



## Sequential alterations of Stensen's duct and parotid gland after radical surgeries in buccal cancer

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### ABSTRACT

**Objectives:** The marsupialization of Stensen's duct after buccal cancer excision and free flap reconstruction has seldom been reported. In this study, we evaluated the alteration in Stensen's duct and parotid gland, without marsupialization or relocation, between the time of surgery and 24 months postoperatively to determine whether ductal management is needed in patients with buccal squamous cell carcinoma (BSCC).

**Methods:** Eighty-five patients with BSCC receiving primary radical surgery and free flap reconstruction were recruited. Alterations in Stensen's duct and parotid gland were assessed by computed tomography during the postoperative period.

**Results:** The 81 males and 4 females enrolled in study had a tumor status of cT2 (n = 52, 61%) or cT3 (n = 33, 39%). In total, 52 (61%) patients received surgery alone, and 33 (39%) received adjuvant concurrent chemoradiotherapy (CCRT) postoperatively. Stensen's duct on the affected side was significantly dilated compared to the non-affected side (p < 0.001). The difference in diameter of Stensen's duct between the surgery plus CCRT group and the surgery alone group was not significant (p > 0.05), indicating that changes in parotid gland occurred mainly due to surgery. In both the surgery and surgery plus CCRT groups, inflammation of parotid gland had regressed by 24 months.

**Conclusions:** Stensen's duct in BSCC dilatation peaked in the 3rd month after surgery. Changes in parotid gland on the surgically treated side regressed into fatty change by 24 months after surgery.

### Introduction

The buccal mucosa is the most frequent site of oral squamous cell carcinoma [1]. Patients with buccal squamous cell carcinoma (BSCC) are usually treated by radical surgery and free flap reconstruction in combination with adjuvant concurrent chemoradiotherapy (CCRT). To achieve clearance of BSCC and negative margins, the primary excision commonly sacrifices the peripheral division of Stensen's duct. If the duct is resected or ligated without management, several complications such as sialocele, fistula and delayed wound healing would occur [2], resulting in delayed adjuvant CCRT. However, the preservation, relocation or re-anastomosis of Stensen's duct during BSCC surgery has

been largely ignored, although a recently introduced procedure consists of cannulating the Stensen's duct and draining the saliva into the different sites of oral cavity [2]. Nevertheless, the relocation and anastomosis of the parotid duct during BSCC surgery are not routine procedures in most medical centers and thus have not been widely studied. Given the controversy associated with the relocation of Stensen's duct in patients undergoing BSCC surgery, we retrospectively analyzed 85 patients with T2 and T3 BSCCs treated with primary radical surgery and free flap reconstruction. In all of these patients, Stensen's duct was transected without ligation or marsupialization during surgery. We recorded changes in Stensen's duct and the parotid gland during the post-operative period with the aim of determining whether routine

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relocation or re-anastomosis of Stensen's duct are needed in these patients.

## Materials and methods

The 85 patients in whom tumor status staged as cT2 or cT3 BSCC were recruited after a retrospective review of 360 consecutive patients with BSCC treated with primary radical surgery between March 2003 and March 2018. The 2010 guidelines of the American Joint Committee on Cancer (AJCC) were adopted for TNM staging [3].

Pre-operative tumor staging examinations, consisting of a detailed medical history, physical examination, hematological investigations and routine blood biochemistry, and chest radiographs, were performed in all patients. Tumor status was assessed using computed tomography (CT) or magnetic resonance imaging (MRI) of the head and neck, abdominal sonography, and positron emission tomography or bone scan.

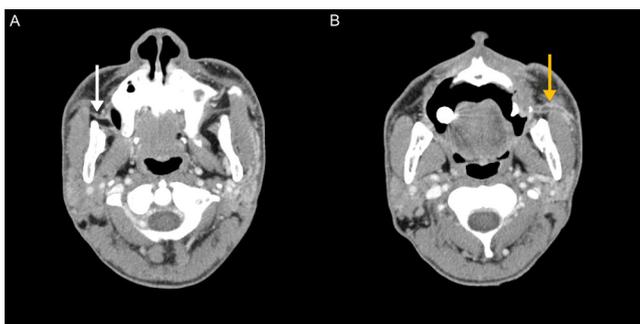
The primary buccal tumor was excised with a margin of at least 10 mm and negative resection margins were confirmed using frozen sections prepared from intraoperatively obtained specimens. Depending on the tumor extent, marginal mandibulectomy, or inferior maxillectomy was conducted. Following wide excision of the tumor, reconstruction with a free flap was performed by the plastic surgeon. The free flap consisted of an anterolateral thigh flap, anteromedial thigh flap, or a forearm flap, depending on the plastic surgeon's judgment, the surgical site, the structures near the defects and the availability of blood vessels.

