RESEARCH ARTICLE

Pathways involved in the spread of buccal carcinoma on contrast-enhanced multislice CT

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Objectives: To explore how buccal carcinoma spread, using contrast-enhanced multislice CT (CEMSCT).

Methods: We retrospectively analysed the extent of lesions in 56 patients with primary buccal squamous cell carcinoma (SCCA). Abnormal manifestations on CEMSCT at oral subsites and involved adjacent structures were documented and evaluated, which were compared with the results of surgery and histopathology.

Results: Infiltration and spread to oral subsites and/or adjacent structures was confirmed in 33 patients (58.9%). The opening of the Stensen duct was the most commonly invaded oral subsite (72.7%); other sites included the gingivobuccal sulcus (60.6%), pterygomandibular raphe (54.5%), gingiva (24.2%), retromolar trigone (24.2%), orbicularis oris (18.2%) and the floor of mouth (15.2%). Of the involved adjacent structures, the buccal space was the most common site of spread (69.7%), followed by the masticatory muscles and spaces (57.6%), bone (54.5%), skin and subcutaneous fat (39.4%), pharynx (30.3%), investing fascia (15.2%) and the base of the skull (6.1%). CEMSCT manifestations of the involvement in buccal SCCAs had correlations with pathological findings ($p < 0.05$). The sensitivities, specificities and accuracies of two radiologists' evaluation on buccal carcinoma involvement were 50.00%, 23.21% and 73.21%; and 51.79%, 32.14% and 83.93%, respectively.

Conclusions: Buccal SCCAs could superficially and deeply spread to multiple oral subsites and/or adjacent structures. CEMSCT could delineate their spread pathways and extents.

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Introduction

Oral cavity cancers accounted for approximately half of all head and neck squamous cell carcinomas (SCCAs).¹ The buccal mucosa was a subsite within the oral cavity mucosa. Buccal SCCA was prevalent in Southeast Asia and India because of tobacco use and the chewing of betel nuts. As buccal carcinoma frequently demonstrated aggressive behaviour, and the buccal mucosa had few

anatomical barriers, buccal SCCA could easily extend to neighbouring intraoral subsites and adjacent structures,²⁻⁴ which affected its T-staging, surgical resectability and radiation planning.

Although mucosal spread of buccal SCCA was easily assessed by clinical physical examination, its deep extent of spreading along muscles, invasion of mandible or maxilla and involvement of neurovascular bundles, all of which were indicative of an advanced stage of the disease, were not directly visualized and not easily detected on clinical examination. CT was usually the first imaging

modality used to assess and stage SCCA of the oral cavity and oropharynx. Contrast-enhanced multislice CT (CEMSCT) might detect the abnormalities of deep spread of buccal SCCA. Therefore, CEMSCT could play a critical role in the understanding spread patterns of buccal SCCA, which is helpful for its staging and treatment options.

A few studies had described the spread pathways of buccal carcinoma as a subdivision of oral cavity cancers.^{1,5,6} To the best of our knowledge, no extensive studies had been performed to assess the extent of buccal carcinoma. On the other hand, buccal carcinoma could also involve multiple sites, which requires further discussion. The purpose of this study was to investigate and report the spread of buccal carcinoma to additional sites of involvement that were apparent using modern CEMSCT.

Methods and Materials

56 patients with buccal carcinoma were admitted to The First Affiliated Hospital of Chongqing Medical University, Chongqing, China, between August 2011 and December 2013. The study was approved by the ethics committee of the university (reference number 2011007), and all patients had provided informed consent. 34 males and 22 females were enrolled (mean age, 66.8 years; median age, 66 years; range, 41–88 years), and 39 patients (69.6%) were aged 60 years or older. Clinical manifestations included an ill-defined, mainly unmovable buccal mass, with or without ulcer, base infiltration, pain and easy haemorrhage. All cases were classified as primary buccal lesions, and no patients had a history of radiation treatment. Histopathological results were obtained for every patient via biopsy or surgery.

