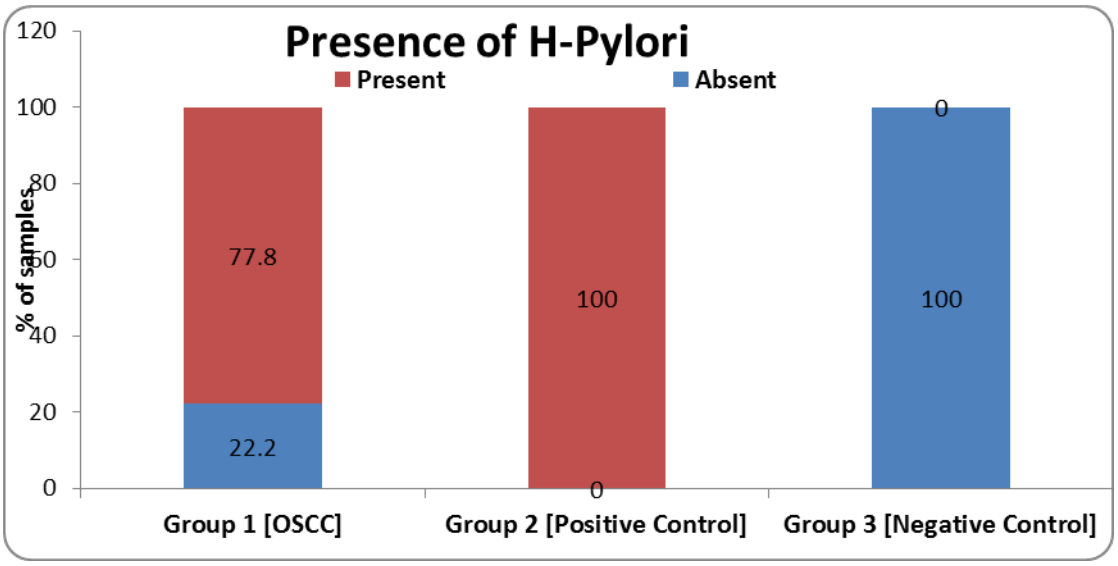
Values are n (% of cases). P-value by Chi-Square test with Post-Hoc Bonferroni's test for multiple group comparisons. P-value<0.05 is considered to be statistically significant. *P-value<0.05, **P-value<0.01, ***P-value<0.001, NS: Statistically Non-Significant. NA – Not applicable.

Table 3: Distribution of H-Pylori in the histological sections (n=60).

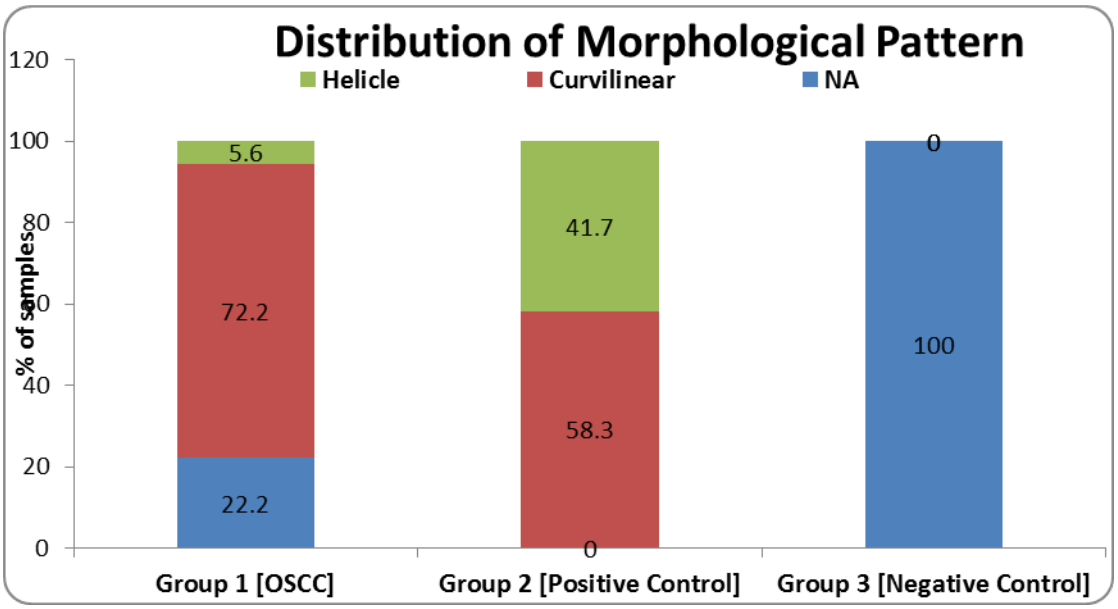
Distribution Status	Group 1 [OSCC]		Group 2 [Positive Control]		Group 3 [Negative Control]		P-values [Inter-Group]		
	N	%	n	%	n	%	Group 1 v Group 2	Group 1 v Group 3	Group 2 v Group 3
NA	8	22.2	0	0.0	12	100.0	0.003**	--	--
Isolated	25	69.4	0	0.0	0	0.0			
In Groups	3	8.3	12	100.0	0	0.0			
Total	36	100.0	12	100.0	12	100.0			

Values are n (% of cases). P-value by Chi-Square test with Post-Hoc Bonferroni's test for multiple group comparisons. P-value<0.05 is considered to be statistically significant. *P-value<0.05, **P-value<0.01, ***P-value<0.001, NS: Statistically Non-Significant. NA – Not applicabl

