

Malignant adenomyoepithelioma with *HRAS* G13R mutation: A case report

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Received July 30, 2025; Accepted January 14, 2026

DOI: 10.3892/ol.2026.15475

Abstract. Adenomyoepithelioma (AME) of the breast is a rare tumor characterized by epithelial and myoepithelial differentiation, with a hotspot mutation in *HRAS* Q61. Malignant transformation occurs from either the luminal or myoepithelial component. In the present study, a malignant AME (M-AME) with pulmonary metastasis is reported. M-AME is rare, and the present report highlights the genomic alteration of primary and metastatic lesions. A 53-year-old Japanese woman presented to a neighboring clinic with a palpable and non-tender mass in the left breast. The mass was suspected to be AME, and left mastectomy was performed at Osaka Medical and Pharmaceutical University (Takatsuki, Japan). Breast tumor specimens exhibited epithelial and myoepithelial proliferation with myoepithelial mitoses and necrosis, and the tumor was diagnosed as M-AME. Following chemotherapy, a lung tumor was identified 2 years after the operation. The pulmonary tumor indicated myoepithelial cell proliferation along the bronchiolar epithelium, and myoepithelial cells exhibited clear or epithelioid features. Both the breast and lung tumor exhibited the *HRAS* G13R mutation, which is frequently observed in M-AME. This mutation may be considered a driver mutation in mammary M-AME.

Introduction

Adenomyoepithelioma (AME) of the breast, which was initially described by Hamperl in 1970 (1), is a biphasic tumor characterized by small epithelium-lined spaces with inner luminal ductal cells and a proliferation of variably enlarged myoepithelial cells. AME is rare, and one AME case (0.048%) was found

in a series of 2078 consecutive breast tumors diagnosed by core needle biopsy, with a benign clinical course (2,3). In contrast to these observations, malignant AME (M-AME) is AME with carcinoma features, in which the malignancy may arise from either luminal epithelial or myoepithelial components, or from both cell types. M-AME is extremely rare, and no data on incidence have been reported (4) and the prognosis depends on the histological subtype of the invasive diseases (5). The low number of reported cases and short periods of follow-up limit the available information on prognostic and genetic features in M-AME. Genetically, breast cancers display complex somatic mutations and significant molecular heterogeneity, and *TP53*, *PIK3CA* and GATA binding protein 3 (*GATA3*) are the only three genes recurrently mutated in >10% of unselected breast cancers (6), while, the genetic drivers of AME depend on the estrogen receptor (ER). ER-positive AME display *PIK3CA* or *AKT1* activating mutations, while ER-negative AME harbor highly recurrent *HRAS* Q61R hotspot mutations (5,7). Mitogen-activated protein kinase (MAPK) and PI3K-AKT pathways are the two major downstream intracellular pathways of oncogenesis (8). The MAPK pathway is composed of *RAS*, *RAF*, *MEK* and extracellular signal-regulated kinase (*ERK*). *RAS* contains 3 closely related proteins encoded by the three following genes: *HRAS*, *KRAS* and *NRAS*. The activation caused by these gene mutations leads to uncontrolled cell growth and promotes oncogenesis (9). The global incidence of *HRAS*-mutant tumors is 7% of all *RAS*-mutant tumors (10), and the identification of the *HRAS* mutation aids in confirming a diagnosis, and selecting an effective medicine. For example, *HRAS* mutations are a frequent tumorigenic gene alteration in salivary gland epithelial-myoepithelial carcinoma (EMC), which indicates biphasic tubular structures, usually composed of tightly coupled inner ductal and prominent outer myoepithelial cells (11). The *HRAS* mutation is useful for diagnosing salivary gland EMC, and discriminates it from its mimics (11). Moreover, medical drugs that target *HRAS* mutant tumors are available (12).