CT technique

56 patients were examined using multislice spiral CT (MSCT) (GE 64-slice Lightspeed[®] Volume CT; GE Medical Systems, Milwaukee, WI). Patients were examined from the base of the skull to the thoracic inlet using 5-mm-thick contiguous sections, 0.984 pitch and 512 × 512-pixel matrix. All patients were scanned after administration of a contrast material (370 mg iodine per millilitre of iopromide; Bayer Schering Pharma AG, Berlin, Germany) via intravenous injection (0.2 ml per kilogram of body weight). CEMSCT images were reconstructed into 0.625-mm-thick slices and processed at a GE ADW 4.4 workstation in order to create multiplanar reconstructions.

CEMSCT images of each patient were retrospectively evaluated for the involvement of buccal carcinoma. Abnormal signs on CEMSCT indicative of buccal carcinoma involving structures included space-occupying growth, obliteration of fat planes, infiltration of the mucosa, muscle, skin or subcutaneous fat, bone destruction and inhomogeneities on contrast imaging. These findings were interpreted by two head and neck diagnostic

radiologists (A and B) who were blind to surgical and histopathological results. Final results were reached by consensus.

Statistical analyses were performed by SPSS[®] software v. 17.0 (SPSS Inc., Chicago, IL) or Statistics Analysis System 9.2. Categorical variables were summarized as frequencies and percentages. For the analysis of interobserver variability, kappa statistics were used (Cohen's and Fleiss kappa coefficient). The comparison of two observers was performed with the McNemar's χ^2 test with continuity correction. The contingency coefficient was used to evaluate correlation. All statistical tests were two sided with a significance level of $p < 0.05$.

Results

In this study, all the histopathological results were SCCAs. 33 patients (58.9%, 33/56) were confirmed to have buccal carcinoma involvement by surgery and histopathology. Tables 1 and 2 show the consistency of the results about primary buccal SCCAs and their involvement evaluated between two radiologists. The difference in each group between them had no statistical significance ($p > 0.05$).

The correlation of the involvement of primary buccal SCCAs on CEMSCT with the involvement in the pathological diagnosis was calculated (Tables 3 and 4). The involvement of primary buccal SCCAs on CEMSCT evaluated by two radiologists had correlations with the involvement in the pathological diagnosis ($p < 0.05$).

Oral subsites involved

The opening of the Stensen duct was involved in 24 cases (72.7%, 24/33), and among those, Stensen duct was involved in 10 cases (30.3%, 10/33). Gingivobuccal sulcus was involved in 20 cases (60.6%, 20/33), and those could be divided into upper gingivobuccal sulcus involved in 7 cases, lower gingivobuccal sulcus involved in 8 cases, upper and lower gingivobuccal sulcus both involved in 5 cases and hyomandibular furrow involved in 1 case. Pterygomandibular raphe (PMR) was involved in 18 cases (54.5%, 18/33). Gingiva was involved in eight cases (24.2%, 8/33), which could be divided into the lower gingiva involved in six cases, three of the six cases also involved lingual gingiva, and the upper and lower gingiva both involved in two cases. Retromolar trigone (RMT) was

Table 1 Comparison of primary buccal squamous cell carcinomas on contrast-enhanced multislice CT evaluated between two radiologists

A	B		Total	χ^2	p-value
	(+)	(-)			
(+)	48	1	49	0.3333	0.5637
(-)	2	5	7		
Total	50	6	56		

(+), positive finding; (-), negative finding.

McNemar test: $p = 0.5637 > 0.05$; kappa coefficient, 0.7393; standard error, 0.1432; 95% confidence interval, 0.4585–1.0000. A and B represented two radiologists, respectively.

Table 2 Comparison of the involvement of primary buccal squamous cell carcinomas on contrast-enhanced multislice CT evaluated between two radiologists

A	B		Total	χ^2	p-value
	(+)	(-)			
(+)	30	8	38	1.3333	0.2482
(-)	4	14	18		
Total	34	22	56		

(+), positive finding; (-), negative finding.

McNemar test, $p = 0.2482 > 0.05$; kappa coefficient, 0.5359; standard error, 0.1162; 95% confidence interval, 0.3082, 0.7637. A and B represented two radiologists, respectively.

involved in eight cases (24.2%, 8/33). Orbicularis oris was involved in six cases (18.2%, 6/33) and the floor of mouth was involved in five cases (15.2%, 5/33).

