RESEARCH ARTICLE

Spontaneously infarcted parotid tumours: MRI findings

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Objectives: To report MRI findings of spontaneous infarction in parotid tumours.

Methods: 14 patients (13 male, 1 female; mean age 73 years) with spontaneously infarcted parotid tumours were reviewed retrospectively. MR images were assessed for the location, the presence of synchronous parotid masses, margin characteristics, signal intensity on T_1 and T_2 weighted images, and internal architecture according to the distribution of T_2 signal hyperintensity.

Results: 12 tumours were located in the parotid tail and 2 in the superficial lobe. Synchronous parotid masses were seen in four tumours, three of which were located in the ipsilateral parotid tail and one in the contralateral parotid tail. Seven tumours had well-defined margins and seven had ill-defined margins. The signal intensities on T_1 weighted images were a mixture of high and intermediate in all cases; in 11 tumours, hyperintense areas were dominant. On T_2 weighted images, all tumours also showed a mixture of high and intermediate signal intensities. Internal architectures on T_2 weighted images were mosaic hyperintensity in three tumours, central hyperintensity in five, and multiseptated hyperintensity in six.

Conclusions: Spontaneously infarcted parotid tumours were mostly located in the parotid tail and showed mixed signal intensities with predominant hyperintensity on T_1 weighted images. Half of the tumours had ill-defined margins, and the internal architectures varied.

Dentomaxillofacial Radiology (2019) **48**, 20180382. doi: [10.1259/dmfr.20180382](https://doi.org/10.1259/dmfr.20180382)

Cite this article as: Kashiwagi N, Hyodo T, Ishi K, Maenishi O, Enoki E, Chikugo T, et al. Spontaneously infarcted parotid tumours: MRI findings. *Dentomaxillofac Radiol* 2019; **48**: 20180382.

Keywords: parotid neoplasms; adenolymphoma; infarction; magnetic resonance imaging

Introduction

Infarction of salivary gland tissue can occur either with or without neoplasia. The latter is recognized as necrotizing sialometaplasia, which is a self-limiting inflammatory disorder with a preferential site of the minor salivary gland on the hard plate.¹ Infarction with neoplasia usually occurs in benign parotid tumours with most histological types being Warthin's tumour, followed by pleomorphic adenomas and oncocytomas.²⁻¹⁵ The main cause of infarction occurring in parotid tumours is fine-needle aspiration (FNA), and its

typical and characteristic course is of acute parotid pain or swelling, which occur within a few weeks post-FNA, followed by regression in size, and occasionally the tumours show almost complete involution.²⁻⁵ Less frequently, parotid tumours can also infarct spontaneously,⁶⁻¹⁵ and in their cytological diagnosis, secondary changes to the infarction such as squamous metaplasia with extensive necrosis have occasionally led to the erroneous diagnosis of a parotid carcinoma.^{3,4,7,14,15} Therefore, in patients without a history of FNA procedures, MRI is expected to play an important role in the diagnosis of infarcted parotid tumours. However, to our

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Received 16 October 2018; revised 07 January 2019; accepted 17 January 2019

Figure 1 Schema of internal architecture classification on T_2 weighted images. (a) Mosaic hyperintensity was defined as a lesion with a diffuse mixture of high and intermediate signal intensities. (b) Central hyperintensity was defined as a ring-shaped lesion with central hyperintensity and peripheral intermediate intensity. (c) Multiseparated hyperintensity was defined as an overall hyperintense lesion separated by multiple intermediate-signal septa.

knowledge, MRI findings have only been reported for a single case.⁹ Here, we report MRI findings in 14 patients with spontaneously infarcted parotid tumours and add a brief description of their clinical features.