In the present study, a M-AME case with pulmonary metastasis is reported. Genetical analysis of breast M-AME with distant metastasis is limited, and this is the first M-AME case with *HRAS* Q13R mutation, which was confirmed during the examination of primary and metastatic lesions. Moreover,

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Key words: malignant adenomyoepithelioma, *HRAS*, breast cancer, lung, case report

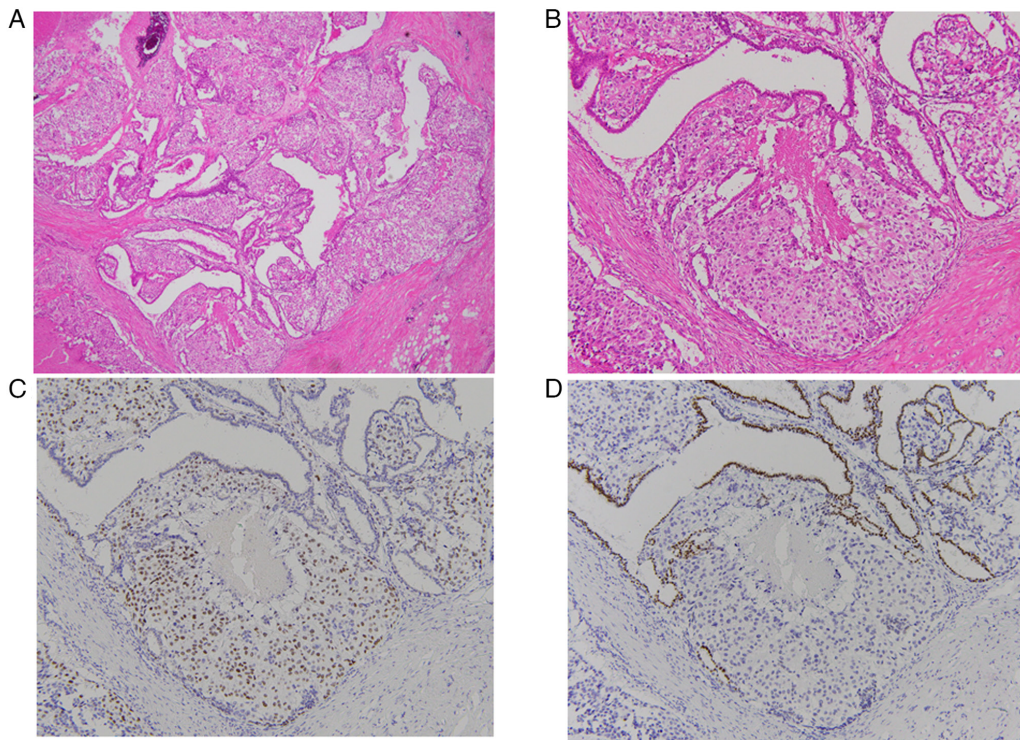


Figure 1. Microscopic findings of the breast tumor. (A) Biphasic proliferation of myoepithelial cells and papillary epithelial cells (hematoxylin-eosin stain; magnification, x40). (B) High power view of (A) (hematoxylin-eosin stain; magnification, x100). (C) Immunohistochemistry indicated that the myoepithelial cells were positive for p63 (magnification, x100). (D) Immunohistochemistry indicated that epithelial cells were positive for GATA binding protein 3 (magnification, x100).

16 cases of M-AME are reviewed including the present case, and the incidence of the frequent *HRAS* G13R mutation in M-AME is highlighted.

Case report

Case presentation and pathology. A 53-year-old Japanese woman presented to a local clinic with a palpable and non-tender mass in her left breast in October 2022. The patient had no other symptoms. The mass was located at the C region, with a maximum diameter of 2.5 cm. The core needle biopsy was indicative of AME and malignancy was not ruled out. Her medical and family history was unremarkable. Total mastectomy and sentinel lymph node dissection were performed at Osaka Medical and Pharmaceutical University in November 2022. A gray mass (2.5x2.0x1.8 cm) was noted. For histological analysis, the surgical specimens were fixed in 10% buffered formalin for 24 h at room temperature (RT) and embedded in paraffin. The sections (4 μ m-thickness) from the paraffin block were stained with hematoxylin for 5 min and eosin for 1 min at RT. The stained sections were examined under a light microscope (BX53; Olympus Corporation). Histologically, clear and epithelioid myoepithelial cells surrounding the papillary glandular epithelium were noted in hematoxylin-eosin (H&E) stain (Fig. 1A and B). Myoepithelial cells exhibited mitoses (12/10 high power fields) and moderate or severe atypia. Necrosis was also present. Metaplastic change was not observed. Sentinel lymph nodes indicated lack of metastasis. For subsequent analysis, immunohistochemistry (IHC) was carried out. Briefly, 4 μ m-thick sections obtained from the paraffin block

