Searching for mammary analog secretory carcinoma of salivary gland among its mimics

Andre Pinto¹, Vania Nosé², Claudia Rojas¹, Yao-Shan Fan¹ and Carmen Gomez-Fernandez¹

¹Department of Pathology, University of Miami Miller School of Medicine, Miami, FL, USA and ²Department of Pathology, Massachusetts General Hospital, Boston, MA, USA

Mammary analog secretory carcinoma of salivary gland is a recently described entity with unique morphologic, clinical, and genetic characteristics, including the characteristic t(12;15)(p13;q25) with *ETV6-NTRK3* translocation found in secretory carcinomas of the breast. Before their initial description, these salivary gland tumors were generally diagnosed as acinic cell carcinoma or adenocarcinoma. For the purpose of this study, all cases of salivary gland acinic cell carcinoma, cribriform cystadenocarcinoma, and adenocarcinoma, not otherwise specified (NOS), diagnosed over a 10-year period were retrieved from our surgical pathology files. There were a total of 11 cases diagnosed as acinic cell carcinoma, 10 cases of adenocarcinoma, NOS, and 6 cases of cribriform cystadenocarcinoma. All slides were reviewed by two pathologists (AP, CGF) and tumors that show morphologic features of mammary analog secretory carcinoma according to the recent literature were selected. This process narrowed down the initial number to six cases originally diagnosed as acinic cell carcinoma, three cases originally diagnosed as adenocarcinoma, NOS, and one case originally diagnosed as cribriform cystadenocarcinoma. The 10 cases were subjected to immunohistochemistry for S-100, mammaglobin, and ANO1, as well as fluorescence *in situ* hybridization analysis for t(12;15)(p13;q25) with *ETV6-NTRK3* fusion rearrangement. The *ETV6-NTRK3* gene rearrangement was detected in three tumors. These three tumors, initially diagnosed as acinic cell carcinomas, stained positive for S-100 and mammaglobin, and negative for ANO1 by immunohistochemistry. Two of the three patients were male (2/3). In summary, mammary analog secretory carcinoma is a newly described diagnostic entity that should be in the differential diagnosis of salivary gland tumors that morphologically mimic other neoplasms, mainly acinic cell carcinomas. They differ from conventional acinic cell tumors immunohistochemically and molecularly. Positivity for mammaglobin and S-100, and negativity for ANO1 are useful screening tools before confirmatory molecular studies.

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Mammary analog secretory carcinoma of the salivary gland is a recently described tumor with unique characteristics. The first description of mammary analog secretory carcinoma reported by Skalova *et al*¹ in 2010 included 16 cases of this new entity that shares morphologic and genetic characteristics with its mammary counterpart, including the *ETV6-NTRK3* fusion product previously described in secretory carcinomas of the breast. The authors selected their cases among salivary gland

neoplasms that morphologically resemble secretory carcinomas of the breast; ie, well-circumscribed nodules of neoplastic epithelial cells arranged in solid, microcystic, and tubular growth patterns associated with intraluminal secretions. Cytologically, the cells are bland, have vesicular nuclei and abundant pale-pink granular or vacuolated cytoplasm.^{1,2}

Following the initial publication describing mammary analog secretory carcinoma, other investigators began to look back at their own institutional cases in order to reanalyze some of their salivary gland tumors. Acinic cell carcinomas, adenocarcinomas, not otherwise specified (NOS), polymorphous low-grade adenocarcinomas, mucoepidermoid carcinomas, and cystadenocarcinomas are examples of cases re-evaluated in a small number of recently published studies, describing additional cases of mammary analog secretory carcinoma. Most cases of

Correspondence: Dr C Gomez-Fernandez, MD, Department of Pathology, University of Miami Miller School of Medicine, 1400 NW 12 Avenue, Room 4076, Miami, FL 33136, US.
E-mail: CGomez3@med.miami.edu

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mammary analog secretory carcinoma had been initially classified as acinic cell carcinomas.^{3–7}