Adjacent structures involved

Buccal space was involved in 23 cases (69.7%, 23/33). The masticatory muscles and spaces were involved in 19 cases (57.6%, 19/33), which could be divided into the tendon of the temporal muscle involved in 8 cases, temporal muscle involved in 14 cases, medial pterygoid muscle involved in 8 cases, medial and lateral pterygoid muscles both involved in 4 cases, pterygomandibular space involved in 6 cases, masseteric space involved in 5 cases, infratemporal space involved in 6 cases and pterygopalatine fossa involved in 2 cases.

In 18 cases (54.5%, 18/33), the bone was involved, which could be divided into the upper alveolar ridge involved in 7 cases, of which maxillary tuberosity was involved in 3 cases, maxillary sinus was involved in 4 cases and mandibular ramus was involved in 7 cases; in 5 of the 7 cases, the anteromedial aspect of the mandibular ramus was involved; and in 2 of the 7 cases, the mandibular body was involved.

The skin and subcutaneous fat were involved in 13 cases (39.4%, 13/33). The pharynx was involved in 10 cases (30.3%, 10/33), which could be divided into the oropharynx wall involved in 10 cases, nasopharynx involved in 2 cases, soft palate involved in 9 cases, tonsil involved in 5 cases, palatoglossal arch involved in 5 cases and tongue base involved in 2 cases.

In this study, we calculated the sensitivity, specificity and accuracy of the evaluation of two radiologists on primary buccal SCCAs and their involvement (Table 5). Sensitivity and accuracy of primary buccal SCCAs on CEMSCT were high (87.50% and 89.29%, respectively). For the involvement of primary buccal SCCAs, sensitivity

and specificity on CEMSCT were relatively low, but the accuracy was high (73.21% and 89.93%, respectively).

Discussion

Buccal carcinoma is an oral cavity cancer, and its incidence significantly differed between regions. Buccal carcinoma was also common and accounted for approximately 30% of oral cavity cancers in China.⁴ All cases in this study were confirmed/identified as SCCAs of buccal mucosa. Primary buccal SCCAs and their involvement could be evaluated on CEMSCT between two radiologists, in which the differences between them had no statistical significance. The involvement of primary buccal SCCAs on CEMSCT had correlations with the pathological diagnosis, therefore, CEMSCT could be used for evaluating primary buccal carcinomas and their involvement.

Most primary buccal SCCAs were confined to the mucosal layer during the early stages. As disease evolved, carcinoma infiltrated the underlying submucosa and muscle and extended submucosally and posteriorly along the buccinator muscle to the PMR, which was considered the most common spread pattern,^{5–7} and anteriorly to the orbicularis oris and lip (Figure 1). Following infiltrative growth, buccal SCCA might extend to the subcutaneous fat tissue and dermis in the cheek, including the investing fascia, which presented as linear reticulations in the subcutaneous fat, skin thickening and sagging on CEMSCT (Figure 1). Spector et al⁸ reported that peritumoural inflammation—rather than tumour invasion—mostly developed in head and neck squamous cell carcinoma. It was important to determine the extent of dermal involvement, as dermal infiltration was often a characteristic of T4a stage disease. We also found that sagging skin owing to tumour infiltration could be used to differentiate inflammation on CEMSCT. Buccal SCCAs could infiltrate all layers of the cheek if they spread laterally. The buccal mucosa was continuous, with the mucosa covering the superior and inferior gingivobuccal sulcus, the maxillary and mandibular alveolar ridges and the RMT. Superficial buccal mucosa carcinoma might spread superiorly and inferiorly, thereby infiltrating the gingivobuccal sulcus, buccal gingiva and the RMT, and these three parts were called the gingivobuccal complex.^{9,10} Buccal SCCA could also spread medially, cross the alveolar ridges and involve

Table 3 Correlation of the involvement of primary buccal squamous cell carcinoma on contrast-enhanced multislice CT (CEMSCT) evaluated by radiologist A with the pathological diagnosis

Involvement on CEMSCT	Involvement in pathological diagnosis			Contingency coefficient	p-value
	(+)	(-)	Total		
(+)	28	10	38	0.3995	0.0011
(-)	5	13	18		
Total	33	23	56		

(+), positive finding; (-), negative finding.