Methods and Materials

Patients

Local regional ethics committee approval was obtained for this retrospective analysis. 14 consecutive patients (13 male, 1 female; mean age, 73 years; range, 60–90 years) with spontaneous infarctions in parotid tumours documented between January 2009 and June 2018 were identified in our Kindai University hospital's radiologic database and electronic medical record system. All patients presented with acute enlargement or pain of a pre-existing parotid mass and had no clinical and laboratory evidence of infection. In three of the patients, surgical resection was performed resulting in the histological diagnosis of a Warthin's tumour with extensive necrosis, and in one patient, cytological diagnosis of the FNA 2 years prior to the onset of acute symptoms had revealed a Warthin's tumour. In the 10 patients who were observed and treated conservatively, follow-up imaging studies confirmed spontaneous regression of the tumour.

MRI examination

The duration from onset of symptoms to MRI examination ranged from 3 to 39 days. MRI examinations were performed using a 1.5 Tesla magnet in 12 patients and a

3.0 Tesla magnet in 2. On pre-contrast axial planes, T_1 weighted images [repetition time (TR)/echo time (TE), 466–680/14–20 ms] and T_2 weighted images (TR/TE, 2500–5200/80–120 ms) were obtained for all patients. Short inversion time inversion recovery (STIR) images (TR/TE, 3400–500/30–36 ms, inversion time 150 ms) were obtained for 13, and diffusion-weighted (DW) images (b factors, 0 and 1000 s mm⁻²) were obtained for 13. Apparent diffusion coefficient (ADC) maps were constructed automatically from DW images. On coronal planes, STIR images were obtained for 13 patients and T_1 weighted images were obtained for 2. In two patients, axial and coronal post-contrast T_1 weighted images were obtained with or without a fat-suppression technique. The section thickness was 4–5 mm and the pixel size ranged from 0.4 × 0.5 mm to 0.9 × 1.0 mm.

Imaging interpretation

Two experienced radiologists (TH and NK, with 18 and 25 years of clinical experience in diagnostic radiology, respectively) independently reviewed all MRI for location of the infarcted tumour in the parotid gland, the presence of synchronous parotid masses, size, margin characteristics, signal intensity on T_1 weighted, T_2 weighted, and STIR images, the internal architecture according to the distribution of hyperintense area on T_2 weighted images, ADC values on DW images, and enhancement patterns.

In cases with multiple or bilateral parotid tumours, we evaluated the largest lesion on the symptomatic side. The location in the parotid gland was categorized into

three areas: superficial lobe, deep lobe, and parotid tail. The superficial lobe was defined as an area lateral to the retromandibular vein, the deep lobe was defined as an area medial to the retromandibular vein, and the tail was defined as the inferior 2.0 cm area of the gland. Size was measured as the maximum diameter of the lesion. Margin characteristics were classified as well- or ill-defined. The signal intensity on T_1 weighted images was defined as follows: low if the signal intensity was defined as less than or equal to that of cerebrospinal fluid; intermediate if the signal intensity was greater than that of cerebrospinal fluid but less than or equal to that of muscle; and high if the signal intensity was greater than that of muscle. When the tumour showed mixed signal intensities, the dominant signal intensity was also recorded. The signal intensity on T_2 weighted images was defined as follows: low if signal intensity was defined as less than that of muscle; intermediate if the signal intensity was greater than or equal to that of muscle but less than that of fat; and high if the signal intensity was greater than or equal to that of fat. The signal intensity on STIR images was defined as follows: low if the signal intensity was defined as less than that of muscle; intermediate if the signal intensity was greater than or equal to that of muscle but less than that of cerebrospinal fluid; and high if the signal intensity was

greater than or equal to that of cerebrospinal fluid. According to the distribution of hyperintense areas on T_2 weighted images, the internal architecture was classified as mosaic hyperintensity, central hyperintensity, or multiseptated hyperintensity (Figure 1). Mosaic hyperintensity was defined as a lesion with a diffuse mixture of high and intermediate signal intensities, central hyperintensity was defined as a ring-shaped lesion with central hyperintensity with peripheral intermediate signal intensity, and multiseptated hyperintensity was defined as an overall hyperintense lesion separated by multiple intermediate-signal septa. For ADC measurements in tumours showing diffuse internal architecture (mosaic or multiseptated hyperintensity on T_2 weighted images), regions of interest were placed on the central area of the tumour on the ADC maps. For the measurements in tumours showing ring-shaped internal architecture (central hyperintensity on T_2 weighted images), regions of interest were placed on peripheral area of the tumour on the ADC maps by referring to morphological information on T_2 weighted images. Enhancement pattern was assessed subjectively as none, ring-shaped, homogeneous, or inhomogeneous.