Immunohistochemistry studies may be useful in the differential diagnosis between mammary analog secretory carcinoma and its mimics. The former are reactive for S-100 protein and mammaglobin in the vast majority of cases,^{1,3–6,8} however, these two markers, S-100 in particular, are likewise found to be occasionally positive in other adenocarcinomas of salivary gland origin. Acinic cell carcinomas tend to be negative for mammaglobin, but can rarely demonstrate immunoreactivity for S-100. ANO1 (also known as DOG1), initially described in gastrointestinal stromal tumors, is a marker of salivary acinar and intercalated duct differentiation. A recent study by Chenevert *et al*, described the positive immunohistochemical expression of the ANO1 protein in all of their cases of acinic cell carcinomas, whereas the mammary analog secretory carcinomas were largely negative for this marker.⁹

Molecularly, mammary analog secretory carcinomas, like their breast counterpart, harbor a balanced translocation t(12;15)(p13;q25), which creates an *ETV6-NTRK3* fusion gene.¹ This same genetic aberration is also seen in cellular mesoblastic nephroma,¹⁰ congenital fibrosarcoma,¹¹ and in some hematologic malignancies,^{12,13} but has not been described in other neoplasms of the salivary gland.

The aim of this study is to reanalyze our own database of salivary gland cases for the presence of mammary analog secretory carcinoma using the recently described morphologic criteria, together with immunohistochemistry for mammaglobin, S-100, and DOG1 and fluorescent *in situ* hybridization (FISH) analyses for *ETV6-NTRK3* translocation product.

Materials and methods

All cases of acinic cell carcinoma, adenocarcinoma NOS, and cribriform cystadenocarcinoma of salivary gland origin, from three institutions (Jackson Memorial Hospital, Sylvester Comprehensive Cancer Center, and University of Miami Hospital) over a 10-year period, were retrieved. A total of 27 cases were identified, including 11 cases of acinic cell carcinoma, 10 cases of adenocarcinoma NOS, and 6 cases of cribriform cystadenocarcinoma.

Hematoxylin and eosin stained slides were reviewed, and tumors that morphologically resembled mammary analog secretory carcinoma according to the recent literature were selected. Those features included epithelial cells with abundant vacuolated cytoplasm, minimal nuclear pleomorphism, papillary, and microcystic architecture with cellular hobnailing and eosinophilic secretions (Figure 1).

This process narrowed down the initial number to 10 cases (6 acinic cell carcinomas, 3 adenocarcinomas, NOS, and 1 cribriform cystadenocarcinoma) on

which S-100, mammaglobin and ANO1 (DOG1) immunostains were performed (Table 1). Immunohistochemistry was performed using Dako antibodies in 5- μ m paraffin-embedded tissues using appropriate tissue-positive controls. All immunohistochemical staining was performed on automatic stainers using antibody dilutions previously determined by pathologists during antibody validation, after appropriate antigen retrieval. Antigen retrieval was performed in a pre-treatment station using both low pH (pH 6, citrate buffer) and high pH (pH 9, EDTA buffer) environments. Staining detection was carried out using a modified biotin–streptavidin–horseradish peroxidase method (Figure 2).

FISH analysis for the characteristic t(12;15)(p13;q25) with *ETV6-NTRK3* fusion was performed using slides prepared from formalin-fixed paraffin-embedded tissues with 3- μ m-thick sections. A dual color *ETV6-NTRK3* break-apart FISH probe (Abbot Molecular, Des Plaines, Illinois, USA) was used to detect translocation and copy number changes of the *ETV6-NTRK3* gene. FISH was performed using a protocol recommended by the manufacturer of the DNA probe. The major steps included slide deparaffinization, codenaturation of probe DNA and tissue materials on slides, hybridization, post hybridization washes, and counterstaining with DAPI. Hybridization signals were analyzed with a fluorescence microscope equipped with a Cyto-vision system for image capturing and storage (Applied Imaging Corp, USA). For each case, 200 cells were analyzed with result described using the standard cytogenetics nomenclature (ISCN, 2009).¹⁴

Results

FISH studies confirmed *ETV6-NTRK3* rearrangement in 3 of the 10 cases. These three tumors had been initially diagnosed as acinic cell carcinoma. Immunohistochemistry studies demonstrated that each was positive for S-100 and mammaglobin and negative for ANO1 (Table 2).