Postoperative adjuvant CCRT was indicated for patients positive for any major pathological parameter (extranodal extension, a positive margin, or pN2b) or any three minor pathological parameters (pT4, pN1, margin  $\leq$  4 mm, poor differentiation, perineural invasion, vascular invasion, lymphatic invasion, and tumor depth  $\geq$  10 mm) and was planned for within 6 weeks after surgery. Radiation therapy was given five times per week in 33 fractions for a total of 66 Gy [4,5]. Concurrent chemotherapy consisted of cisplatin-based chemotherapy was given [4,6–10].

After surgery, all of the patients were followed by periodic examinations in the clinic. CT was performed at 3, 6 and 12 months postoperatively during the first year and annually during the following 5 years.

### Measurement of Stensen's duct

On the non-surgical side (Fig. 1A), Stensen's ducts closely abutted



**Fig. 1.** (A) Measurement of Stensen's duct on the non-surgical side: The duct closely abuts the anterior margin of the masseter muscle. A contrast-enhanced venule ventral to the ductal insertion site in the buccal mucosa is an important landmark in the correct identification of Stensen's duct (white arrow). (B) Measurement of Stensen's duct on the surgical side: resection of the duct medial to the masseter muscle resulted in filling of tram-like structures filled with fluid and wall enhancement ventral or lateral to the muscle, or even inside the parotid gland. The structures most likely represent the remnant of Stensen's duct (yellow arrow). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

the anterior margin of the masseter muscle. An important landmark allowing identification of the duct is the venule located ventral to the ductal insertion site into the buccal mucosa, as detected on contrast-enhanced CT. The ductal segment is widest between this insertion site and the anterior masseter muscle margin and can thus be easily visualized; this widest point of the ductal segment was used for measurement in this study. On the surgical side (Fig. 1B), ductal segments medial to the masseter muscle were resected. A tram-like structure filled with fluid, showing wall enhancement, and located lateral to the masseter muscle or inside the parotid gland was considered the remnant of Stensen's duct and its widest diameter was measured. Additional measurements to compare the affected and contralateral parotid glands were made and scored by the radiologist (Dr. Chin S.C.), as follows: 1 = normal, 2 = inflammation, 3 = fatty change (atrophy). The score represented the postoperative course of the parotid gland over time.

### Exclusion criteria

Patients with cT1 and cT4 of BSCC were excluded from the study; only those with cT2 and cT3 BSCCs were included, for several reasons: (1) To homogenize the defects as much as possible, the study population was limited to patients with inner mucosal and muscular defects without skin sacrifice. Patients not receiving free flaps were also excluded. (2) Advanced-stage tumors have complicated structures that directly influence the patency of Stensen's duct and the function of the parotid gland. (3) Our aim was to compare the ductal changes between patients treated by surgery alone versus surgery plus adjuvant CCRT. Most of the reviewed T1 patients underwent surgery alone, whereas most of the T4 patients received surgery plus adjuvant CCRT. Other grounds for exclusion were: the absence of free flap reconstruction, severe postoperative deep neck space infection, residual cancer, cancer recurrence, or second primary cancers of the oral cavity diagnosed within the first 2 years follow-up. The patients with the ligation or the marsupialization of Stensen's duct was excluded as well. To minimize the bias caused by differences among imaging studies, patients who had post-operative MRI at follow-up were also excluded.

After applying these criteria, our study population comprised 85 patients. Their medical records and images were analyzed retrospectively in detail.

### Statistical analysis

The data were analyzed using MedCalc software (ver. 18.6; MedCalc, Ostend, Belgium). Because a preliminary Kolmogorov-Smirnov test showed that the data were not normally distributed, Wilcoxon signed-rank, Mann-Whitney and Kruskal-Wallis tests were used to evaluate differences and relationships between categorical parameters. A  $p$  value  $<$  0.05 was considered indicative of a statistically significant difference.