were stained with primary antibodies. For IHC, automated immunostaining devices, VENTANA BenchMark ULTRA (Roche Diagnostics) was used. After deparaffinization with EZ buffer (Roche Diagnostics), blocking endogenous peroxidases and antigen retrievals, the used antibodies were p63 (4A4, cat. no. 518-101961), S100 (polyclonal, cat. no. 518-110109), GATA3 (L50-823, cat no. 518-111953), thyroid transcription factor-1 (TTF-1, SP141, cat. no. 518-110871), estrogen receptor (ER, SP1, cat. no. 518-107932), progesterone receptor (PgR, 1E2, cat. no. 518-102463) and HER2(4B5, cat no. 790-2991). The stained sections were observed under a light microscope (BX53). IHC revealed that myoepithelial cells were positive for p63 (Fig. 1C) and S-100, and epithelial cells were positive for GATA3 (Fig. 1D) and negative for TTF-1. From these findings, malignant adenomyoepithelioma (T2N0M0) was diagnosed. Both epithelial and myoepithelial cells were negative for ER, PgR and HER2. After the operation, the administration of epirubicin combined with cyclophosphamide followed by docetaxel was performed with the patient's consent. There were no adverse and unanticipated events.

Twenty-six months later, chest computed tomography indicated a mass in the right lung (S6), and segmentectomy of the lung was performed. The tumor cells were 1.5 cm with a clear margin, and histologically, myoepithelial cells had proliferated along with bronchiolar epithelium (Fig. 2A). Myoepithelial cells with clear or epithelioid features were positive for p63 (Fig. 2B) and S-100, and epithelial cells were positive for TTF-1 (Fig. 2C); a low number of epithelial cells was positive for GATA3. From these findings, pulmonary metastasis from breast tumor was diagnosed. The patient was seen in

Table I. PCR primers used for Sanger sequencing.

Gene	Direction	Sequence (5'-3')
<i>HRAS</i> exon 2	Forward	CAGGCCCTGAGGAGCGATG
	Reverse	TTCGTCCACAAAATGGTTCT
<i>HRAS</i> exon 3	Forward	TCCTGCAGGATTCCTACCGG
	Reverse	GGTTCACCTGTACTGGTGGGA

