MOLECULAR DETECTION OF ORAL *HELICOBACTER PYLORI* WITH *VAC*A, CAGA, AND *DUP*A VIRULENCE GENES IN RECURRENT APHTHOUS STOMATITIS PATIENTS IN DUHOK, KURDISTAN REGION, IRAQ

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ABSTRACT

Background: Recurrent Aphthous Stomatitis (RAS) is an inflammatory condition of unknown etiology characterized by recurrent and painful lesions, with single or multiple ulcerations that are confined to the oral cavity mucosa. The current study aimed at the molecular detection of oral *H. Pylori* and its *vac*A, *cag*A, and *dup*A virulence genes in patients with Recurrent Aphthous Stomatitis in Duhok, Kurdistan Region, Iraq.

Method: This is a cross-sectional study. It has been conducted in the laboratories of the college of medicine, Duhok city, Kurdistan Region, Iraq. A total of ninety-two individuals were included in the present study; forty-six patients with RAS consisted of 11 females and 35 males and forty six apparently healthy individuals as control group composed of 23 females and 23 males. A swab was taken from the RAS lesion of each in the patients' group and the control group's oral cavity (cheek) and submitted to a conventional PCR-based assay to detect the *H. Pylori* DNA using specific primers targeting *16SrRNA* gene. The family history for RAS of both the patients and the control group was investigated. *H. pylori* virulence genes *vacA*, *dupA*, and *cagA*(m1) were investigated in all extracted DNA samples using specific primers.

Results: *H. Pylori* DNA was detected only in 2 (4.34%) of the patients and one (2.17%) of the control group. The family history of RAS disease was positive in 24 (52.17%) of the cases, while only one individual (2.173%) of the control group had a positive family history of RAS disease. One of the *H. pylori* positive RAS patients showed a positive result for the three *vacA*, *dupA*, and *cagA*(m1) virulence genes, whereas the other one was positive for only *dupA* and *cagA*(m1) virulence genes. In addition, the *H. pylori* positive healthy control showed a positive result for all the three *vacA*, *dupA*, and *cagA*(m1) virulence genes.

Conclusion: There was no significant attribution *H. Pylori* in the etiology of RAS, while there was a highly significant relation of recurrent RAS with the family history of the patients ($p < 0.01$).

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Keywords: cagA, dupA, *Helicobacter pylori*, Recurrent aphthous stomatitis, vacA.

Recurrent Aphthous Stomatitis (RAS) is an inflammatory condition of unknown etiology characterized by recurrent and painful lesions, with single or multiple ulcerations that are confined to the oral cavity mucosa[1]. Clinically, RAS is characterized by recurrent, multiple, round or oval in shape, small size ulcers with demarcated margins, and grey or yellow floors with erythematous haloes. It usually occurs first in childhood or adolescence and later on occurs in adult life[2-3]. In general, RAS has been reported as affecting about 20% of the general population at any given time[4]. The prevalence of RAS was determined by the most comprehensive

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RAS study conducted on more than 10,000 young adults in more than 21 different countries; about 49.7% of females and 38.7% of males reported two or more previous episodes of RAS. Nearly about 25% of the participants reported at least one episode of RAS during the last year [5]. RAS can be classified based on the size of the ulcer and the number of ulcerations into three clinical types; minor recurrent aphthous stomatitis (MiRAS), major recurrent aphthous stomatitis (MaRAS), and herpetiform ulceration (HU)[6]. The episodes of RAS are self-limiting and recover within one to two weeks without leaving any scars[7].

The underlying etiology is unknown, but there are several local factors and underlying systemic diseases and conditions that predispose to the appearance of RAS, including genetic factors, food allergens, local trauma, endocrine alterations (menstrual cycle), stress and anxiety, smoking cessation, certain chemical products, and microbial agents[1-7]. Data introduced by some researchers suggest that the oral cavity may be a reservoir for *H. pylori* in some individuals, and the transmission of the disease may be via an oral-to-oral route[8]. *H. pylori* is a Gram-negative, S-shaped bacterium that has long been associated with gastritis and chronically infected duodenal ulcers [10]. Worldwide, the prevalence of *H. pylori* is about 50% (approximately 4.4 billion individuals infected). In developed countries, the prevalence rate is between 20 and 40%, and it reaches up to 90% in Africa and other non-developed countries[9]. The variation of the prevalence of infection among populations is related to race, ethnicity, geographical location, or method of testing[10]. *H. pylori*-associated diseases establishment and progression is attributed to a group of virulence factors[11]. There is a number of genes encode these factors; they are the cytotoxin-associated gene A (*cag*A), which has been described as an oncoprotein, vacuolating cytotoxin gene A (*vac*A), which plays a significant role in immune modulation as well as in the induction of gastric cancer and duodenal ulcer promoting gene A (*dup*A)[12,13,14]. Detection of these virulence factors in *H. pylori* is vital in determining the risk of the disease. In some studies, the *cag*A, *dup*A, and *vac*A genes have been reported as potential virulence factors of gastroduodenal illnesses in children and adults[15].

