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Oral mucosa pressure ulcers in intensive care unit patients: A preliminary observational study of incidence and risk factors

Chul-Hoon Kim[a], Myoung Soo Kim[b,*], Myung Ja Kang[b], Hyun Hee Kim[c], Nam Jung Park[d], Hyun Kyeong Jung[e]

[a] Department of Oral and Maxillofacial Surgery, College of Medicine, Dong-A University, Busan, South Korea
[b] Department of Nursing, Pukyong National University, Busan, South Korea
[c] Respiratory Intensive Care Unit, Pusan National University Hospital, Busan, South Korea
[d] Infection Control Team, Pusan National University Hospital, Busan, South Korea
[e] Urology Ward, Pusan National University Hospital, Busan, South Korea

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ABSTRACT

Purpose: This study examined the incidence of oral mucosa pressure ulcers (PUs) in intensive care unit (ICU) patients and the relationship between biomechanical and physiological variables in onset of PUs. Methods: A prospective observational descriptive study design was used. We recruited patients over 18 years of age with endotracheal tube (ETT) insertion in three ICUs in a tertiary hospital in Korea. We analysed 113 patient-days of data. Patient assessments and medical record reviews were conducted to gather biomechanical and physiological data. Fisher's exact tests and χ^2 test and Spearman's rank correlations were used to compare data. Results: The highest incidence of oral mucosa PUs occurred in lower oral mucosa (36.3%). There was a significant relationship between lower oral mucosa PU stage and bite-block or airway use ($r = .20$, $p = .036$), commercial ETT holder use ($r = 0.19$, $p = .048$), sedative use ($r = -0.22$, $p = .022$), and plasma protein ($r = 0.20$, $p = .033$). Upper oral mucosa PU stage was related to commercial ETT holder use ($r = 0.19$, $p = .044$), haemoglobin($r = 0.24$, $p = .011$), haematocrit ($r = 0.27$, $p = .004$), and serum albumin ($r = -0.24$, $p = .012$). Stage was related to commercial ETT holder use in both sites ($r = 0.28$, $p = .003$), haematocrit ($r = 0.19$, $p = .039$), and serum albumin ($r = -0.23$, $p = .015$). Conclusion: Oral mucosa PUs developed more frequently and healed more quickly than general skin PUs. Taken together, these data indicate that biomechanical and haematological variables are risk factors associated with PU incidence should be considered in intensive care patients.

1. Introduction

Medical device-related pressure ulcers (PUs) developed due to prolonged unrelieved pressure from rigid and inelastic devices [1], specifically related to difficulties in adjusting to the presence of the device on or in the body, and safe removal [1,2]. Patients who exhibit sensory impairment, reduced capacities for communication, and unconsciousness are at the highest risk for medical device-related PUs [3]; therefore critically ill patients in intensive care units (ICUs) are expected to be high-risk for PUs. Epidemiologic studies have reported various prevalence rates for medical device-related PUs, from 11.9% [4] to 40% [5], among hospital-acquired PUs. The most commonly encountered medical device-related PUs recorded by registered nurses were endotracheal tube (ETT)- related PUs in all ages [5–7].

For intubated patients, unplanned extubation or inadvertent movement of the ETT can induce potentially life-threatening problems, such as aspiration, airway injury, and death as a result of airway problems [8]; notably, the ability to maintain a secure ETT is considered a quality assurance marker for ventilator use [9]. Airway stability is affected by the type of securing method [9]. The ETT should be secured using non-commercial methods or commercial that minimize tube movement, ensure quick and easy application, and demonstrate of a low risk of injury to the body [10]. Non-commercial techniques including tying or fixing with adhesive tape and string, respectively, are more form-fitting to the patient's face [9] and effective at preventing oral mucosa breakdown [11,12]. Commercial ETT holders, which are wrapped using a quick fixation method (such as Velcro), around the back of the neck for stability, are advantageous because they improve

* Corresponding author. Pukyong National University, 599-1, Daeyeon 3 dong, Namgu, Busan, 48513, South Korea.
E-mail address: kanosa@pknu.ac.kr (M.S. Kim).

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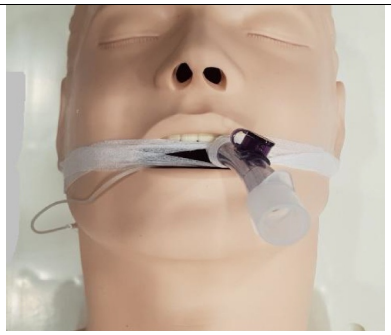
1. Non-commercial endotracheal tube (ETT) holding method; using adhesive tape with (or without) bite-block or airway	
2. Non-commercial ETT holding method; tying with gauze with (or without) bite-block or airway	
3. Commercial ETT holder	

Fig. 1. Endo-tracheal tube securing methods in this study.

rigid fixation, ease of application, reduce and relocation. However, commercial devices exert a greater force on the patient's face, and may cause destruction of mucosal integrity and frequent pain [9].