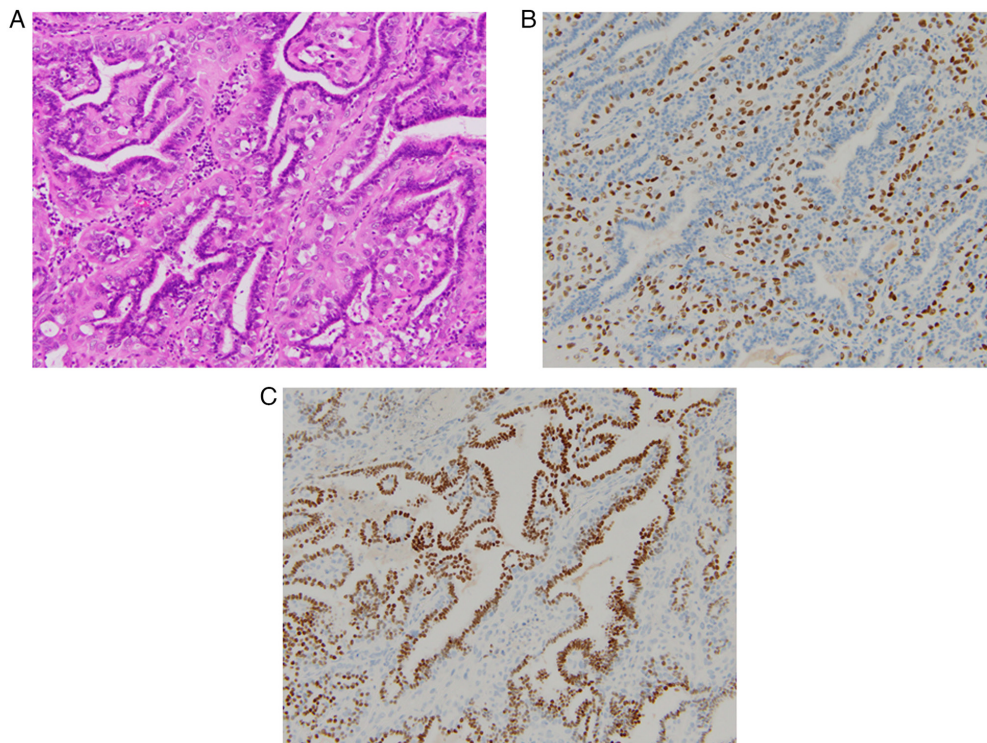


Figure 2. Microscopic findings of the lung tumor. (A) Myoepithelial cells beneath bronchiolar epithelium (hematoxylin-eosin stain; magnification, x200). (B) Immunohistochemistry indicated that myoepithelial cells were positive for p63 (magnification, x200). (C) Immunohistochemistry indicated that bronchiolar epithelium was positive for thyroid transcription factor-1 (magnification, x200).

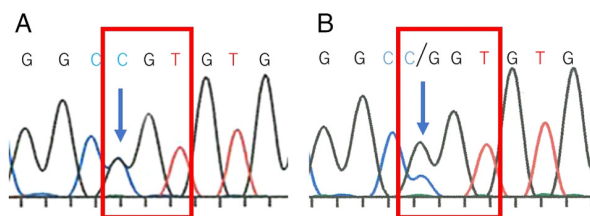


Figure 3. DNA sequence of *HRAS* in (A) breast and (B) lung tumors. Both breast and lung tumors exhibited *HRAS* G13R. The arrow indicates the mutation (c.37G>C) and the box indicates codon 13 (p.G13R).

formalin-fixed paraffin-embedded tissue was extracted with a QIAamp DNA FFPE Tissue Kit (Qiagen, Inc.). The tumor component of the slides was microdissected to increase the tumor cell ratio. PCR products were purified using a QIAquick Spin Kit (Qiagen, Inc.). Each purified product was directly sequenced using a forward primer with a BigDye Terminator version 3.1 cycle sequencing kit on an ABI 3730 instrument (Thermo Fisher Scientific, Inc.). A mutation analysis of *HRAS* (exons 2 and 3) was performed, and the primer sequences are listed in Table I. The present case harbored *HRAS* G13R mutation in breast and lung tissues (Fig. 3).

December 2025, and complained of occasional dizziness, but exhibited no evidence of recurrence on computed tomography. Follow-up visits are scheduled every three months.

Molecular genetic analysis. To investigate whether the mutational status of *HRAS* in breast and lung is the same, a polymerase chain reaction (PCR) followed by Sanger's sequencing was performed (11,13). Briefly, DNA from

Literature review

Literature analysis was performed using PubMed database (<https://pubmed.ncbi.nlm.nih.gov/>) using the key words 'adenomyoepithelioma' and 'breast'. Within these terms, M-AME with genetic analyses and clinical findings was selected. In total, 16 M-AME including the case reported in the present study were selected (14-16). The clinical and pathological

Table II. Cases of malignant adenomyoepithelioma with genetical analyses.