Contingency coefficient, 0.3995; $p = 0.0011 < 0.05$.

Table 4 Correlation of the involvement of primary buccal squamous cell carcinomas on contrast-enhanced multislice CT (CEMSCT) evaluated by radiologist B with the pathological diagnosis

Involvement on CEMSCT	Involvement in pathological diagnosis			Contingency coefficient	p-value
	(+)	(-)	Total		
(+)	29	5	34	0.5545	<0.0001
(-)	4	18	22		
Total	33	23	56		

(+), positive finding; (-), negative finding.

Contingency coefficient, 0.3995; $p < 0.0001$.

the lingual gingiva (Figure 2) and hyomandibular furrows. Involvement in the superficial gingivobuccal sulcus and gingiva could be directly screened in clinical practice, and the extent of mucosal spread could be estimated on physical examination; however, submucosal spread could not be evaluated so easily. In this study, the involvement in superficial gingivobuccal sulcus and gingiva were not exhibited on axial CEMSCT in four patients. Superficial mucosal and submucosa lesions were rarely radiographically evident, although they could be imaged on puffed-cheek CT,^{1,11} especially on coronal reformatted CEMSCT for showing gingivobuccal sulcus and gingiva. The extent of carcinoma involvement in the RMT could not be clinically determined easily.

The RMT was a small triangular area posterior to the mandibular and maxillary last molar and directly contiguous with the anterior pillar of the tonsil, soft palate and the floor of the mouth. The mucosa of RMT overlay the attached muscles of mastication and the PMR. The RMT could be shown in its entirety on reformatted oblique MSCT.¹² Owing to its unique anatomy, tumours in the RMT might demonstrate numerous complex routes of spread, which have been described in the literature.^{1,6,13–17} The RMT tumours might be primary or result from regional extension. In this study, nine patients with the RMT involvement via posterior extension of buccal carcinoma were identified (Figure 3b), and the PMR was also involved in these cases because the RMT carcinomas extended submucosally. Involvement of the floor of the mouth might have been owing to the cancer spreading inferiorly (Figure 3b,c). The pterygomandibular and masseteric spaces were involved owing to the spread of cancer along the medial pterygoid muscles (Figure 3b). Cancers could also extend posteriorly to the tonsils or medially to the adjacent soft palates.

The openings of the Stensen's ducts were invaded in most patients included in this study. Because Stensen's

duct crossed the masseter muscle, transversely coursed through the buccal fat pad and pierced the buccinator muscle, its opening was located at the buccal mucosa opposite the second maxillary molar where buccal carcinoma often occurred; therefore, the tumour could easily invade this opening and infiltrate along the duct. Owing to the increase in soft-tissue obstruction at the opening, the duct demonstrated irregular thickening and heterogeneous enhancement on CEMSCT (Figure 1a). The parotid gland swelling was also evident (Figure 1a). Four cases of the Stensen's duct opening involvement showed no abnormality on CEMSCT because of tumour slight infiltration and lack of increased soft tissue. Although the involvement in the opening of Stensen's duct was easy to examine in clinical settings, imaging could also show deep involvement in the duct, which was helpful when planning surgical excision. Few cases have been previously reported.

The buccal space was the second most involved structure. The buccal space was located outside the buccinator and its fascia, which filled the process of buccal fat pad. When a large-volume mass penetrated the buccinator, it could enter and involve the buccal space (Figures 1a and 3b). Because Stensen's duct coursed through the buccal fat pad, the main duct might become involved following invasion into the buccal space. This was another way the carcinomas could spread to the Stensen's main duct.