The ages for the three patients with tumors harboring the *ETV6-NTRK3* translocation ranged from 29 to 61 years (average 47.6, median 53 years). Two of the patients were male. Grossly, the tumors were generally described as circumscribed, gray-tan cystic masses, and ranged from 1.8 cm to 3.0 cm in greatest dimension. Histologically, all cases demonstrated a mixture of micropapillary, microcystic, and solid patterns of growth with an overall lobulated architecture. The microcystic spaces contained characteristic eosinophilic bubbly secretions, whereas the micropapillary growth pattern consisted of tumor cells surrounding a fibrovascular core in a ‘hobnail’ pattern. The tumor cells had minimal nuclear pleomorphism with abundant eosinophilic or vacuolated cytoplasm. No necrosis, perineural, or lymphovascular invasion was identified in any of the cases in our series. Lymph node

Figure 1 Mammary analogue secretory carcinoma (MASC) of the parotid gland showing large epithelial cells with plump, eosinophilic cytoplasm arranged in a microcystic pattern of growth (a). Solid sheets of tumor cells can also be seen (b). Bland, vesicular nuclei are characteristic, as well as hobnailing of the cells (c). At higher magnification, a bubbly cytoplasm is noticeable, and pale pink secretion is easily identified within the microcystic spaces (d). a: x100 original magnification, b: x100 original magnification, c: x200 original magnification, d: x400 original magnification. All images are hematoxylin and eosin (H&E).

Table 1 Cases selected for the study

Case	Age (years)/ sex	Initial diagnosis	Final diagnosis	S-100	MMG	ANO1 (DOG1)	FISH
1	53/M	AciCC	MASC	+	+	–	Rearrangement of ETV6
2	29/M	AciCC	MASC	+	+	–	Rearrangement of ETV6
3	61/F	AciCC	MASC	+	+	–	Rearrangement of ETV6
4	48/M	AciCC	AciCC	+	+	–	Negative
5	83/M	AciCC	AciCC	+	–	+	Deletion of ETV6
6	59/F	AciCC	AciCC	–	–	+	Negative
7	82/F	Adenocarcinoma, NOS	Adenocarcinoma, NOS	+	–	–	Negative
8	76/M	Adenocarcinoma, NOS	Adenocarcinoma, NOS	–	–	–	Amplification of ETV6
9	57/M	Adenocarcinoma, NOS	Adenocarcinoma, NOS	+	–	–	Negative
10	73/M	Cribriform cystadenocarcinoma	Cribriform cystadenocarcinoma	–	+	–	Negative

Abbreviations: AciCC, acinic cell carcinoma; Diff: differentiated; FISH, fluorescent *in situ* hybridization; MMG, mammoglobin.

Figure 2 Immunohistochemical profile of the MASC cases identified in this study. All tumors were immunoreactive for S-100, which in this example had strong and diffuse staining (a). Mammaglobin (MMG) is positive in MASC, as this entity represents the salivary gland counterpart of the breast carcinoma (b). DOG1, a marker for acinic cell differentiation, is negative in MASC. Notice the remaining non-neoplastic acini with the typical intercalated duct staining in the upper left corner (c). All figures: immunohistochemical stains; x200 original magnification.

Table 2 Clinico-pathologic data on cases with confirmed ETV6 gene rearrangement

Sex	Age (years)	Specimen	Initial diagnosis	LN	Tumor size (cm)	PNI	Stage	S-100	MMG	ANO1 (DOG1)	FISH
M	53	Parotid	AciCC	0/2	3.0	—	T1 N0 Mx	>50%, strong	>50%, strong	—	Rearrangement of ETV6
F	61	Parotid	AciCC	0/9	1.8	—	T1 N0 Mx	10–50%, strong	1–10%, strong	—	Rearrangement of ETV6
M	29	Parotid	AciCC	4/21	3.0	—	T3 N2b Mx	>50%, strong	>50%, strong	—	Rearrangement of ETV6

Abbreviations: AciCC, acinic cell carcinoma; LN, lymph nodes; PNI, perineural invasion.

metastasis was present in one confirmed case on which node dissection was performed. No distant metastases were identified at the time of presentation in any of patients (Figure 3).

With respect to the other cases in our study subjected to FISH analysis and immunohistochemistry, one case demonstrated amplification of the

ETV6 gene, and one case had deletion of *ETV6*. Both of these cases belonged to male patients. The remaining five cases had no cytogenetic abnormalities detected. By immunohistochemistry, the three cytogenetically confirmed cases were positive for S-100 and mammaglobin and negative for DOG1. Interestingly, there was one other case with the same

Figure 3 Adenocarcinoma, NOS. Although this case had areas that resembled MASC-like abundant eosinophilic cytoplasm, nuclear pleomorphism and histological grade were higher than typically seen on this recently recognized entity. This case was also strongly positive for S-100 (**b**). **a**: H&E, x400 original magnification, **b**: x200 original magnification.

immunohistochemistry results, which was morphologically compatible with mammary analog secretory carcinoma, but failed to demonstrate the characteristic *ETV6-NTRK3* translocation.