The oral mucosa consists of two layers: the surface stratified squamous epithelium and the deeper lamina propria; the epithelium is classified as non-keratinized or keratinized depending on the region of the mouth [13]. The skin consists of three layers: epidermis, dermis and hypodermis. The epidermis is a keratinized stratified squamous epithelium with four or five sub-layers; and the dermis and hypodermis are thick layers of connective tissue [14]. Histological differences make it difficult to discriminate the oral mucosa PU stage. Further, mucosal healing is clinically distinguished from skin healing in terms of speed and degree of scar formation [15]. An important characteristic of wound healing is re-epithelialization, where wound edges undergo proliferation and migration, and replace keratinocytes lost as result of injury [16]. Oral mucosa wounds show rapid re-epithelialization and reduction in scarring [17], suggesting a greater proliferative capacity of oral mucosa compared with general skin.

Based on these histological and healing characteristics, the skin PU staging system cannot be used to grade oral mucosa PUs. Standardised staging systems have been provided for assessing and documenting PUs that monitoring, reporting, communicating and management of wound progression [18,19]; however, the available staging systems are not designed to the mucosa; therefore, anatomically tissue comparisons cannot be made. Therefore, a stage classification system for oral mucosa PUs is required for patient care in the ICU, which would improve communication among clinicians, incident reporting, and monitoring of oral injuries [20], as well as enabling the examination of outcomes associated with particular risk factors [21].

The conceptual framework of PU development [22] describes biomechanical and physiological factors as critical determinants of PUs. Biomechanical factors include magnitude, time, and type of mechanical load [22]; physiological factors include PU status, perfusion, and nutrition [23]. In oral mucosa PUs, the accompanying mechanical loads on mucosal membranes are unknown. In addition, mucosa-specific mechanical loads must be determined in the context of medical device usage. Oral mucosa PUs may be elicited by ETT holders or the ETTs themselves [24]. Furthermore, bi-directional relationships between biomechanical and physiological factors have been reported previously [22].

To date, no clear differentiation between mucosa and traditional PUs has been identified [24]; and few studies have measured the

relationship between PU incidence, and biomechanical and physiological variables. Therefore, the current study sought to determine the PU incidence rates and to investigate the relationship between oral mucosa PUs and biomechanical/physiological risk factors.

2. Methods

2.1. Study design

This study used a prospective observational study design. Two steps were undertaken: 1) a prospective patient assessment of oral mucosa PUs and 2) a prospective medical record review.

2.2. Settings and participants

This study was conducted in a 1500-bed tertiary hospital with 8 ICUs in a metropolitan city in Korea. Permission to obtain PU data was obtained for 3 ICUs (1 neurosurgery ICU with 13- beds and 2 traumatic ICUs with 16- beds each). At two surgical ICUs, it was not possible to observe for longer than two days because patients were relocated to other wards. One ICU was temporarily closed due to relocation. At two-medical ICUs, researchers were not permitted to enter the ICU due to infection control. The nurse:patient ratio was 1:2; and the mean ICU stay was 7.1 days. Institutional PU prevention guidelines state that ETTs should be changed or repositioned every 8 h. In this hospital, the ETT was secured via non-commercial methods, such as adhesive tape and gauze string; or via single commercial device (MultiFix Endo-III; Fig. 1).

The inclusion criteria was: 1) > 18 years of age, 2) ETT use with non-commercial methods (adhesive tape, tied in gauze) or commercial ETT holders, and 3) consent for participation from patient family members. There was a limited number of observable intubated patients to examine the relationships between risk factors and oral mucosa PUs; therefore, we analysed patient-days rather than patients. One hundred forty-five patient-days from 21 patients were assessed, and 113 patient-days from 17 patients, were included in the final analysis. Twenty -six patient-days were excluded due to the absence of physiological data, 4 were excluded due to withdrawal of caregiver consent, and 2 were excluded due to lack cooperation from attending physicians.