Therefore, the current study aims to detect *H. pylori* in oral samples of recurrent aphthous stomatitis patients and the presence of virulence genes *vac*A, *cag*A, and *dup*A.

MATERIALS AND METHODS

A cross-sectional conducted in the laboratories of Duhok medical college between the period June 2016 and January 2017, forty six RAS patients were included; they were presented with idiopathic recurrent aphthous stomatitis, the clinical diagnosis was achieved by a specialized dentist, the cases were recruited from high schools and the university of Duhok. Also, another, forty-six apparently healthy volunteer individuals were involved in the study as a control group. The ethical committee has approved the study of the health directorate of Duhok province. Verbal consent has been obtained from each subject. All the patients should have

experienced a minimum of three episodes of RAS within the last one year. Patients were subjected to an oral assessment protocol that included careful history review. All patients and healthy controls were questioned about gastroesophageal reflux's classic symptoms (heartburn, acid taste, and regurgitation). In addition, they were asked whether they had been treated previously for gastroesophageal reflux disease or *H pylori* infection in their stomachs. Well-known systemic disease patients (such as Behçet's syndrome, Sweet's syndrome, PFAPA syndrome, Reiter's syndrome, Crohn's disease, and gluten-sensitive enteropathy) were excluded. Patients under medication that could be associated with oral ulcers or *H. pylori* (such as antibiotics, proton pump inhibitors, H2 receptor blockers, Bismuth derivatives, non-steroidal anti-inflammatory drugs, chemotherapeutic agents, antibiotics, or vitamin supplements) for one month prior to the study were excluded. Women during pregnancy and menstruation were excluded as well. Swabs were collected from the oral cavity lesions of the 46 RAS patients and the cheek of the controls with the use of sterile cotton swabs. Genomic DNA was extracted from each of the oral swabs using DNA extraction kit (Qiagen, Germany) according to the manufacturer's instructions. PCR was conducted on extracted genomic DNA samples to detect the *H. pylori* DNA using a conventional PCR kit (Kapa Biosystem, USA). Primers used to target the *H. pylori-16SrRNA* gene were (F- 5′ GCG ACC TGC TGG AAC ATT AC 3′) and (R- 5′ CGT TAG CTG CAT TAC TGG AGA 3′) designed by Gramley *et al*[16]. The primers are expected to yield a 138bp PCR product. The PCR reaction was conducted according to the method mentioned by Roesler *et al*[12]. Briefly, the PCR reaction mix of each sample was made up to 25 µl. Each 25 µl PCR reaction mixture contained 12.5 µl PCR master mix (Promega, GoTag® Green Master Mix, USA), 0.5 µl each of primer (Metabion, Planegg, Germany), 5 µl of template DNA, and 6.5 µl of PCR grade water. For each PCR experiment, appropriate positive and negative controls were included. The *H. pylori* strain J99 and nuclease-free water were used as positive and negative controls, respectively. Forty thermal cycles were carried out, with each cycle consisting of a 30-second denaturation at 95°C, 1 min annealing at 60°C, and 1 min extension at 70°C and an additional 5 min extension at 70°C was needed to allow full product extension. To detect the PCR products, 5 µl of amplicons were electrophoresed in 2% agarose gel, ethidium bromide-stained, and visualized under UV light. *H. pylori* virulence genes *vacA*, *dupA* and *cagA*(m1) were investigated using PCR and the following specific primers: *cag*A(F: ACCGCTCGAGAACCCTAGTCGGTAA TGGG), (R: CAGGTACCGCGGCCGCTTAAGATTT TTGGAAACCAC),PCR product size 981 bp[17]. *vac*Am1 (F: GGTCAAAATGCGGTCATGG),(R:CCA TTGGTACCTGTAGAAAC), PCR product size 290 bp[17]. *dup*A (F:GACGATTGAGCGATGGGAATAT), (R:CTGAGAAGCCTTATTATCTTGTTG G), PCR product size 971 bp[18]. The same PCR conditions are followed, as described previously. Chi-square test was used to describe the association between RAS and the oral *H. pylori* presence as well as

virulence factor genes as attributed risk factors, using the SPSS statistical software package version 18.0 (SPSS, Inc., Chicago, IL). P values < 0.05 were considered statistically significant.