### Ethics statement

This study was approved by the Institutional Review Board (IRB) of the Chang Gung Medical Foundation (IRB no. 201800734B0). The data were collected retrospectively, and the patients were anonymized before data analysis.

## Result

The 81 males and 4 females (Table 1) enrolled in the study had a mean age of  $51.16 \pm 10.92$  years. The primary BSCC status according to the AJCC tumor staging criteria was cT2 in 52 (61%) patients and cT3 in 33 (39%) patients. In the first month after surgery, seven patients had parotid gland sialadenitis on the surgical side and one patient had a parotid gland abscess; these were treated by antibiotics. Two patients had large sialoceles ( $>$  5 cm) in the parotid gland and were managed by

**Table 1**  
Clinicopathological characteristics in 85 patients.

Characteristics	N (%)
Sex	
Male	81 (95)
Female	4 (5)
Stage	
cT2	52 (61)
cT3	33 (39)
Treatment	
Surgery alone	52 (61)
Surgery with adjuvant CCRT	33 (39)
Free flap	
Anterolateral thigh flap	60 (70)
Profunda artery perforator flap	5 (6)
Fibular osteoseptocutaneous flap	5 (6)
Radial flap (forearm)	4 (5)
Ulnar flap	3 (4)
Anteromedial thigh flap	3 (4)
Medial sural artery perforator flap	2 (2)
Vastus lateralis flap	1 (1)
Tensor fascia lata flap	1 (1)
Rectus femoris perforator flap	1 (1)

N = numbers; CCRT = concurrent chemoradiotherapy.

repeated aspiration and compression. Between months 3 and 6 after surgery, five patients had small sialoceles (1–2 cm in size) that were managed by observation alone. Twenty-one patients had clinical mild parotitis on the surgical side but did not receive any medication. Five patients developed a small abscess in the parotid gland area and were successfully treated by oral antibiotics.

During the postoperative period, 52 (61%) patients were observed and 33 (39%) received adjuvant CCRT. Thus, the patients were first divided into surgery alone (n = 52) and surgery plus adjuvant CCRT (n = 33) groups. Based on CT scans performed at 3, 6, 12, and 24 months postoperatively, ductal size on the affected (surgical) and non-affected (non-surgical) sides was evaluated in each patient, as shown in Table 2. In all of the studied patients, the ductal diameters were significantly larger on the affected than the non-affected side (Table 2,  $p < 0.001$ ). In the surgery alone group, a comparison of the affected and non-affected sides of each patient showed that the mean diameter of Stensen's duct was significant larger on the affected than the non-affected side at 3, 12, and 24 months postoperatively (all  $p$ -values  $< 0.05$ ; Table 2). In the surgery plus adjuvant CCRT group, the ductal diameter was also larger on the affected at 3, 6, 12, and 24 months (all  $p$ -values  $< 0.05$  vs. the non-affected side at the same time-point; Table 2).

A comparison of the affected and non-affected sides in patients who had surgery alone and those who had surgery plus adjuvant CCRT was also performed (Table 3). None of the differences among the subgroups reached statistical significance, suggesting that surgery plays a major causative role in ductal dilation.

**Table 2**

Comparison of the same patient's ductal size (mm) between affected side and non-affected side in different treatment course.

Time (M)	Surgery alone			Surgery and CCRT			All		
	Affected	Non-affected	$p$ value	Affected	Non-affected	$p$ value	Affected	Non-affected	$p$ value
3	4.10 ± 1.95	2.51 ± 1.04	<b>&lt; 0.001*</b>	3.77 ± 1.35	2.42 ± 0.75	<b>0.020*</b>	4.02 ± 1.81	2.49 ± 0.97	<b>&lt; 0.001*</b>
6	2.92 ± 1.24	2.45 ± 0.75	0.098	3.11 ± 0.62	2.16 ± 0.32	<b>0.004*</b>	2.98 ± 1.11	2.37 ± 0.67	<b>&lt; 0.002*</b>
12	3.70 ± 1.90	2.36 ± 1.03	<b>&lt; 0.001*</b>	3.24 ± 1.22	2.19 ± 0.52	<b>0.020*</b>	3.58 ± 1.75	2.31 ± 0.92	<b>&lt; 0.001*</b>
24	3.00 ± 1.43	2.30 ± 1.05	<b>0.002*</b>	3.22 ± 1.44	2.22 ± 0.51	<b>0.017*</b>	3.06 ± 1.42	2.28 ± 0.93	<b>&lt; 0.001*</b>

CCRT = concurrent chemoradiotherapy; M = months.