First author/s, year	Case	Age, years	Size, cm	Follow-up, months	Lymph node metastasis	Metastasis	Architecture	Myoepithelial cells	Mitosis	Necrosis	ER	<i>HRAS</i>	<i>PIK3CA</i>	<i>AKT1</i>	<i>APC</i>	ATM	(Refs.)
Lubin <i>et al</i> , 2019	1	69	0.7	85	NA	No	Papillary	Clear, epithelioid	12/10 HPF	NA	(+)	NA	NA	E17K	NA	NA	(14)
	2	55	1.4	8	NA	No	Tubular	Epithelioid	15/10 HPF	(+)	(-)	NA	NA	E17K	NA	F858L	
	3	73	2.5	NA	NA	NA	Tubular	Clear	5/10 HPF	(+)	(-)	NA	NA	NA	P870S, A1564P	NA	
	4	67	1.0	NA	NA	NA	Lobulated	Clear	10/10 HPF	NA	(+)	NA	NA	NA	E1317Q	NA	
	5	78	1.5	NA	NA	NA	Tubular	Clear	16/10 HPF	(+)	(+)	NA	H1047R	NA	NA	NA	
	6	78	1.6	NA	NA	NA	Tubular	Clear	4/10 HPF	NA	(-)	NA	NA	NA	NA	NA	
	7	65	1.0	12	NA	Chest wall	Spindle	Spindle	10/10 HPF	NA	(-)	Q61K	H1047R, H1065L	NA	NA	NA	
Ginter <i>et al</i> , 2020	8	78	1.4	23	NA	No	Papillary, lobulated	Epithelioid, clear	12/10 HPF	NA	(-)	NA	H1047P	NA	NA	NA	(15)
	9	66	1.2	37	(+)	No	Lobulated, spindled	Spindled, clear	5/10 HPF	NA	(+)	NA	NA	E17K	NA	NA	
	10	42	4.8	16	(-)	No	Lobulated, tubular	Clear	20/10 HPF	NA	(-)	NA	NA	NA	NA	NA	
	11	56	2.2	24	(+)	DOD	Papillary	Epithelioid, spindle, clear	13/10 HPF	NA	(-)	G12D	NA	NA	NA	NA	
Bièche <i>et al</i> , 2021	12	84	2.5	12	NA	Recurrence	Tubular, lobulated	Clear	3/mm ²	(+)	(+)	G13R	wt	wt	NA	NA	(16)
	13	76	1.8	NA	NA	NA	Tubular, lobulated	Clear	3/mm ²	(+)	(-)	G13R	H1047R	wt	NA	NA	
	14	60	1.9	75	NA	No	Tubular, spindle, cystic	Clear, spindle	6/mm ²	(+)	(-)	G13R	H1047R	wt	NA	NA	
	15	55	5.5	11	(+)	Lung	Tubular	Clear	10/mm ²	(-)	(+)	G12S	wt	wt	NA	NA	
Present study	16	53	2.5	31	(-)	Lung	Papillary	Clear, epithelioid	12/10 HPF	(+)	(-)	G13R	ND	ND	ND	ND	-

DOD, died of disease; HPF, high power field; NA, not available; ND, not done; ER, estrogen receptor; wt, wild-type.

characteristics of these cases are detailed in Table II. All patients were female, and the median patient age at diagnosis was 66 years (range, 42-84 years). Metastasis to a lymph node and lung was seen in 3 and 2 cases, respectively. Two cases indicated recurrence. A total of 3 cases harbored mutations in both *HRAS* and *PIK3CA* genes, and within the *HRAS* mutation, G13R, Q61K, G12D and G12S were noted in 4 (25%), 1 (6%), 1 (6%) and 1 case (6%), respectively. ER-negative *HRAS* G13R and *PIK3CA* H1047R were noted in two cases.