Discussion

The recent identification of mammary analog secretory carcinoma as a primary salivary gland tumor with a characteristic molecular alteration, but with morphologic features that overlap primarily with those of acinic cell carcinoma has prompted several groups to review their archival cases. Acinic cell carcinomas have a broad spectrum of histologic patterns, including solid, microcystic, papillary-cystic, and follicular. Thus, salivary gland tumors with these architectural features, have been historically classified as acinic cell carcinomas, and to a lesser extent, as adenocarcinomas NOS.^{3,4,7} Other entities like cystadenocarcinoma and mucoepidermoid carcinoma have also been used as potential diagnoses for what we now understand may represent mammary analog secretory carcinoma.³

In determining the diagnostic categories for the retrospective analysis of our archival cases, acinic cell tumors were selected as most of the newly diagnosed mammary analog secretory carcinomas have been shown to be initially classified as such. Cribriform cystadenocarcinomas have also been cited as possible diagnoses for mammary analog secretory carcinomas. We also decided to include cases diagnosed as adenocarcinoma, NOS, as 'unclassifiable' salivary gland adenocarcinomas may be inadvertently placed into this category as a diagnosis of exclusion. We chose not to review other tumors types (polymorphous low-grade adenocarcinoma, mucoepidermoid carcinoma, among others) as these have specific histomorphologic characteristics, which we believe are separate and distinct

from those in the classic description of mammary analog secretory carcinoma.

In the review of our cases, we found that the overlap between mammary analog secretory carcinoma and acinic cell carcinoma, in particular, may be quite striking. As an example, one of our cases diagnosed as acinic cell carcinoma was morphologically and immunophenotypically indistinguishable from mammary analog secretory carcinoma but failed to show the characteristic *ETV6-NTRK3* gene rearrangement by FISH, underscoring the importance of this ancillary technique in this differential (Figure 4).

Reclassifying tumors is becoming a regular exercise for the modern pathologist. Supported by the increasing sophistication and feasibility of molecular techniques, this practice is becoming more and more common on a routine basis, and this may or may not have significant clinical implications for the patient. When, for instance, two neoplasms that have distinct genetic profiles, but similar therapeutic options and outcomes, are separated, no major impact is produced clinically. However, if tumors are biologically distinct enough to the point that the treatment will differ, we as pathologists have a major role in patient care.

This phenomenon still seems unclear when analyzing the behavior of mammary analog secretory carcinoma because of the paucity of cases reported. A male predilection is undoubtedly noted,^{1,3-5} and differs from acinic cell carcinoma, which has a slight female predominance. Parotid is the salivary gland most commonly involved,^{1,3,5} although these tumors may occur in other major and minor salivary glands. A recent study has suggested that 'acinic cell carcinomas' that occur outside of the parotid are actually mammary analog secretory carcinomas.¹⁵ A higher incidence of nodal metastasis and poorer disease-free survival seem to occur when compared with acinic cell carcinoma.^{3,4}

Figure 4 The overlap between MASC and AcicC is evident in these examples of cases from our study. (a) Illustrates an AcicC (negative for ETV6-NTRK3 translocation), while panel b is a case of MASC with proven translocation. Notice that both tumors have microcysts, low-grade nuclei, and proteinaceous secretions, among other similar features. FISH showing rearrangement of the *ETV6* gene as indicated by separation of the green signal (centromeric part of the gene) from the red signal (telomeric part of the gene). The normal *ETV6* gene in the same cell is visualized as a green-red fusion signal (c). a, b: H&E, x200 original magnification.