Affected = mean and standard deviation of ductal size of surgical side.

Non-Affected = mean and standard deviation of ductal size of non-surgical side.

\* =  $p$  value  $< 0.05$ . Significant differences are shown in bold.

The change in ductal diameter on the surgical side at 3, 6, 12, and 24 months postoperatively was also analyzed, as shown in Fig. 2A, and was determined to be significant ( $p = 0.015$ ). The respective means were  $4.10 \pm 1.95$ ,  $2.92 \pm 1.24$ ,  $3.70 \pm 1.90$ , and  $3.00 \pm 1.43$  mm. Specifically, in the surgery alone group, there was a significant difference between 3 and 6 months ( $p = 0.012$ ), and between 3 and 24 months ( $p = 0.011$ ), postoperatively. A similar analysis of the diameter of Stensen's duct in the surgery plus adjuvant CCRT (Fig. 2B) did not reveal a significant difference ( $p = 0.831$ ). The means at 3, 6, 12 and 24 months were  $3.77 \pm 1.35$ ,  $3.11 \pm 0.62$ ,  $3.24 \pm 1.22$  and  $3.22 \pm 1.44$ , respectively, but none of the differences were statistically significant.

Changes in parotid gland, as seen on CT scans at the four postoperative follow-up examinations, were evaluated with score by the radiologist. In the surgery alone group, the mean score at 3, 6, 12 and 24 months was  $1.41 \pm 0.50$ ,  $1.63 \pm 0.61$ ,  $1.95 \pm 0.86$  and  $2.46 \pm 0.84$ , respectively. As shown in Fig. 3A, the increase over time was significant ( $p < 0.001$ ). By contrast, in the surgery plus adjuvant CCRT group, the respective mean score was  $1.45 \pm 0.69$ ,  $1.76 \pm 0.44$ ,  $1.67 \pm 0.66$  and  $2.15 \pm 0.93$ . Despite the increasing trend in score (Fig. 3B), the difference over time was not significant ( $p = 0.089$ ).

Fig. 4A shows the status of the parotid gland on the affected side in patients undergoing surgery alone. The percentages of patients with a postoperative score of 1 (normal) decreased from 59% at 3 months to 44% at 6 months, 39% at 12 months and 22% at 24 months. A similar trend was seen for patients with a score of 2 (inflammation), which decreased from 41% at 3 months to 11% at 24 months postoperatively. The percentage of patients with parotid gland atrophy (score of 3) increased from 0% at 3 months to 6% at 6 months, 34% at 12 months to 67% at 24 months.

Fig. 4B shows the changes in the parotid gland in patients in the surgery plus adjuvant CCRT group. The trend was similar to that in the surgery alone group, with parotid gland atrophy detected in 50% of the patients after 24 months (9% of patients after 3 months, 0% after 6 months, 9% after 12 months).