Any discrepancies between the two readers were resolved by consensus for qualitative evaluations, and measurements of lesion size and ADC were averaged.

Table 1 Summary of clinical and morphologic features

Case no	Age/sex	Symptom	Cytologic feature of post-onset FNA	Size (mm)	Location	Synchronous parotid mass	Margin
1	74/M	Acute painful enlargement of PG	Inflammatory cells	38	Tail	None	Ill-defined
2	68/M	Acute painful enlargement of PG	Inflammatory cells	39	Superficial lobe	None	Well-defined
3	69/M	Acute painless enlargement of PG	Inflammatory cells	31	Tail	None	Well-defined
4	72/M	Acute painful enlargement of PG	N/A	45	Tail	Present	Ill-defined
5	60/M	Acute painful enlargement of PG	Necrotic debris, Inflammatory cells	64	Tail	None	Ill-defined
6	88/M	Acute painful enlargement of PG, facial paralysis	Necrotic debris, Inflammatory cells, Squamous cells	49	Superficial lobe	present	Well-defined
7	65/M	Acute painful enlargement of PG	Necrotic debris, Inflammatory cells	40	Tail	None	Well-defined
8	90/F	Acute painless enlargement of PG	N/A	27	Tail	None	Well-defined
9	61/M	Acute painful enlargement of PG	N/A	20	Tail	None	Well-defined
10	70/M	Acute painless enlargement of PG	Inflammatory cells, Columnar cells,	39	Tail	None	Ill-defined
11	63/M	Acute painful enlargement of PG	Necrotic debris,	49	Tail	Present	Ill-defined
12	69/M	Acute painful enlargement of PG	Inflammatory cells, Squamous cells	32	Tail	None	Ill-defined
13	81/M	Acute pain of PG	Inflammatory cells	24	Tail	Present	Ill-defined
14	71/M	Acute painful enlargement of PG	N/A	33	Tail	None	Well-defined

M = male; F = female; PG = parotid gland; FNA, fine-needle aspiration.

Table 2 Summary of SI on MRI

Case no	SI on T_1 WI	Predominant SI on T_1 WI	SI on T_2 WI	SI on STIR	Internal architecture on T_2 WI	Contrast enhancement	ADC value (10^{-3} mm ² /s)
1	Mixture of high and intermediate	Intermediate	Mixture of high and intermediate	Mixture of high and intermediate	Mosaic hyperintensity	N/A	2.0
2	Mixture of high and intermediate	High	Mixture of high and intermediate	Mixture of high and intermediate	Mosaic hyperintensity	Ring enhancement	2.0
3	Mixture of high and intermediate	High	Mixture of high and intermediate	Mixture of high and intermediate	Mosaic hyperintensity	N/A	N/A
4	Mixture of high and intermediate	Intermediate	Mixture of high and intermediate	Mixture of high and intermediate	Central hyperintensity	N/A	0.9
5	Mixture of high and intermediate	Intermediate	Mixture of high and intermediate	Mixture of high to low	Central hyperintensity	N/A	1.1
6	Mixture of high and intermediate	High	Mixture of high and intermediate	Mixture of high and intermediate	Central hyperintensity	N/A	1.0
7	Mixture of high and intermediate	High	Mixture of high and intermediate	Mixture of high to low	Central hyperintensity	N/A	1.7
8	Mixture of high and intermediate	High	Mixture of high and intermediate	Mixture of high and intermediate	Central hyperintensity	N/A	1.2
9	Mixture of high and intermediate	High	Mixture of high and intermediate	Mixture of high and intermediate	Multiseparated hyperintensity	N/A	1.2
10	Mixture of high and intermediate	High	Mixture of high and intermediate	N/A	Multi separated hyperintensity	Ring enhancement	1.8
11	Mixture of high and intermediate	High	Mixture of high and intermediate	Mixture of high and intermediate	Multiseparated hyperintensity	N/A	1.7
12	Mixture of high and intermediate	High	Mixture of high and intermediate	Mixture of high and intermediate	Multiseparated hyperintensity	N/A	1.8
13	Mixture of high and intermediate	High	Mixture of high and intermediate	Mixture of high and intermediate	Multiseparated hyperintensity	N/A	1.3
14	Mixture of high and intermediate	High	Mixture of high and intermediate	Mixture of high to low	Multiseparated hyperintensity	N/A	1.4