2.3. Study procedures

2.3.1. Oral mucosa PU assessment

Observation of oral mucosa PUs were performed over 30 consecutive days which was the permission period granted by the IRB. Patients were observed from enrolment time (start of intubation, admission to ICU, or permission date) until finishing time (extubation, discharge from ICU, or death). The observer was a researcher who had performed mucous membrane studies with the authors' research group members and a wound, ostomy, and continence nurse (WOCN). The observer participated in a 4-h seminar and was trained by a dentist to assess PUs. Prospective patient assessment was conducted at the same time each day using the one-item oral mucosa PU scale [20]. Oral mucosa PUs are scored from 0 to 3 points according to the stage: zero = no PU; Stage 1 = redness and demarcation of the lip and buccal mucosa with no visible destruction or loss of epithelial tissue, ulceration, or blisters. In addition, non-blanchable erythema was present on the corners of the mouth; Stage 2 = destruction and differentiation of buccal mucosa, as manifested by blisters, soft coagulum, or clotting on mucosal tissue; superficial loss of non-keratinized epithelial tissue; or damage to epidermal and dermal layers of the corners of the mouth, without evidence of damage to underlying fascia; and Stage 3 = loss of mucosa and sub-mucosal tissue as evidenced by damage to/exposure of the fascia and underlying muscle in the lips or corners of the mouth.

The observer assessed two portions of the lower and upper oral mucosa in the not-eliminated ETT holder and recorded photographs of the oral cavity. Biomechanical load acts in the direction of two axes (force under the lower and upper lip acting downward by the movement of the jaw joint [14], force by movement of the ETT). We assumed the same pressure was loaded on each side because nurses changed location of ETT every 8 h; therefore, we did not separate between right and left. The observer recorded the absence or presence oral mucosa PUs and determined the stage. If more than two mucosal PUs were observed in the oral mucosa, the most severe stage of lesion was recorded. If the PU incidence could not be determined, the presence or absence was recorded after discussion with the research team based on observation documentation and photographs.

2.3.2. Medical record review

Medical records consisting of biomechanical and physiological factors were reviewed by a research assistant using a structured documentation form.

2.4. Instrument

2.4.1. Oral mucosa PUs

The Reaper Oral Mucosa Pressure Injury Scale (ROMPIS) [20] was used for classify PUs into stages. The inter-rater reliability (IRR) of this scale among experts from photographs is 0.443 [20]. We did not obtain permission to use photographs; therefore, we used this instrument alone, and sought to improve the reliability of the attributes by comparing with the mucositis staging system [21]. Translation and back-translation into Korean were conducted by two bilingual experts; content validity verification to assess the language and stage suitability of oral mucosa PUs was conducted using a 4- point Likert scale by two oral health experts. All items scored > 80% validity. To judge non-blanchable erythema, a 2-s press was administered to the lesion with a finger.

2.4.2. Risk factors

Previous literature has focused on risk factors for medical device-related PUs [1,2,5,6]. We have assessed the potential risk factors for oral mucosa PUs, which included of biomechanical and physiological variables, which were selected based on a conceptual framework [22] and systematic review for developing PUs [23] described in previous studies. Biomechanical conditions consisted of 5 items. 'Bite-block or airway use' and 'commercial ETT holder' were classified as Yes (coded 1) or No (coded 0). 'Duration of ventilator use' was calculated from the start of ventilator use until the time of observation. The ETT was usually changed to a tracheostomy within 2 weeks, approximately 75% patients with acute respiratory distress syndrome across 50 countries receiving a tracheostomy within 14 days [24]. Therefore, duration of ventilator use was classified with the half-life of ETT administration, 7-days, as the dividing factor.

The Richmond Agitation and Sedation Scale (RASS), measured by ICU staff nurses, was used for 'level of sedation' scoring. The RASS includes is a 10 point scale, ranging from - 5 to 4. In this study, negative scores we were recorded for all patients, which indicated the level of sedation was drowsiness, light, moderate, and deep sedation. Patients were classified as above - 3 and below - 4 if they responded to voice [25]. To score the 'risk of general PUs,' we used daily mean total score recorded on the Braden scale. Patients were usually monitored two times a day by ICU staff nurses, and total score close to the time of oral observation was used. Patients were classified into low and high risk groups. We could not use the APACHI II score to reflect severity because these data were not available for most patients.

Physiological data consisted of 11 items. 'Sedative' and 'vasopressor' use were classified as Yes (coded 1) or No (coded 0). 'Fraction of inspired oxygen (FiO2)' was recorded as the percentage of inspired oxygen at the observation time. 'PaO2 (arterial pressure of oxygen)', 'white blood cell', 'haemoglobin', 'haematocrit', 'plasma protein', 'serum albumin', 'BUN', and 'serum creatinine' were also recorded.