RESULTS

In the present study, ninety-two individuals were enrolled as two groups: the first group was the patients' group, which composed of forty-six patients with RAS disease, consisting of 11 females (23.91%) and 35 males (76.08%), their ages range was (15-40 years). The family history of RAS disease was positive in 24 patients (52.17%). The second group was the control group of 46 healthy individuals, consisting of 23 females (50%) and 23 males (50%), their ages range was (15-30 years), only one individual (2.173%) had a positive family history of RAS disease. There was a significant difference between the two groups regarding gender ($p < 0.05$), and there was a highly significant difference between the two groups regarding family history ($p < 0.001$) in table 1.

Table 1: ***H. pylori* DNA positive among RAS patients and the control group**

Studied groups	Gender	*H. pylori* (+)
RAS patients	35 Male (76.08%) 11 Female (23.91%)	2 (4.34%) 0 (0%)
Healthy controls	23 Male (50%) 23 Female (50%)	0 (0%) 1 (2.17%)

The 138 bp PCR product of *16SrRNA* gene of *H. pylori* was detected in the extracted DNA from oral swabs of only two RAS patients (4.34%) both of them were males, and it was detected in only one female DNA sample (2.17%) of an apparently healthy individual (control group) (Figure 1). There was no statistically significant difference between the two groups concerning the *H. pylori* DNA.

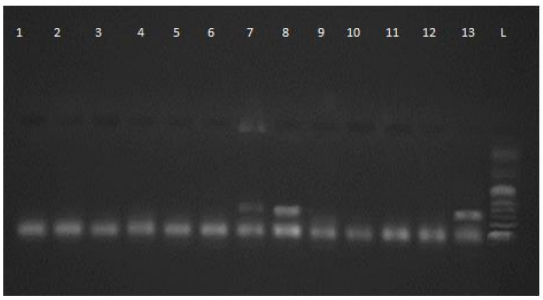

Figure1: **PCR products were obtained from extracted DNA of RAS lesions on 2% agarose gel electrophoresis, using specific primers targeting *16SrRNA* of the *Helicobacter pylori*. Lanes 7 and 8 are 138 bp products (*H. pylori* positive), lane L is a 50 bp DNA ladder, lane 13 is positive control, and the rest lanes are *H. pylori* negative in RAS patients.**

Table 2 shows the presence of *vacA*, *dupA*, and *cagA*(m1) virulence genes in *H. pylori* positive RAS cases and healthy controls. One of the *H. pylori* positive RAS patients showed positive results for the three *vacA*, *dupA*, and *cagA*(m1) virulence genes, whereas the other one was positive for only *dupA* and *cagA*(m1) virulence genes. Besides, the *H. pylori* positive healthy control showed positive results for all the three *vacA*, *dupA*, and *cagA*(m1) virulence genes table 2.

Table2: The presence of *vacA*, *dupA*, and *cagA*(m1) virulence genes in *H. pylori* positive RAS cases and the healthy controls.

Study groups	*H. pylori* positive cases *n*(%)	*vacA*	*dupA*	*cagA* (m1)
RAS patients	1 (2.17%)	+	+	+
	1 (2.17%)	-	+	+
Healthy control	1 (2.17%)	+	+	+