N/A, not available; SI, signal intensity; T_1 WI, T_1 weighted images; T_2 WI, T_2 weighted images.

The level of interobserver agreement was analyzed using prevalence-adjusted bias-adjusted κ values, which were interpreted using Landis and Koch's categorization: 0–0.20, slight agreement; 0.21–0.40, fair agreement; 0.41–0.60, moderate agreement; 0.61–0.80, substantial agreement; and 0.81–1.00, almost perfect agreement.¹⁶

Results

Clinical presentation

Table 1 summarizes the clinical features. All 14 patients were aware of pre-existing parotid masses, 10 of which presented with acute painful swelling, 3 with acute painless swelling, and 1 with acute pain. Facial palsy was noted in one patient. FNA had been performed 2 years prior to the onset of symptoms in one patient, resulting in a diagnosis of Warthin's tumour. After onset, FNA was performed in 10 cases, but the results did not provide any specific histological information for the type of parotid tumour because the biopsy specimens usually consisted of necrotic debris and inflammatory cells such as foam cells, neutrophils, or lymphocytes. In three patients, bacterial cultures of aspirated material were performed, but these did not yield any organisms.

MRI findings

Tables 1 and 2 summarize the MRI findings and representative images are shown in Figures 2–4. According to the qualitative evaluations, the interobserver agreements were found to be substantial to almost perfect as follows. The prevalence-adjusted bias-adjusted κ values were 0.68 for the location, 1.00 for the presence of a synchronous mass, 0.68 for margin characteristics, 0.78 for the signal intensity on T_1 weighted images, 0.78 for that on T_2 weighted images, 0.89 for that on STIR, and 0.78 for the internal architecture.

12 of the tumours were located in the parotid tail and two in the superficial lobe. Four tumours had synchronous parotid masses, three of which were located in the ipsilateral parotid tail and the other in the contralateral parotid tail. The mean maximal cross-sectional diameter of evaluated lesions was 3.8 cm (range, 2.0–6.4 cm). Seven tumours had well-defined margins and seven had ill-defined margins.

The signal intensities on T_1 weighted images were a mixture of high and intermediate in all cases; in 11 cases, the hyperintense areas were dominant. On T_2 weighted images, the signal intensities were a mixture of high and intermediate in all cases. On STIR images, they

Figure 2 Case 2. A 68-year-old male presented with an acute painful enlargement of a parotid mass that had been noted for 4 years. (a) Axial T_1 weighted MR image shows a well-defined mass with predominant hyperintensity. (b) On a T_2 weighted image, the mass shows a mixture of high and intermediate signal intensities with mosaic pattern. (c) Coronal post-contrast fat-suppressed T_1 weighted image shows ring enhancement of the tumour. (d) ADC mapping shows high values of this tumour ($2.0 \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$). The outline shows the region of interest in the tumour. (e) Axial T_1 weighted image taken 2 years prior to the onset of infarction shows a mass of intermediate signal intensity with a well-defined margin. ADC, apparent diffusion coefficient.

were a mixture of high and intermediate in 10 cases, and a mixture of high to low in 3. Internal architectures according to the distribution of T_2 signal hyperintensities were mosaic hyperintensity in three tumours, central hyperintensity in five, and multiseptated hyperintensity in six. The mean ADC value for the tumours was $1.5 \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$ with a standard deviation of $\pm 0.38 \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$. Post-contrast images were available in two cases, with both tumours showing ring-shaped enhancement.