2.5. Data analysis

Data were analysed using SPSS 23.0 (SPSS, Inc., Chicago, IL, USA). The Shapiro-Wilk test was used to check for normality. Risk factor variables were checked for sphericity using Mauchly's test for sphericity [26]. Sphericity is a measure of how equal the variances of the differences between all possible pairs in the within-subject conditions. The degrees of relevance between all time points should be constant for analysis of variance tests [27]. These variables met the assumption of sphericity. Descriptive statistics were used to describe the incidence of oral mucosa PUs, as well as biomechanical and physiological factors. Incidence rate was calculated by dividing the number of patient-days with an oral mucosa PU staging system score > 1 by the total number of patient-days. This value was represented as a percentage by multiplying by 100. Comparisons between subgroups of categorical variables within the biomechanical and physiological characteristics were calculated using the χ^2 or Fisher's exact test. To identify relationships between the incidence of oral mucosa membrane PU and research variables, we performed Spearman's rank correlation analysis.

2.6. Ethical consideration

This study was approved by the Institutional Review Board (No. H-1708-014-057). Additional approvals were obtained from severe trauma team and nursing department. During the participant recruitment period, the purpose, voluntary nature of participation, confidentiality of information, and procedures of the study were explained to family members. All participants were sedated or severely mentally compromised; therefore, family members were considered as the legal representative with power of attorney, based on the International Council for Harmonization [28]. Informed consent was obtained from each family.

3. Result

3.1. Characteristics of oral mucosa PU incidence rates and progression

The mean incidence rates of oral mucosa PUs in the lower, upper, and combined sites were 36.3%, 11.5% and 7.1%, respectively (Fig. 2). Fig. 3 shows an example of the progression of oral mucosa PUs. The patient was 71 years old and had undergone an operation to remove an intracranial haemorrhage. We observed stage 2 lower oral mucosa PUs with stage 2 on the second day of ICU admission. These progressed stepwise from stage 2 to normal after 4 days, then showed recurrence at stage 2, stage 1, and stage 2 for the last 3 days.

3.2. Incidence of oral mucosa PUs according to biomechanical and physiological factors

Oral mucosa PU incidence according to biomechanical and physiological factors is shown in Table 1. In the lower oral mucosa, incidence of mucosal PUs differed according to bite-block or airway ($p = .047$) and sedative ($p = .021$) use. There was no significant difference in incidence between characteristics in upper mucosal PUs. The incidences of mucosal PUs in when sites were combined differed according to commercial ETT holder use ($p = .040$).

3.3. Correlation between PU stage and biomechanical or physiological factors

Table 2 shows the correlation between lower, upper, and combined site PU incidence, and multiple biomechanical and physiological factors. There were significant correlations between the lower oral mucosa PU stage and bite-block or airway use ($r = 0.20$, $p = .036$), commercial ETT holder use ($r = 0.19$, $p = .048$), sedative use ($r = -0.22$, $p = .022$), and plasma protein ($r = 0.20$, $p = .033$). Upper oral mucosa PU stage was correlated with commercial ETT holder use ($r = 0.19$, $p = .044$), haemoglobin($r = 0.24$, $p = .011$), haematocrit ($r = 0.27$, $p = .004$), and serum albumin ($r = -0.24$, $p = .012$). Combined site stage was correlated with commercial ETT holder use ($r = 0.28$, $p = .003$), haematocrit ($r = 0.19$, $p = .039$), and serum albumin ($r = -0.23$, $p = .015$).

4. Discussion

Current guidelines state that oral mucosa PUs must be diagnosed without staging [29]; however, a consensus for staging of mucosal PUs has been developed [30]. Few studies have assessed PUs with lower incidence rates when compared with general skin PUs. Notably, mucous membranes are located in diverse areas and can be frequently injured by equipment or catheters [30]. Therefore, mucosal PUs may occur more frequently than has been previously recorded. This study sought