DISCUSSION

The RAS is a chronic inflammatory disorder characterized by the appearance of one or more ulcers on the oral mucosa with nonspecific histological features that persist for several days to several weeks, causing pain, and recurs after different periods of remission[19]. The etiology of RAS is unknown[20,21]. But there are several theories and studies that have been suggested and conducted to look for the etiology and predisposing factors of the RAS[22,23]. The present study aimed to evaluate the attribution of *H. pylori* as an etiologic factor in RAS in Duhok city, Iraq. The molecular technique (PCR) was used to detect the *H. pylori* in the RAS lesion). In the current study, the age range of the RAS patients was 16-28 years, and the mean was 22±2.2 years, and of the control group was 15-30 years with a mean of 18.41±3.2 years. Infection with *H. pylori* is, basically, asymptomatic, and the individual will be a carrier through life till the time when eradication treatment is done[24]. The exact mechanism by which *H. pylori* induces tissue injury is not clear. Some immune-mediated mechanisms are suggested[25]. Due to the similarity between the histological characteristics of gastric ulcers and oral aphthous ulcers, which respond to the treatment by broad-spectrum antibiotics, it looks logical to suppose that *H. pylori* could play a role in the etiopathogenesis of RAS disease, but still, the data regarding the potential relation between RAS and *H. Pylori* infection are limited and controversial [25]. In the current study, we found that there was no significant attribution of the *H. pylori* infection with the RAS, since it was detected in only 4.3% and 2.1% of RAS and controls, respectively. Other authors came out with results consistent with our results [26,27,28,29,30], while in other studies, the results were inconsistent with ours when Yi-Jian et al. (2015) found a significant association of *H. pylori* infections with oral diseases including periodontal diseases and caries[31]. In a review study, a search in PubMed (MEDLINE) databases was made of articles published up until July 2015, Gomes, *et al* stated that the *H. pylori* could be occasionally detected in RAS lesions and the eradication of the infection may affect the clinical course of RAS lesions by undetermined mechanisms. However, most of the studies do not support the association of RAS ulcers with the presence of the bacteria in the oral cavity, and the presence of the bacteria in the ulcer may reflect a passenger infection and not the trigger event. There is no convincing evidence of a direct cause- consequence effect of *H. pylori* infection and RAS ulcer development. This association requires further investigation by well-design prospective studies[32]. So, the relationship between *H. pylori* and RAS remains controversial, and these discrepancies in the findings of different studies remain unexplained, but some factors could explain these discrepancies; such as the small sample size of RAS patients as in our

study, variations in techniques and tests used in studies, the variations of the ways used in the collection of specimens, the density of the bacterial samples taken with swabs, the differences of ethnics of the studied patient populations, and various primers and target DNA used in the PCR assays [33]. In the present study, RAS's family history was positive in about (52.2%) of the patients' group, while (2.2%) of the control group had a family history of RAS. The current study results are consistent with those of Zand *et al*, 2012 who reported that the rates of positive family history of RAS in patients and control group were (54.2%) and (9%) respectively, which is a statistically significant risk factor for having RAS, and in the same time, they found that the family history (among the predisposing factors of RAS disease) have the strongest correlation with the RAS[23]. Also, Compilato *et al.* (2010) reported that family history was significantly associated with RAS. Some of the detected gene polymorphisms, such as pro-inflammatory cytokine encoding genes, explain the increased susceptibility to developing stimulated immune response to some oral antigens leads to aphthous formation erosions and oral ulcers[34]. Based on the previous data, family history should be highly considered during the management of RAS patients [35].

CONCLUSIONS

There was no significant association between the prevalence of *H. pylori* and the RAS. The family history of the included subjects had a highly significant impact on the etiology of RAS of the patients involved in the study.

CONFLICTS OF INTEREST

The authors have nothing to declare.

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پوختە

ناساندنا مولیکیولی یا بەکتریا زۆری دەرگەەی و فاکتەرێن ئەگەر dup A, cag A, rac A ل نەخوشێن ئەلسەرا دەڤی یا دووبارە ل دەوکێ هەرێما کوردستانێ عیراق

پێشەکی

ئە لسە را دەڤی یا دووبارە رەوشەکا گەلەمپێری یا میکوزا دەڤی یە. فاکتەرێن سەدەما هندێ ژی نە دیارن ئەڤ لێکولینە ئارمانج دکەت بو دیارکرنا مولیکیولی یا بەکتریا زۆری دەرگەەی و فاکتەرێن ئەگەر dup A, cag A, racA ل دەڤ نەخوشێن ئەلسەرا دەڤی یا دووبارە و دیارکرنا پەیوەندیا وان دگەل ئێك.

رێکێن کاری

46 نەخوشێن ب ئەلسەرا دەڤی یا دووبارە ، 11 مێ و 35 نێر هاتنە دەستنیشانکرن ، مەروەسا 46 کەسێن ساخلەم ژی وەکی کوما کونترول کو ژ 23 مێ و 23 نێر هاتنە دیارکرن ژ کوما نەخوشان ئەلسەرا دەڤی و ژ کوما کونترول چەولیکا دەڤی سواپ هاتنە وەرگرتن داکو جینا Sr RNA 16 بو بەکریا زۆری دەرگەهی و فاکتەرێن ئەگەر dup A, cag A, racA بێنە دیارکرن.