Discussion

Previous reports have postulated several explanations for the propensity of Warthin's tumours to undergo infarction. First, these tumours contain many oncocyctic epithelial cells, with abundant mitochondria requiring a high oxygen supply, making them susceptible to vascular compromise. Second, Warthin's tumours have relatively few blood vessels within the body of the mass, which might also contribute to this tendency. Third, the advanced age of patients with a Warthin's tumour might increase their propensity to develop vascular thromboses.^{3,7,10} Among our cases, the histological type of the underlying tumour could be diagnosed as a Warthin's tumour in four cases (three were histologically diagnosed by surgically resected

material, and one was diagnosed cytologically prior to onset). In the remaining 10 cases, histological confirmation of the underlying tumour has not been possible, because surgical resection of parotid lesions showing significant reductions in size would be difficult to justify ethically. However, we speculate that considerable cases of the remaining 10 cases were also Warthin's tumours, because all of these occurred in elderly patients and were located in the parotid tail or had an associated synchronous parotid mass. These demographic and morphologic features are typical for a Warthin's tumour.^{17,18}

The usual presentation in our cases was of acute or subacute painful swelling of a pre-existing parotid mass, occasionally with facial nerve paralysis. These presentations are consistent with most previous reports,⁹⁻¹⁵ with tumours mimicking the appearance of carcinoma ex pleomorphic adenomas.¹⁹ The difficulty in making a cytological diagnosis from FNA biopsies in our cases was also consistent with previous case reports: among the reported seven cases of spontaneous infarcted parotid tumours, the cytological diagnosis was inconclusive in five and suspicious for a carcinoma in two.⁹⁻¹⁵ This difficulty in cytological diagnosis appeared to result from secondary changes to the infarction such as squamous metaplasia with extensive necrosis.^{3,13-15} Even based on a cytological

Figure 3 Case 5. A 60-year-old male presented with acute painful enlargement of a parotid mass that had been noted for 2 years. (a) Axial T_1 weighted image shows an ill-defined mass with a hyperintense portion. (b, c) On axial T_2 weighted and STIR images, the mass consisted of a central hyperintense area showing fluid–fluid interfaces and a peripheral intermediate intense area. (d) Microscopy of a low-power field in the tumour center shows a large cystic space including erythrocytes, inflammatory cells, and ghost cells. The viable tumour consisted of lymphoid stroma and papillary proliferation of epithelial cells, which are typical features for Warthin's tumour (hematoxylin and eosin staining). (e) Microscopy of a high-power field in the tumour margin shows fibrous connective tissue (arrows) and granulation tissue in the boundaries between tumour cells (Tumour) and normal parotid gland (Normal gland), which seems to have caused the ill-defined margins on MR images (hematoxylin and eosin staining). STIR, short tau inversion-recovery.

evaluation, these secondary changes are worrisome findings mimicking malignancy.^{8,20} Therefore, it is important for radiologists to be familiar with MRI findings of infarcted parotid tumours so that they can suggest their possibility to the treating clinicians.

The mean maximum cross-sectional tumour diameter (3.8 cm) in our cases was larger than those of typical Warthin's tumours and pleomorphic adenomas, which have been reported as 2.1–2.8 cm.^{21–25} This propensity for a larger size in our cases was consistent with previously reported spontaneously infarcted Warthin tumours of a mean diameter of 4.7 cm (range, 2.5–6.4),^{9–12} but not with infarcted pleomorphic adenomas of a mean diameter of 2.2 cm (range, 2.0–2.5).^{13–15} This larger size might be a characteristic of spontaneous infarction in Warthin's tumours.