Buccal carcinoma involving the masticators and masseteric spaces was another common pathway. The masseter muscle, mandible and lateral and medial pterygoid muscles outlined the posterior margin of the buccal space, which was incomplete and communicated with the masticator space. Eight patients with involvement in the tendon of the temporal muscle were identified in this study. Because the temporal muscle tendon attached to the anterior margin of the mandibular ascending ramus, small-volume cancer could often

Table 5 Sensitivity, specificity and accuracy of two radiologist's evaluations on primary buccal squamous cell carcinomas (SCCAs) and their involvement ($n = 56$)

	True positive	True negative	False positive	False negative	Sensitivity (%)	Specificity (%)	Accuracy (%)
Radiologist A							
Primary buccal SCCAs	49	0	0	7	87.50	–	87.50
Buccal SCCA involvement	28	13	10	5	50.00	23.21	73.21
Radiologist B							
Primary buccal SCCAs	50	0	0	6	89.29	–	89.29
Buccal SCCA involvement	29	18	5	4	51.79	32.14	83.93

Figure 1 A 41-year-old male with left buccal squamous cell carcinoma. A mass (lightning) with a penetrating ulcer is shown spreading anteriorly along the buccinator to involve the upper (a) and lower (b) lips (large arrow) on axial contrast-enhanced multislice CT. The buccal space has also been invaded (a) (triangle). The carcinoma extends outwards to infiltrate all layers of the cheek (a) (small arrow); linear reticulations in the subcutaneous fat and skin thickening and sagging are also shown (b) (small arrow). The involved Stensen's duct is irregularly thickened and enhanced (a) (curved arrow). Enhanced left parotid swelling is also evident (a) (arrow head).

extend first into the buccal space to invade this tendon and then infiltrate upwards to invade the temporal muscle. When a large-volume cancer extended into the posterior portion of the buccal space, it might invade the masseter, masseteric space, mandibular ramus, pterygoid muscles, pterygomandibular space and infratemporal space (Figure 3b).¹⁸ When the carcinoma extended into the infratemporal fossa, it might medially enter the pterygopalatine fossa. Buccal carcinoma could also spread to the pterygopalatine fossa via the involved pterygopalatine process of the buccal fat pad (Figure 3a).

The PMR was involved in 18 patients (Figure 3b), this is a band of connective tissue situated beneath the mucosal surface of the RMT. It also serves as a common insertion point for the buccinator, orbicularis oris muscle and superior pharyngeal constrictor muscle.

Figure 2 A 75-year-old edentulous male patient with left buccal squamous cell carcinoma. Reformatted coronal contrast-enhanced multislice CT shows a left buccal mass that involves the lower gingivobuccal sulcus and is spreading across the alveolar ridge to involve the lingular gingiva (arrows).

Thus, buccal cancers could grow laterally and sub-mucosally along the buccinator muscle to the PMR. The PMR might also involve the RMT in the gingivobuccal complex and extend from the buccal carcinoma. The PMR attached superiorly to the hamulus of the medial pterygoid plate and inferiorly to the mylohyoid line of the mandible, thereby forming a junction between the oral cavity, oropharynx and nasopharynx. In this study, two patients developed involvement of the nasopharynx owing to the superior growth along the PMR. The oropharynx and tonsils were involved because the buccal carcinoma grew posteriorly along the PMR and the superior pharyngeal constrictor muscle (Figure 3b). From there, carcinomas could invade the anterior tonsillar pillars and extend along the palatoglossus muscles, thus involving the palatoglossus arches. The soft palate and the base of the tongue could also become involved as lesions in the anterior tonsillar pillars extended superiorly to the soft palates and inferiorly to the base of the tongue. Inferior growth along the PMR resulted in the invasion of the floor of the mouth (Figure 3b,c). Another spread pattern was large-volume buccal carcinoma that destroyed the mandible and directly entered the floor of the mouth. One patient had an internal carotid artery that was encased by the subsequent involvement of the base of the skull base (Figure 4), which indicated that this patient's buccal carcinoma was stage T4b and could not be surgically resected.