ئەنجام

ترشێ نەوەوی یێ جینا Sr RNA 16 هاتە دیارکرن لجەم دوو (4.34٪) نەخوشان کو ئێك ژ وان فاکتەرێن ئەگەر dup A, cag A, racA مەبوون ویێ دن فاکتەرێن ئەگەر cag A, dup A هەبوون مەروەسا ترشێ نەوەوی یێ جینا Sr RNA 16 هاتە دیارکرن لجەم ئێك (2.17٪) ژ کوما کونترول کو فاکتەرێن ئەگەر dup A, cag A, racA هەبوون د دیروکا کوما نەخوشاندا دیروکا مالباتێ دا یا نەخوشییا ئەلسەرا دەڤی یا دووبارە د 24 (52.17٪) هاتە دیتن، د هەمان دەمدا بتنێ ئێك کەس (2.17٪) یا کوما کونترولا خودان پاشناڤەك مالباتی بو بو نەخوشییا ئەلسەرا دەڤی یا دووبارە.

دەرئەنجام

مە دیارکر کو بەکتریا زۆری دەرگەەی نابیتە ئەگەرێ پەیدابوونا ئەلسەرا دەڤی یا دووبارە، د هەمان دەمیدا تێکەلیەکا گرنگ و بەێز هەیە دناڤبەرا ئەلسەرێن دەڤی بێن دووبارە و دیروکا مالباتی دا

الخلاصة

الكشف الجزيئي عن البكتيريا الملوية البوابية وعوامل الضراوة *vacA, cagA, dupA* لدى مرضى التقرحات الفموية المتكررة في دهوك – أقليم كردستان - العراق

خلفية البحث

تعتبر التقرحات الفموية المتكررة من الحالات المرضية التي تصيب الأغشية المخاطية للفم. لاتزال العوامل المسببة للمرض غير واضحة. الدراسة الحالية تهدف إلى الكشف الجزيئي عن البكتيريا الملوية البوابية وعوامل الضراوة *vacA*, *cagA*, *dupA* لدى مرضى التقرحات الفموية المتكررة والتحقق من مدى العلاقة بينهما.

المرضى وطرق البحث

تم تشخيص 46 فرداً من ذوي التقرحات الفموية المتكرر، 11 من الإناث و35 من الذكور وكذلك تم أختيار 46 من الأفراد الأصحاء كمجموعة سيطرة وتتكون من 23 إناث و23 من الذكور. أخذت مسحة من القرحة الفموية لكل فرد من مجموعة الدراسة ومن تجويف الفم (الخد) من مجموعة السيطرة للكشف عن الحمض النووي للجينة 16SrRNA للبكتيريا الملوية المعدية وعوامل الضراوة *vacA*, *cagA*, *dupA* وقد تم التحقيق في تاريخ الأسرة فيما يتعلق بالتقرحات الفموية المتكررة لكل من مجموعة الدراسة ومجموعة السيطرة.

النتائج

تم الكشف عن الحمض النووي للجينة 16 SrRNA للبكتيريا الملتوية المعدية في حالتين 2 (4.34٪) من مجموعة الدراسة احداهما احتوت على عوامل الضراوة *vacA*, *cagA*, *dupA* والاخرى احتوت على عوامل الضراوة *cagA*, *dupA*، وتم الكشف عن الحمض النووي للجينة 16 SrRNA للبكتيريا الملتوية المعدية في حالة واحدة فقط (2.17٪) من مجموعة السيطرة واحتوت على عوامل الضراوة *vacA*, *cagA*, *dupA*. تم العثور على تاريخ عائلي من مرض التقرحات الفموية المتكررة في 24 (52.17٪) من مجموعة الدراسة، في حين أن شخص واحد فقط (2.173٪) من مجموعة السيطرة كان لديه خلفية عائلية لمرض التقرحات الفموية المتكرر.

الاستنتاجات

توصلنا إلى أنه لم يكن هناك تأثير للبكتيريا الملتوية المعدية في التقرحات الفموية المتكررة، في حين كانت هناك علاقة معنوية عالية بين التقرحات الفموية المتكررة والتاريخ العائلي للمرضى.