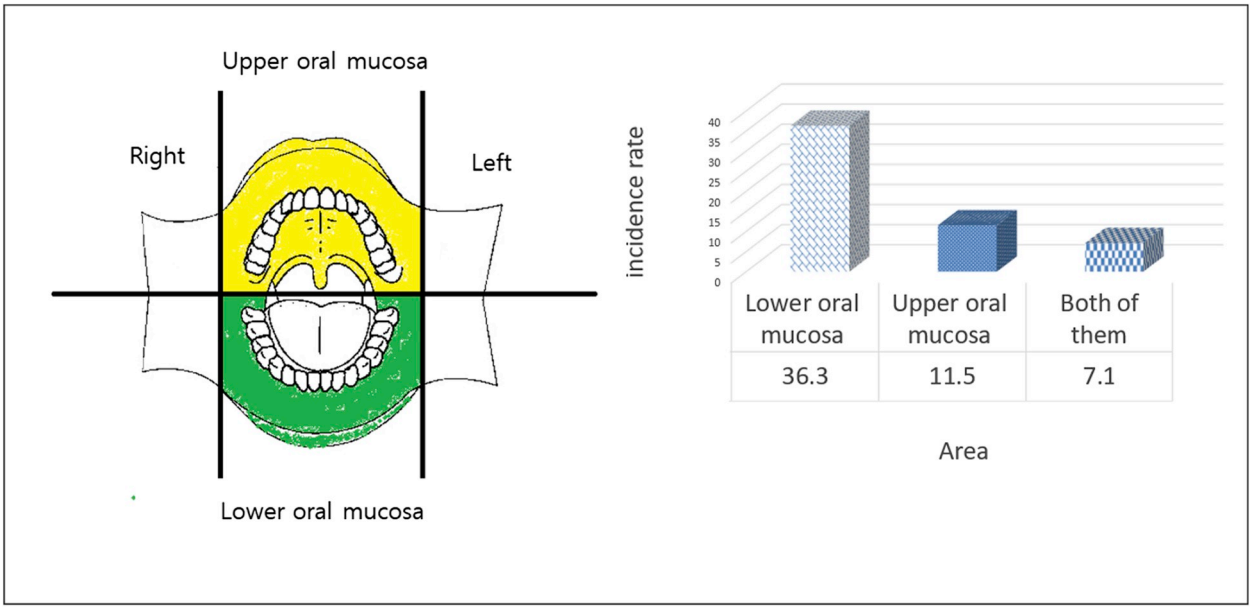


Fig. 2. The incidence rates of oral mucosal pressure ulcers.