18 patients were diagnosed with bone involvement, and of these, the superior alveolar ridge was also invaded in 7 patients, maxillary tuberosity in 3 patients and maxillary sinus in 4 patients. Because the buccinator muscle originated from the alveolar processes of the maxilla and mandible, buccal carcinoma could erode the underlying alveolar ridges of maxilla and mandible, which was the most common spread pattern for buccal carcinoma.⁵⁻⁷ Involvement of the posterior aspect of the maxilla might allow superior spread into the proximal maxillary sinus. Seven cases that involved the ramus of mandible were erosive; of these, five cases involved local invasion in the anterior aspect of the ascending ramus

Figure 3 A 65-year-old male with left buccal squamous cell carcinoma that spread via multiple pathways. On axial contrast-enhanced multislice CT, the left buccal mass is shown invading the pterygopalatine process of the buccal fat pad (a) (small arrow) and the buccal space (b) (curved arrow). The mass (b) (lightning) also grows along pterygomandibular raphe (small black arrows) and involves nasopharynx, oropharynx and tonsil (a, b) (large arrow). The retromolar trigone is also invaded (b) (triangle). The carcinoma involves the masticator and space posteriorly (b) (arrows with circles) and grows inferiorly to involve the floor of the mouth (b, c) (small white arrows). The anterior aspect of the ascending ramus of the mandible is erosive (d) (arrow).

(Figure 3d). Two cases invaded both the ramus and the body of the mandible. The anterior aspect of the ascending ramus of the mandible was the posterior margin

Figure 4 A 78-year-old male patient with left buccal squamous cell carcinoma. On axial contrast-enhanced multislice CT, left buccal carcinoma (lightning) extends posteriorly along pterygomandibular raphe (small arrows) and involves the base of skull (large arrow) and parapharyngeal space, which cannot be delineated from the pharynx. The left internal carotid artery is encased (triangle).

of the buccal space, which buccal carcinoma could easily extend into and erode,¹⁸ because the RMT mucosa overlay the ascending ramus of the mandible and maxillary tuberosity. On the other hand, the periosteum of the mandible was close, and carcinoma in the RMT usually eroded the mandible at the early stage,^{13,15} which contrasted with the opinion that bone involvement was less common owing to both separation and the presence of the PMR.¹⁹ A 64-slice MSCT scanner provided 0.625-mm-thick slices for isotropic coronal, sagittal and oblique reformations with bone algorithms, which was advantageous for assessing minor cortical erosion. Advanced-stage large-volume buccal carcinoma might significantly destroy the ramus and/or the body of the mandible. Osseous involvement might directly result from cancer extension from the primary site. Perineural or intramedullary spread to the mandible was not observed in this study.^{1,7,13} Bone involvement was as important as skin involvement to buccal carcinoma in terms of T4a staging.

As for CEMSCT diagnosis in primary buccal SCCA, it had a high sensitivity and accuracy, which might be related to sample selection; all cases selected in this study were confirmed SCCAs, and most were infiltrative masses with heterogeneous enhancement, which were obvious on CEMSCT for easy diagnosis. There were several false-negative cases, which related to superficial mucosa or submucosa lesions that were not shown on routine CEMSCT. For the involvement of primary

buccal SCCAs, sensitivity and specificity on CEMSCT were relatively low, but the accuracy was high. Signs and manifestations of the involvement on CEMSCT were heterogeneous, and some of them were not easy to differentiate from normal and other abnormalities such as inflammation, so there existed some false-positive and false-negative cases. Other imaging modalities like high field intensity MRI would be needed to investigate for increasing the sensitivity and specificity.

There were some limitations in this study. Firstly, only two observers were used to evaluate the images, which existed subjective nature to some extent. Secondly, MRI provided superior soft-tissue contrast and better depicted the detailed anatomy of the oral cavity,

therefore, could more accurately delineate the extent of oral cavity carcinoma. Unfortunately, a minority of patients examined by MR in our research protocol were not included in this study. Future studies should compare the advantage between MR and MSCT for delineating the extent of buccal SCCA.

In conclusion, buccal SCCAs might superficially infiltrate the oral subsites and also spread to the perimucosa and deeply involve adjacent structures. Here, we did not evaluate the lymphatic dissemination pathways. CEMSCT provided valuable information for evaluating the extents of buccal SCCAs. Radiologists must be familiar with the locoregional and oral cavity anatomy to help understand the spread pathways of buccal SCCAs.

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