As in a previous case that appeared to show malignancy,⁹ seven of our 14 cases showed ill-defined margins on MR imaging, suggesting inflammatory reactions. Generally, with the exception of low-grade mucoepidermoid carcinomas, ill-defined margins of parotid masses on MR images have been reported to predict high-grade malignant tumours or inflammatory disorders.^{26–30} Therefore, radiologists should keep in mind that even in cases of benign parotid tumours, the occurrence of infarction can cause ill-defined margins.

In accordance with MR features of pituitary apoplexy,^{31,32} all our cases had hyperintense areas on

T_1 weighted images, which occupied the predominant portion of the tumours in most cases. According to the cases in which histological correlations, cytological examinations, or post-contrast images were available, T_1 hyperintensity seems to indicate proteinaceous fluid containing haemorrhage and necrotic debris. Similarly, 36–63% of typical Warthin's tumours also had hyperintense areas on T_1 weighted images, reflecting cysts containing red cells, cholesterol crystals, foamy cells, and neutrophils, but these areas are usually small and focal.^{21,22} Therefore, we surmise that many Warthin's tumours involve subclinical infarction, resulting in small focal hyperintense areas on T_1 weighted images. In occasional cases, extensive infarction causes clinically relevant apoplectic symptoms with extensive hyperintense areas on T_1 weighted images, followed by spontaneous regression.

On T_2 weighted and STIR images, all tumours in our cases showed mixed signal intensities with various internal architectures, which might have depended on the degree of cystic degeneration. To the best of our knowledge, no other type of parotid tumour shows mosaic hyperintensity or multiseptal hyperintensity. In addition, the multiseptal hyperintensity resembled haemorrhagic necrosis of benign tumours in other organs such as ovarian or parathyroid adenomas.^{33–35} Therefore, although non-specific, mosaic hyperintensity or multiseptal hyperintensity on T_2 weighted images might aid in the diagnosis of an infarcted parotid tumour.

Figure 4 Case 12. A 69-year-old male presented with acute painful enlargement of a parotid mass that had been noted for 8 months. (a) and (b) Axial T_1 - and T_2 weighted images show a hyperintense mass with multiple septa of intermediate signal intensities, respectively. (c,d) Axial and coronal STIR images show the mass with infiltrative margins located in the parotid tail. (e) Follow-up MR examination taken 1 year after the onset shows marked reduction of the tumour. STIR, short tau inversion-recovery.

The main differential diagnosis in our cases was a parotid carcinoma, because an ill-defined margin on MRI is reported as suggestive of this type.^{26–30} Considering their differentiation, hyperintense area on T_1 weighted images might indicate an infarcted parotid tumour, because except for secretory carcinomas or acinic cell carcinomas, the presence of such features on T_1 weighted images is relatively rare in malignant parotid tumours.^{36,37} In addition, ADC values seem to be useful: our cases showed high values with a mean of $1.5 \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$, compared with those of parotid gland carcinomas with a mean of $0.8\text{--}1.1 \times 10^{-3} \text{ mm}^2 \text{ s}^{-1}$.^{38–40}

Our retrospective study has several limitations. First, it included a relatively small number of cases. Second, conclusive cytological or pathological diagnoses could be

made in only four cases. Therefore, further studies with a larger patient series with histopathological confirmation are needed. However, our presented MR features of infarcted parotid tumours are meaningful in a clinical setting because these tumours usually show spontaneous regression.

Conclusions

Half of infarcted parotid tumours had an ill-defined margin, which can mimic malignancy. However, a combination of the occurrence in the parotid tail and the presence of extensive hyperintense area on T_1 weighted image might help in their diagnosis.

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