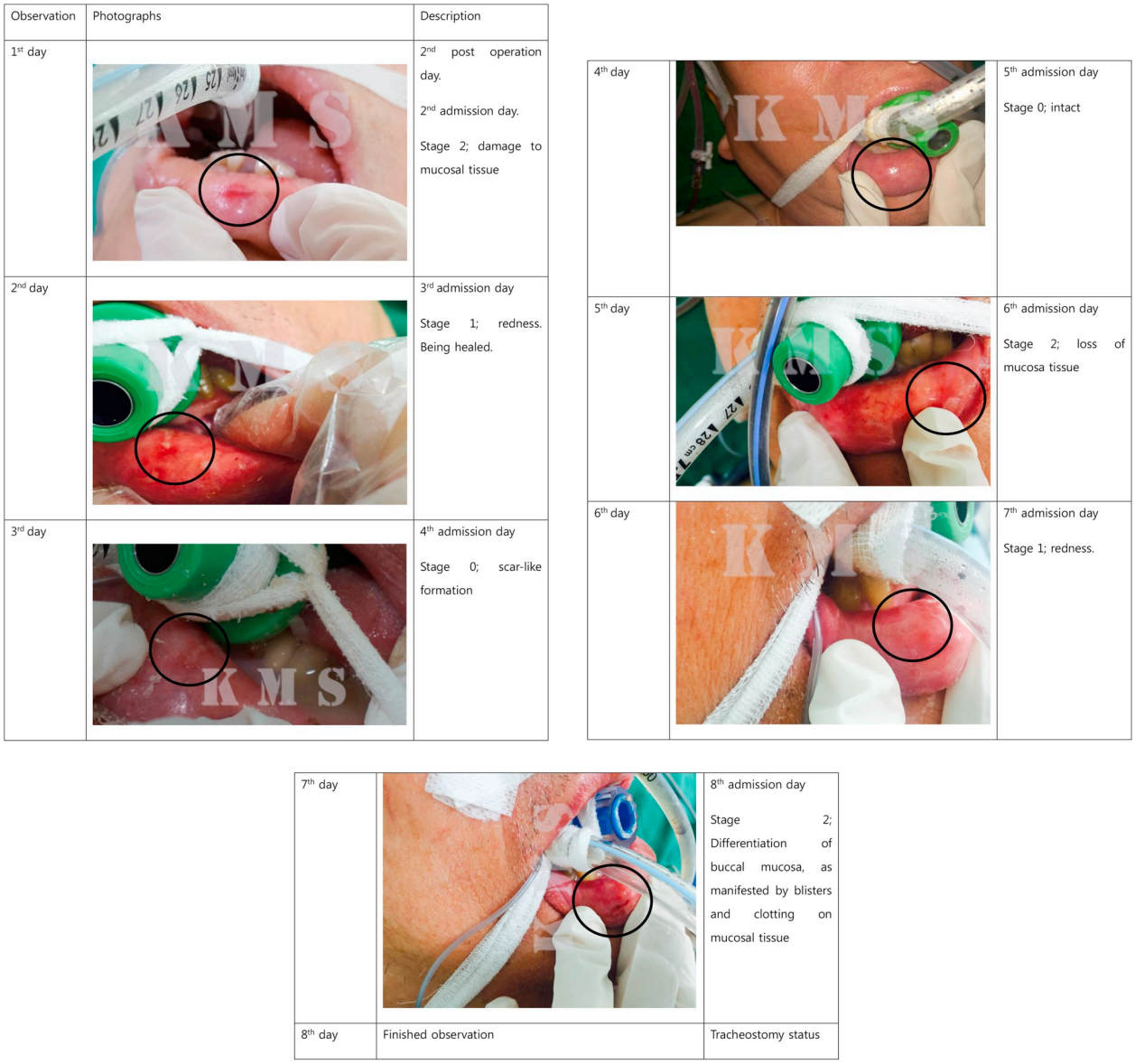


Fig. 3. An example of oral mucosa pressure ulcers progression.

to address the incidence of oral mucosal PUs and their relationship to biomechanical and physiological factors.

The incidence rate of PUs in the lower oral mucosa were relatively high in this study compared with previous studies, which report a 27.9% overall incidence of medical device-related PUs [31] and ETT-related PUs accounted for 22.6% of all documented PUs [32]. The use of an oral mucosa PU monitoring scale may have helped to detect additional mucosal PUs. In our study, one single observer endeavoured to accurately record stage 1 PUs, which may be easily missed. Therefore, a monitoring scale may enhance more precise staging and early detection of oral mucosa PUs. The incidence rates of lower oral mucosa PUs were high in this study due to mastication. Because chewing is primarily an unconscious act [14], patients may chew the bite-block or airway. Bite-block and airway made from plastic or hard materials which would create pressure on the lower lip that led to a pressure ulcer on the oral mucosa.

In one case, we observed various a change in PUs from stage 2, stage 1, to normal, then stage 1 and stage 2 for 7 days; however, we cannot confirm that this it represents the general incidence of oral mucosa PUs. However, the long-term prognosis of device-related PUs is better than general skin PUs [33], and wound healing in oral mucosa is clinically more rapid than general skin [15]. The exact mechanisms between scarless oral wounds and scar forming skin healing are unknown; however, oral wounds show lower expression of inflammatory markers, such as transforming growth factor (TGF)-β1, which plays a crucial role in tissue regeneration, cell differentiation, and embryonic development. As a result, oral mucosa fibroblasts demonstrated resistance to TGF-β1-driven fibroblast-myofibroblast differentiation, thereby retained their 'non-scarring' from and proliferated faster than the skin counterparts [34]. However, oral mucosa PUs occur more frequently at multiple sites, and they may develop and heal repeatedly and overlap. This made lead to a worsening beyond the mucosa wound and affect the respiratory or systemic health. Therefore, it is important to develop protocols for reducing the recurrence of oral PUs.

Commercial ETT holder use was positively correlated with PU staging in the lower, upper and combined oral mucosa sites. In a previous study, less time was required to move the ETT when using commercial ETT holders, compared with non-commercial techniques [10,32]. Further, the commercial ETT holder in this study anchored the upper jaw tightly, and commercial holders use improved ETT immobility. However, it may worsen the level of pressure and perfusion on the face and mouth because of close contact between the upper oral mucosa and

Table 1
Incidence of oral mucosa pressure ulcers according to biomechanical and physiological characteristics (N = 113).