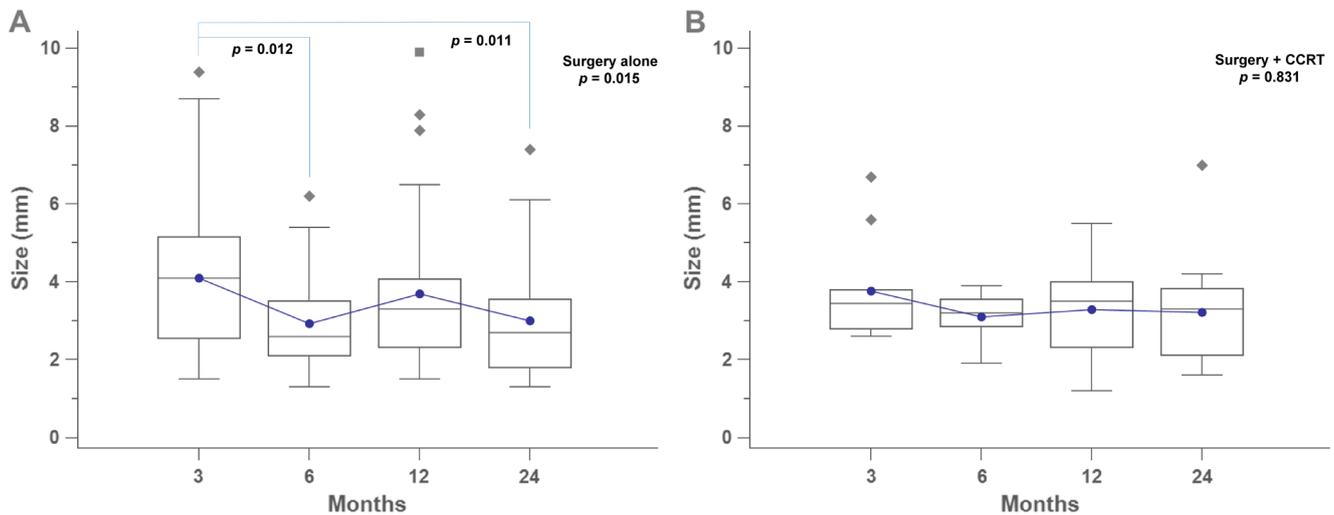
## Discussion

Our study demonstrated postoperative, sequential alterations in Stensen's duct and the parotid gland at 3–24 months after buccal cancer radical excision and free flap reconstruction. In primary surgery for BSCC, a 10-mm margin for cancer clearance is mandated, which frequently results in injury or transection of the duct during radical surgery. According to the protocol of our hospital, adjuvant CCRT must be implemented within 6 weeks after confirmation of multiple adverse pathological findings such as tumor depth  $\geq 10$  mm, perineural invasion, multiple lymph nodes metastasis and extra-nodal invasion. As shown in Table 2, surgery with and without CCRT both led to an increase in ductal size that was significant in all subgroups at every time-point, except at 6th month in the surgery alone group. When we compared the surgery plus CCRT with surgery alone patients, as shown

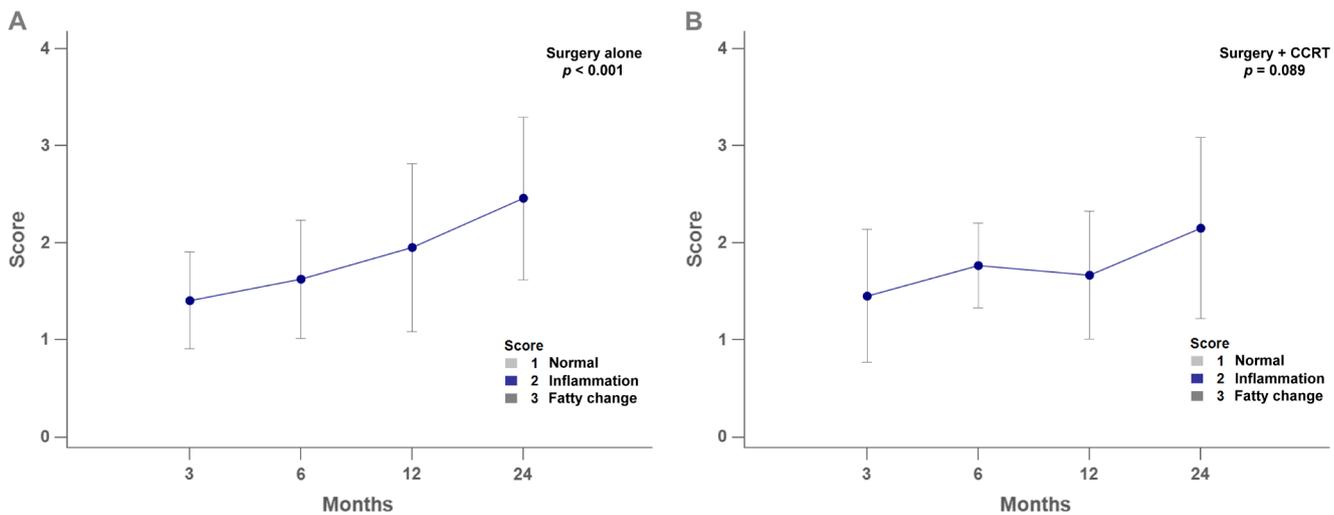
**Table 3**  
Comparison of ductal size (mm) about affected side and non-affected side between those with surgery alone and those with surgery and adjuvant CCRT.