Table 3 Summary of S-100 and mammaglobin immunoexpression in published studies of mammary analog secretory carcinomas

Reference	Cases	Mean age (years)	Sex	Tumor site	S-100	MMG	FISH
Skalova <i>et al</i> ¹	16	46	9M/7F	13 Parotid gland, 3 minor salivary glands	16/16	16/16	15/16
Chiosea <i>et al</i> ⁴	10	45	8M/2F	NA	10/10	NA	10/10
Connor <i>et al</i> ⁵	7	40	6M/1F	4 Oral cavity, 2 parotid, 1 submandibular gland	5/7	NA	7/7
Fehr <i>et al</i> ⁶	4	52	1M/3F	2 Parotid, 1 soft palate, 1 oral mucosa	4/4	4/4	4/4
S Ito <i>et al</i> ⁸	1	37	F	Parotid	1/1	NA	1/1
Bishop <i>et al</i> ¹⁹	11	52	4M/7F	2 Submandibular gland, 9 minor salivary glands	NA	11/11	11/11
Levine <i>et al</i> ²⁰	1	34	F	Accessory parotid	1/1	1/1	NA
Pisharodi <i>et al</i> ²¹	1	69	F	Parotid	1/1	1/1	1/1
Griffith <i>et al</i> ²²	6	44	3M/3F	4 Parotid, 1 buccal mucosa, 1 submandibular gland	2/6	NA	3/6
Rastatter <i>et al</i> ²³	1	14	F	Parotid	1/1	NA	1/1

Our data revealed a higher predominance of these tumors in men, which correlates with the current literature. Immunohistochemistry for S-100 seems to be a very useful technique for distinguishing mammary analog secretory carcinoma, typically positive for this marker, from acinic cell carcinoma,

reported to be positive in only 10% of cases.^{16,17} The S-100 staining pattern for mammary analog secretory carcinoma can range from partial to diffuse and from weak to intense. However, it is important to remember that this marker has to be used in conjunction with morphology, as many

other salivary gland tumors like monomorphic and pleomorphic adenomas, polymorphous low-grade adenocarcinomas, and adenoid cystic carcinomas can be reactive for this immunostain.¹⁸

Similar to its mammary counterpart, mammary analog secretory carcinomas are positive for mammaglobin.^{1,6,19} Although reportedly negative in most cases of acinic cell carcinoma,^{1,15} this marker has not been widely tested among other salivary gland tumors. Table 3 contains a review of studies about the expression of S-100 and mammaglobin in mammary analog secretory carcinomas.

ANO1, also known as DOG1, was recently shown to be an indicator of salivary acinar cell and intercalated duct differentiation.⁹ It tends to be positive in acinic cell carcinoma, with a characteristic apical-luminal membranous and intercalated duct staining, and negative in mammary analog secretory carcinoma. Therefore, this stain may be used as a negative marker in an immunohistochemical panel for distinguishing cases with morphologic overlap between mammary analog secretory carcinoma and acinic cell carcinoma. All the cases initially classified as acinic cell tumors in our series demonstrated negative immunoreactivity for this marker, and were re-classified as mammary analog secretory carcinomas after FISH studies showed a positive ETV6 translocation.

Of note, an unusual finding in our study was the presence of *ETV6* amplification in tumor cells seen in one case, and deletion of this gene in the majority of cells in another. Both tumors were present in the parotid gland of male patients. The significance of these genetic alterations remain unclear, however, they may indicate that alternative pathways involving the *ETV6-NTRK3* gene could be related to the pathogenesis of this tumor, assuming that those cases represent mammary analog secretory carcinoma.

Conclusion

Mammary analog secretory carcinoma is a new diagnostic entity that should be in the differential diagnosis of salivary gland tumors that predominantly mimic acinic cell carcinoma. Although morphologically similar, they differ from conventional acinic cell carcinoma immunohistochemically and molecularly. They predominantly affect male patients. Positivity for mammaglobin and S-100 and negativity for ANO1 are useful screening tools before further confirmatory cytogenetic studies demonstrating the *ETV6-NTRK3* fusion product.

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Disclosure/conflict of interest

The authors declare no conflict of interest.