			Incidence of oral mucosal pressure ulcers					
			Lower site		Upper site		Combined sites	
			Yes	No	Yes	No	Yes	No
Bite-block or airway use	Yes	97(85.8)	39(40.2)	58(59.8)	13(13.4)	85(86.6)	8(8.2)	89(91.8)
	No	16(14.2)	2(12.5)	14(87.5)	0(0.0)	16(100.0)	0(0.0)	16(100)
	χ^2 (p) or (p)		**(.047)**		(.209)		(.598)	
Commercial ETT holder	Yes	5(4.4)	4(80.0)	1(20.0)	2(40.0)	3(60.0)	2(40.0)	3(60.0)
	No	108(95.6)	37(34.3)	71(65.7)	11(10.2)	97(89.8)	6(4.6)	102(94.4)
	χ^2 (p) or (p)		(.057)		(1.000)		**(.040)**	
Duration of ventilator Use (days)	~6	68(60.2)	24(35.3)	44(64.7)	7(10.3)	61(89.7)	5(7.4)	63(92.6)
	7~	45(39.8)	17(37.8)	28(62.2)	6(13.3)	39(86.7)	3(6.7)	42(93.3)
	χ^2 (p) or (p)		.07(.843)		.25(.765)		(1.000)	
Level of sedation (RASS score)	Above −3	26(23.0)	12(46.2)	14(53.8)	3(11.5)	23(88.5)	2(7.7)	24(92.3)
	Below −4	87(77.0)	29(33.3)	58(66.7)	10(11.5)	77(88.5)	6(6.9)	81(93.1)
	χ^2 (p) or (p)		1.42(.252)		(1.000)		(1.000)	
Risk of general PUs (Braden score)	Low risk(13~)	12(11.0)	3(25.0)	9(75.0)	0(0.0)	12(100.0)	0(0.0)	12(100.0)
	High risk(6–12)	101(89.0)	38(37.6)	63(62.4)	13(12.9)	88(87.1)	8(7.9)	93(92.1)
	χ^2 (p) or (p)		(.531)		(.354)		(.597)	
Sedatives use	Yes	93(82.3)	29(31.2)	64(68.8)	12(12.9)	81(87.1)	7(7.5)	86(92.5)
	No	20(17.7)	12(60.0)	8(40.0)	1(5.0)	19(95.0)	1(5.0)	19(95.0)
	χ^2 (p) or (p)		**5.91(.021)**		(.458)		(1.000)	
Vasopressor use	Yes	49(43.4)	20(40.8)	29(59.2)	6(12.2)	43(87.8)	5(10.2)	44(89.8)
	No	64(56.6)	21(32.8)	43(67.2)	7(10.9)	57(89.1)	3(4.7)	61(95.3)
	χ^2 (p) or (p)		.77(.432)		.05(1.000)		(.290)	

Table 2
Correlations among Incidence of Oral Mucosa Pressure Ulcer, Biomechanical and Physiological variables (N = 113).

Categories	Variables	Stage of oral mucosa pressure ulcers		
		Lower site	Upper site	Combined sites
Stage of PUs	Lower oral mucosa	1.00		
	Upper oral mucosa	**.24(.012)**	1.00	
	Both of them	**.43(< .001)**	**.76(< .001)**	1.00
Biomechanical variables	Bite-block or airway use	**.20(.036)**	.15(.122)	.11(.237)
	Commercial ETT holder use	**.19(.048)**	**.19(.044)**	**.28(.003)**
	Duration of ventilator use	-.01(.944)	.06(.507)	.03(.776)
	Level of sedation (RASS score)	-.01(.892)	.08(.378)	.07(.435)
	Risk of general PUs (Braden score)	-.03(.784)	-.07(.448)	.02(.845)
Physiological variables	Sedatives use	**-.22(.022)**	.10(.316)	.04(.693)
	Vasopressor use	.11(.245)	.02(.855)	.11(.261)
	Fraction of inspired oxygen	-.03(.727)	-.16(.099)	-.05(.611)
	PaO2	-.15(.106)	-.14(.137)	-.10(.305)
	White Blood Cell	-.04(.678)	-.07(.443)	-.04(.705)
	Hemoglobin	.16(.088)	**.24(.011)**	.17(.069)
	Hematocrit	.18(.057)	**.27(.004)**	**.19(.039)**
	Plasma protein	**.20(.033)**	-.09(.372)	-.16(.092)
	Serum albumin	-.01(981)	**-.24(.012)**	**-.23(.015)**
	Blood Urea Nitrogen	.14(.148)	-.08(.425)	-.07(.459)
	Serum creatinine	.10(.276)	.07(.477)	.06(.551)

maxilla bone. The upper oral mucosa may be damaged as general skin ulcers usually occur over a bony prominence as a result of the combination of pressure and friction [18,19]. Although this may not induce complete obstruction of the vessels for 24 h or more, static pressure for 8 h without changing location may be possible. Therefore, the use of commercial ETT holders could produce capillary obstruction-associated complications [32], including reduced tissue perfusion and mucosal PUs. Further, because commercial ETT holders with an integrated bite-block interfere with ETT repositioning and oral hygiene care by preventing access to the oral cavity [10]; therefore, PUs development at both sites can occur.