Discussion

M-AME is a rare disease, and its clinical course is not fully clarified. Ahmed and Heller (17) indicated that the hematogenous spread seems to be more frequent than the lymphatic spread, with the lung and brain metastasis. The tumor diameter with distant metastasis was 2 cm or larger, as noted in the present case. Genetic analysis of matched classic and malignant components of M-AME has suggested that it derives from malignant transformation of a pre-existing classic AME (7). Malignancy of M-AME arise from either luminal epithelial or myoepithelial components, or from both cell types (5). In the present case, myoepithelial cells revealed cytological atypia and mitoses, and the pulmonary metastatic lesion indicated myoepithelial proliferation. Myoepithelial cells seemed to be a malignant component. Although genetic data on M-AME are limited, and *PIK3CA* and *HRAS* Q61R hotspot mutations are noted in both AME and M-AME (5,7). *HRAS* mutations are rare in common-type breast cancers (6). The majority of the invasive breast cancers arising in AME display a triple-negative phenotype (ER-, PgR-, HER2-negative) (5), and the *HRAS* Q61R hotspot mutation of AME and M-AME, which is related to ER-negative cases (7). *In vitro* analyses demonstrated that forced expression of mutant *HRAS* Q61R on non-malignant and ER-negative breast epithelial cells, resulted in high grade malignancy (increased proliferation and migration), and oncogenic properties and acquisition of a myoepithelial-like phenotype (7). *HRAS* mutations are a tumorigenic gene alteration noted in salivary gland EMC (11). AME of the breast is identical in histological and immunohistochemical structure to EMC of the salivary gland (18), and the most frequent *HRAS* mutation in the salivary gland EMC is Q61R(82.1%), as noted in breast AME. Other mutations noted were the following: G13R(9.0%), Q61K(7.5%) and Q61(1.5%). Among salivary gland tumors, *HRAS* mutation is 100% specific to EMC and not to any other histological type, and the mutation is useful for the diagnosis of salivary gland EMC (11). In addition, in breast tissues, *HRAS* mutation may be useful in the diagnosis of AME and M-AME.

The present review indicated that the *HRAS* G13R was most frequently noted (4 cases, 25%) in M-AME, and 2 cases harbored mutations in both *HRAS* G13R and *PIK3CA* H1047R. Although *HRAS* Q61 mutation was seen in 4 M-AME cases without clinical findings, the *HRAS* G13R mutation was not seen in AME (7). For distinguishing whether AME is benign or malignant, H&E stain is the most important. In the present case, frequent mitoses, cytological atypia and necrosis of breast myoepithelial cells were recognized, and the diagnosis of M-AME was made. In addition to H&E stain, *HRAS* G13R mutation may be an adjunctive indicator of malignancy. Further

case accumulations of M-AME are required. *HRAS* mutation causes activation of the RAS-MAPK pathway, and *MEK* is a downstream effector of *HRAS* in the MAPK pathway. A recent study has shown that tumors with *HRAS* G13R mutations may be responsive to MEK inhibitors such as trametinib. In metastasis-derived breast M-AME xenografts with *HRAS* mutations (*HRAS* G13R or G125), the trametinib treatment resulted in a marked anti-tumor activity (16). Agminated Spitz nevus with *HRAS* G13R indicated optimal response to trametinib (19). From these findings, it can be deduced that the *HRAS* protein may be a potential target, and the genotypic analysis is important for the treatment selection of M-AME.

The presented patient is followed up regularly, without medication, and exhibits no evidence of disease one year following the lung operation. The patient underwent total mastectomy, sentinel lymph node dissection and adjuvant chemotherapy, but lung metastasis was seen about two years later. Although most M-AME are cured with wide excision with negative margins, local recurrence or hematogenous metastasis is demonstrated (17). The 5-year overall survival of M-AME is as high as 87.5% during median follow-up of 55 months, and it has a relatively good prognosis (20), but distant metastasis and local recurrence tend to occur 4 months to 2-3 years after the first diagnosis (21), as seen in the present case. From these, the early detection of recurrence seems to improve the prognosis, and regular scanning using computed tomography and tumor marker measurements are planned in the current case. M-AME has a high rate of triple negative subtype (5), as seen in the presented case, and endocrine and anti-HER2 therapy is not indicated. Although there is a little objective evidence of radiotherapy or conventional chemotherapy (20), MEK inhibitor may be a candidate in case of recurrence. Genotypic analysis of M-AME is useful for making the diagnosis and selecting a medicine.

In conclusion, a case of *HRAS* G13R mutated M-AME with lung metastasis was reported. Review analyses indicated that *HRAS* G13R was the popular mutation of M-AME, which may be considered a driver mutation in M-AME.

Acknowledgements

Not applicable.

Funding

No funding was received.

Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

HK and RT interpreted and analyzed the data, and drafted the manuscript. RT, YU, TN, YH and KK acquired the data and revised the manuscript. MI designed the study, conducted the treatment and revised the manuscript. MI and TN confirm the authenticity of all the raw data. All authors read and approved the final version of the manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

The patient provided written informed consent for the publication of their data and images.

Competing interests

The authors declare that they have no competing interests.

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