References

- Skalova A, Vanecek T, Sima R, *et al*. Mammary analogue secretory carcinoma of salivary glands, containing the ETV6-NTRK3 fusion gene: a hitherto undescribed salivary gland tumor entity. *Am J Surg Pathol* 2010;34:599–608.
- Tavassoli FA, Norris HJ. Secretory carcinoma of the breast. *Cancer* 1980;45:2404–2413.
- Chiosea SI, Griffith C, Assaad A, *et al*. Clinicopathological characterization of mammary analogue secretory carcinoma of salivary glands. *Histopathology* 2012;61:387–394.
- Chiosea SI, Griffith C, Assaad A, *et al*. The profile of acinic cell carcinoma after recognition of mammary analog secretory carcinoma. *Am J Surg Pathol* 2012;36:343–350.
- Connor A, Perez-Ordóñez B, Shago M, *et al*. Mammary analog secretory carcinoma of salivary gland origin with the ETV6 gene rearrangement by FISH: expanded morphologic and immunohistochemical spectrum of a recently described entity. *Am J Surg Pathol* 2012;36:27–34.
- Fehr A, Loning T, Stenman G. Mammary analogue secretory carcinoma of the salivary glands with ETV6-NTRK3 gene fusion. *Am J Surg Pathol* 2011;35:1600–1602.
- Lei Y, Chiosea SI. Re-evaluating historic cohort of salivary acinic cell carcinoma with new diagnostic tools. *Head Neck Pathol* 2012;6:166–170.
- Ito S, Ishida E, Skalova A, *et al*. Case report of mammary analog secretory carcinoma of the parotid gland. *Pathol Int* 2012;62:149–152.
- Chenevert J, Duvvuri U, Chiosea S, *et al*. DOG1: a novel marker of salivary acinar and intercalated duct differentiation. *Mod Pathol* 2012;25:919–929.
- Rubin BP, Chen CJ, Morgan TW, *et al*. Congenital mesoblastic nephroma t(12;15) is associated with ETV6-NTRK3 gene fusion: cytogenetic and molecular relationship to congenital (infantile) fibrosarcoma. *Am J Pathol* 1998;153:1451–1458.
- Knezevich SR, McFadden DE, Tao W, *et al*. A novel ETV6-NTRK3 gene fusion in congenital fibrosarcoma. *Nat Genet* 1998;18:184–187.
- Chi HT, Ly BT, Kano Y, *et al*. ETV6-NTRK3 as a therapeutic target of small molecule inhibitor PKC412. *Biochem Biophys Res Commun* 2012;429:87–92.
- Euhus DM, Timmons CF, Tomlinson GE. ETV6-NTRK3–Trk-ing the primary event in human secretory breast cancer. *Cancer Cell* 2002;2:347–348.
- Shaffer LG, Slovak ML, Campbell LJ. ISCN 2009: An international system for human cytogenetic nomenclature. Karger Publishing: Basel, Switzerland, 2009.
- Bishop JA, Yonescu R, Batista D, Westra W. Most non-parotid ‘Acinic Cell Carcinomas’ represent mammary analogue secretory carcinomas. *Am J Surg Pathol*; advance online publication, 15 May 2013 (e-pub ahead of print).
- Barnes L, Eveson J, Reichart P, *et al*. World Health Organization Classification of Tumours 2003;216–218.
- Takahashi H, Fujita S, Okabe H, *et al*. Distribution of tissue markers in acinic cell carcinomas of salivary gland. *Pathol Res Pract* 1992;188:692–700.

- 18 Zarbo RJ, Regezi JA, Batsakis JG. S-100 protein in salivary gland tumors: an immunohistochemical study of 129 cases. *Head Neck Surg* 1986;8:268–275.
- 19 Bishop JA, Yonescu R, Batista D, Westra W. Utility of mammoglobin immunostaining in the diagnosis of mammary analogue secretory carcinoma of the salivary glands. *Mod Pathol* [abstract] 2013;26:304A.
- 20 Levine P, Fried K, Krevitt LD, *et al*. Aspiration biopsy of mammary analogue secretory carcinoma of accessory parotid gland: another diagnostic dilemma in matrix-containing tumors of the salivary glands. *Diagn Cytopathol* 2012 (e-pub ahead of print).
- 21 Pisharodi L. Mammary analog secretory carcinoma of salivary gland: cytologic diagnosis and differential diagnosis of an unreported entity. *Diagn Cytopathol* 2013;41:239–241.
- 22 Griffith CC, Stelow EB, Saqi A, *et al*. The cytological features of mammary analogue secretory carcinoma: A series of 6 molecularly confirmed cases. *Cancer Cytopathol* 2013;121:234–241.
- 23 Rastatter JC, Jatana KR, Jennings LJ, *et al*. Mammary analogue secretory carcinoma of the parotid gland in a pediatric patient. *Otolaryngol Head Neck Surg* 2012;146:514–515.