The Braden scale score did not correlate with the risk of oral mucosa PUs, although a previous study has shown that a lower Braden score is a risk factor for development of medical device -related PUs [35]. Oral mucosa PUs in ventilated patients are related to medical device use and bedbound status, it is noteworthy that the skin PU risk score is not related to the risk of oral mucosa PUs. There are two possible explanations. First, risk score detection in this study was conducted for the buttocks (coccyx and ischium) or heels, which are distant from the mouth. In addition, the back of the head could not be assessed in many of the patients in this study due to head dressings. If PU risk score detection was conducted for the face or back of the head, a relationship might be observed. Second, the characteristics of the Braden scale itself may play a role in this lack of relationship. According to a meta-analysis, the predictive validity of pressure ulcer risk in the Braden scale is interpreted as a moderate [36]; therefore, an additional scale to assess the risk of general PUs is required.

Sedative use showed a significant negative correlation with PU staging in the lower oral mucosa. Non-sedated patients predominantly had ulcers related to equipment in a previous study [33]. Similarly, non-sedated patients had a higher staging of mucosa PUs in this study. Non-sedated patients are able to change position in bed in response to

discomfort, which is important for preventing traditional PUs. However, they had a higher rate of device-related PUs because equipment can move due to their movements, causing pressure or friction, which may not be noticed or difficult to re-adjust [33]. Patients in this study were not completely awake, based on the sedation score; they might bite their bite-block or commercial ETT holder with greater power due to discomfort and cause pressure or rub on the lower oral mucosa.

Vasopressor use correlated with PU development [37]. Vasopressor agents induce vasoconstriction to elevate mean arterial pressure and counteract inadequate tissue perfusion; therefore, this may be a contributing factor for the development of PUs [37]. However, there was no significant relationship with oral mucosa PU staging. Vasopressor use should be recorded, in terms of hours of administration of vasopressor agents and dose [38], or duration of vasopressor infusion [39]. These prior studies showed that agent, dose, and duration were important for PU development. However, these were coded as yes or no in this study, because the preliminary study grossly attempted to identify relevant variables for development of oral mucosa PUs.

Haemoglobin was positively related with upper oral PU staging, and haematocrit was positively related with PU staging in upper and combined of oral mucosa sites. Anemic status is associated with an odds ratio of 2.23 for pressure ulcers [40] and haematocrit is a predictive factor for hospital-acquired PUs [41]. However, the relationship between haemoglobin, haematocrit, and oral mucosa PU incidence was reversed in this study. There are two possible explanations. First, there may be confounding variables, such as other physiological factors. Second, haemoglobin and haematocrit may simply exhibit low sensitivity to oral mucosa PUs.

Lower serum albumin levels were associated with advanced PU stage incidence in upper and combined sites. Serum albumin is an important predictor of general PU development and wound healing of patients in acute/postoperative conditions [42]. In addition, it is an indicator of nutritional status because albumin comprises > 50% of serum proteins [43]. Further, metabolic acidosis is associated with low serum albumin, resulting in a deterioration of PUs and delayed wound healing [44]. Based on the relationship between serum albumin and general PUs, we suspect that the low serum albumin level was a potential risk factor for the development of oral mucosa PUs. However, serum albumin is not significantly associated with ulcer healing of patients in chronic/palliative conditions [42] and there was no correlation with lower oral mucosa PUs in this study. Further studies should be conducted to examine the relationship between these two variables.

This study has yielded useful data on the incidence rate of oral mucosa PUs and relationships between biomechanical and physiological factors, but it has a number of limitations. First, other risk factors and clinical treatment factors for PU development were not included in this study because a lack of consecutive -day data and a limited study population. Second, the study findings were based on a small number of participants during a short-study period. Third, severity of the patients could not be considered because of lack of data availability. Therefore, more discursive risk factors, larger sample size, and actual acuity of the patients would rectify these limitations.

5. Conclusion

Oral mucosa PUs were developed frequently and healed quickly. The high incidence rates of oral mucosa PUs may be due to our use of an assessment tool. This study has shown the necessity of oral mucosa PU classification by stages to estimate more precise incidence rates. Biomechanical factors, such as non-commercial and commercial ETT holders; and physiological variables, such as sedative use, haemoglobin, haematocrit, and serum albumin, are associated with oral mucosa PU incidence. Therefore, it is important to consider biomechanical and physiological factors to reduce PU incidence in patients in ICU. Further investigations are required to examine more detailed factors associated oral mucosa PU development.

Conflicts of interest

None.

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