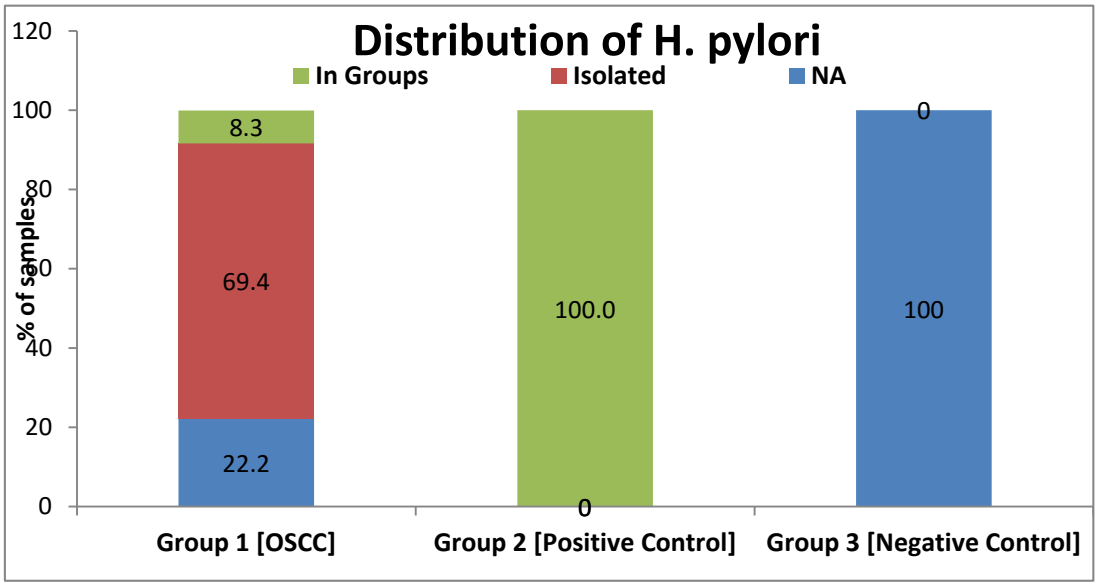
Graph 1: Prevalence of presence of H-Pylori in the histological sections (n=60).



Graph 2: Distribution of morphological pattern of H-Pylori in the histological sections (n=60).



Graph 3: Distribution of H-Pylori in the histological sections (n=60).



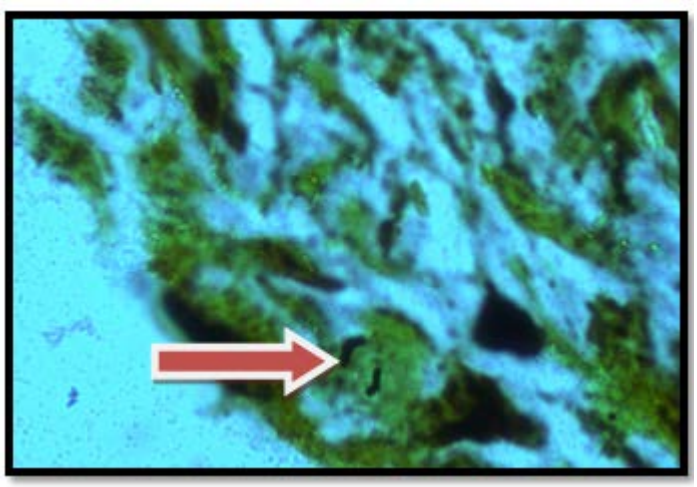


Figure 1

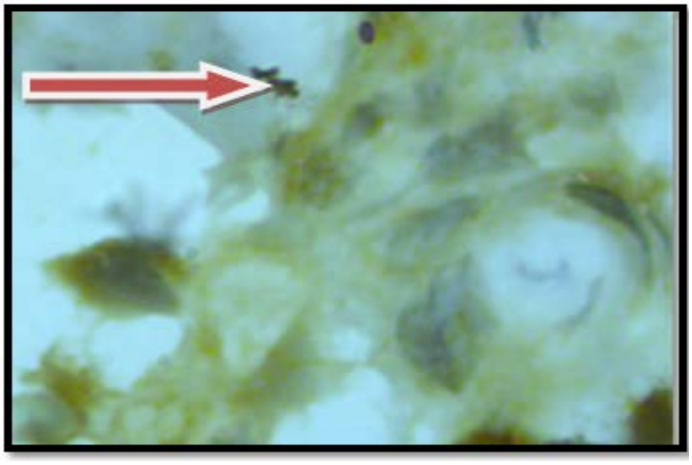


Figure 2

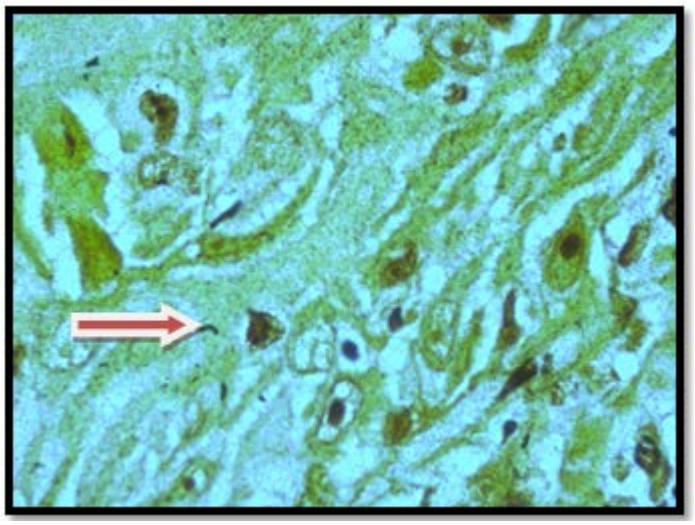


Figure 3

Figure 1, 2, 3: IHC expression of H. pylori in the connective tissue of histopathologically diagnosed case of OSCC. (100X)