Time (M)	Affected			Non-affected		
	Surgery alone	Surgery and CCRT	<i>p</i> value	Surgery alone	Surgery and CCRT	<i>p</i> value
3	4.10 ± 1.95	3.77 ± 1.35	0.723	2.51 ± 1.04	2.42 ± 0.75	0.781
6	2.92 ± 1.24	3.11 ± 0.62	0.206	2.45 ± 0.75	2.16 ± 0.32	0.344
12	3.70 ± 1.90	3.24 ± 1.22	0.762	2.36 ± 1.03	2.19 ± 0.52	0.698
24	3.00 ± 1.43	3.22 ± 1.44	0.516	2.30 ± 1.05	2.22 ± 0.51	0.477

CCRT = concurrent chemoradiation therapy; M = months.  
Affected = mean and standard deviation of ductal size of surgical side.  
Non-affected = mean and standard deviation of ductal size of non-surgical side.



**Fig. 2.** Box-and-whisker plot showing the size distribution of Stensen’s duct over time. Blue dot: mean sizes; diamond dot: mild outliers; square dot: extreme outliers. (A) Within the surgery alone group, the differences reached statistical significance ( $p = 0.015$ ), especially between 3 and 6 months ( $p = 0.012$ ) and between 3 and 24 months ( $p = 0.011$ ). (B) Within the surgery plus adjuvant CCRT group, there were no statistically significant differences over time ( $p = 0.831$ ). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

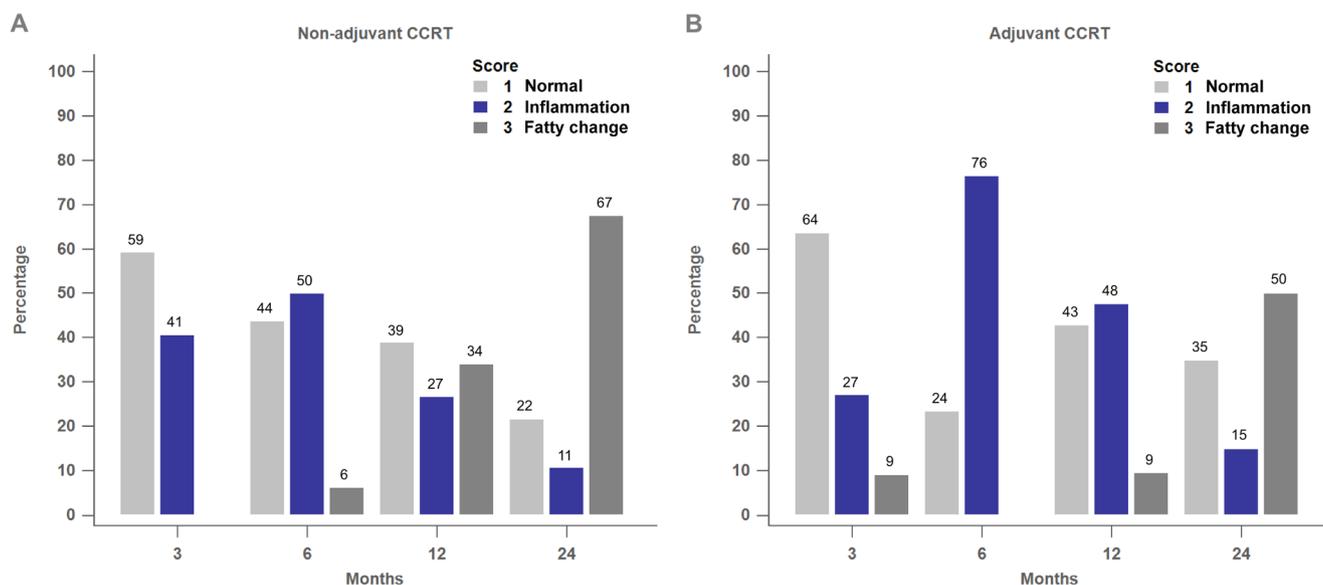


**Fig. 3.** The trend in parotid gland score over time. 1 = normal, 2 = inflammation, 3 = fatty change. (A) In the surgery alone groups, there was a significant increasing trend in score ( $p < 0.001$ ), corresponding to an evolution toward fatty change in the parotid gland. (B) In the surgery plus adjuvant concurrent chemoradiotherapy (CCRT) groups, the score did not increase significantly over time ( $p = 0.089$ ).

in Table 3, CCRT did not further increase ductal size, since none of the differences reached statistical significance. Thus, independent of CCRT, surgery and free flap reconstruction significantly increased the diameter of Stensen’s duct.

Fig. 2 shows the distribution of ductal size in box-and-whiskers plots. Independent of CCRT, the diameter of the Stensen’s duct was

largest at 3 months after surgery. In the plot of the data of the surgery alone group (Fig. 2A), the ductal differences between 3 and 6 months and between 3 and 24 months were significant. However, in the adjuvant CCRT group (Fig. 2B), none of the differences were significant. The reason why adjuvant CCRT reduced the discrepancies among the various time-points is unclear.



**Fig. 4.** Parotid gland scores (%) over time. (A) In the surgery alone groups, the postoperative inflammatory response was highest at 6 months, whereas fatty change was seen in the majority of patients at 24 months after surgery. (B) Similar results were obtained in the surgery plus adjuvant CCRT groups. An inflammatory response predominated at 6 months, and fatty change at 24 months, after surgery.

After BSCC resection with free flap reconstruction, injury of Stensen's duct results in ductal stricture, facial swelling, sialadenitis, sialocele, fistula, or buccal abscess [2,11]. Treatment consists of antibiotics, salivary gland puncture drainage or debridement. These complications and their subsequent management impede the wound healing and postpone the start of CCRT. Mehta et al. suggested that a relocation of Stensen's duct in BSCC resection would markedly lower the incidence of parotitis and sialocele during the early postoperative period [2], a finding supported by a study of parotid gland function by Longo et al. [11].

According to Mehta et al., complications such as those mentioned above occur in 75% of patients [2]. The incidence of inflammation in our patients, especially at 6 months postoperatively, was similar, as it reached 50% in the surgery alone group and 76% in the surgery plus adjuvant CCRT group (Fig. 4). However, our study demonstrated that with adequate postoperative care and regular clinical follow-up examination, the incidence of inflammation and sialocele declines between 12 and 24 months postoperatively, when the parotid gland becomes atrophic and undergoes fatty change (Fig. 3).

Atrophy of the parotid gland was, however, also seen in the surgery alone group. The reason is not known yet but it may be that the newly formed spontaneous drainage tunnels in the parotid gland after the surgery and free flap reconstruction do not function as well as the original Stensen's duct. Alternatively, either the vessels newly grafted to the free flap or the newly formed circulation of the buccal mucosa and parotid gland might have resulted in an inadequate blood supply of the parotid gland, and therefore atrophic change.

In our study, the size of the Stensen's duct on the affected side differed significantly from that of the non-affected side (Table 2), but there was no further impact of CCRT (Table 3). Thus, the observed changes mainly reflected the impact of surgery. In the surgery alone group (Fig. 3A), progressive development of fatty change was observed over 24 months ( $p < 0.001$ ). In the adjuvant CCRT group (Fig. 3B), regression of the parotid gland was delayed, but fatty change was eventually observed. These results demonstrated that there was no significant effect of CCRT on the dilation of Stensen's duct or the repair process of the parotid gland.

There were limitations in our study. Our study lacked a control group in which the Stensen's duct was ligated or marsupialized. In addition, there were no patients who received ductal re-anastomosis

during BSCC radical surgery. Retrospective studies are usually limited by its inadequate data collection. Moreover, T2 patients occupied the majority of this study population, which could be a selection bias but commonly met in retrospective study. However, temporarily dilatation of Stensen's duct after BSCC radical surgeries and spontaneous regression of the dilation and parotid gland atrophy were demonstrated in this study. These could be helpful for clinicians in decision making when following up BSCC patients after surgeries. A better designed case-control study in the future would conquer the inherited flaws and bias that existed in this retrospective study.

## Conclusion

BSCC resection and free flap reconstruction result in the dilation of Stensen's duct and an inflammatory response of the parotid gland, while postoperative adjuvant CCRT does not further impact these changes. Stensen's duct dilatation peaked at 3 months after surgery. The dilatation of Stensen's duct or the inflammation of parotid gland subsided gradually after 6 months of surgery, regardless of whether adjuvant CCRT was given. Without Stensen's ductal re-location, the parotid gland on the surgical side becomes atrophic and exhibits fatty change by 24 months postoperatively.

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None.

## Declaration of Competing Interest

All authors made no disclosure.

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