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Case Report

Synchronous Occurrence of Secretory Carcinoma of the Breast and Squamous Cell Carcinoma of the Buccal Mucosa: A Rare Case Report

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ABSTRACT

Background: Multiple malignancies in the same patient account for 2% to 17% of all cancers. However, multiple synchronous primary tumours in the same patient are extremely rare. The term synchronous is used in oncology to refer to two (or more) independent primary malignancies, when the second malignancy arose within six months of the diagnosis of the first malignancy. Comparatively very less information is available on synchronous presentation of dual malignancies in the literature than individual tumours as such.

About the case: A 58-year-old woman was clinically suspected as breast carcinoma and Squamous cell carcinoma of buccal mucosa in under two months, underwent modified radical mastectomy and mandibulectomy. Histopathology reports suggested Secretory variant of Breast Carcinoma and Squamous cell Carcinoma of Buccal mucosa.

Conclusion: Here we present an unusual combination of Secretory variant of Breast Carcinoma and Squamous cell Carcinoma of Buccal mucosa in synchronous presentation. To the best of our knowledge and based on the currently available literature, this combination has not been previously reported.

Keywords: *Cancers, Synchronous Tumours, Secretory Breast Carcinoma, Next Generation Sequencing, ETV6 NTRK3 fusion, Squamous Cell Carcinoma buccal mucosa, paclitaxel and carboplatin, Malignancy, Metastasis*

INTRODUCTION

As per the reports of the International Agency For Research On Cancer (IARC) 2026, globally one in five people develops cancer in their lifetime. In 2025, there were approximately 20 million new cancer cases with over 10 million deaths world-wide.(1) Multiple primary malignancies are becoming common in cancer patients, with risk factors such as genetics, viral infection, smoking, betel nut chewing, and environmental or treatment-related factors. These multiple malignancies may be synchronous if they get detected within 6 months duration of the first primary tumour. The frequency of multiple primary tumours as synchronous tumours is rare though metachronous tumours appear to be common these days. The exact statistical data on the prevalence of synchronous tumours is not available and the existing data in publications worldwide is based on a study in 2010 by A. Irimie, etal.(2) But a study from AIIMS Jodhpur by Nishtha Choudhary etal in 2025 on 51 cases of dual primary cancers showed that 12 of them were synchronous tumours amounting to 23.53% of dual tumour population in individuals.(3) With the availability of high-end technologically sophisticated diagnostic and imaging facilities, the rate of detection of multiple primary cancers is also increasing. We are reporting an extremely rare set of synchronous tumours, a Secretory type of Breast carcinoma which in itself is a very rare variant (0.15%) among all breast cancers existing with a Squamous Cell Carcinoma of the buccal mucosa. With the available literature, there is no evidence of coexistence of both these tumours in a single patient till date.

ABOUT THE CASE

A 58 years old female presented in the month of December 2025 to the surgery OP of SVRRGGH Tirupathi with a painless lump in the right breast. Core biopsy was reported on 8th Dec 2025 as Duct cell Carcinoma Breast.

Figure 1. Clinical picture of the Right breast mass

Figure 2 Clinical picture of the Nonhealing ulcer in buccal mucosa

She has a history of betel nut chewing and history of injury to buccal mucosa on the left side due to sharp teeth and she developed an ulcer on her left buccal mucosa which was not responding to symptomatic treatments and punch biopsy was conducted and reported on 27th Dec 2025 as Moderately differentiated Squamous cell carcinoma.

Later she was registered as an inpatient and investigated as per the standard operating procedures and surgery was done for both the lesions on 20th January 2026 as per her convenience. Modified Radical Mastectomy was done for the right breast and Left Mandibulectomy was conducted simultaneously in one sitting. Both the specimens were sent to the Department of Pathology, S.V. Medical College, Tirupathi, for Histopathological evaluation.

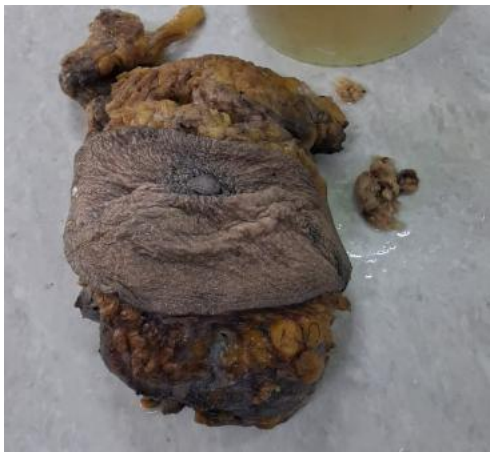


Figure 3. Gross specimen of breast

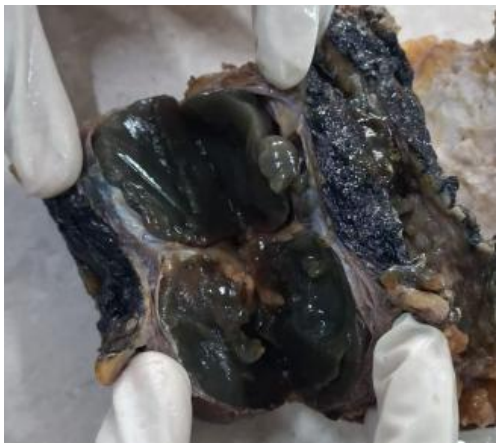


Figure 4. Cross section of breast specimen showing mucin

Grossly breast lesion was measuring 9.5 x 7 x 4cms. Cut surface showed massive areas of mucin. After due processing it was reported as Secretory carcinoma (Grade 2 CAP PROTOCOL) by Histopathology examination which is a very rare entity under breast cancers.

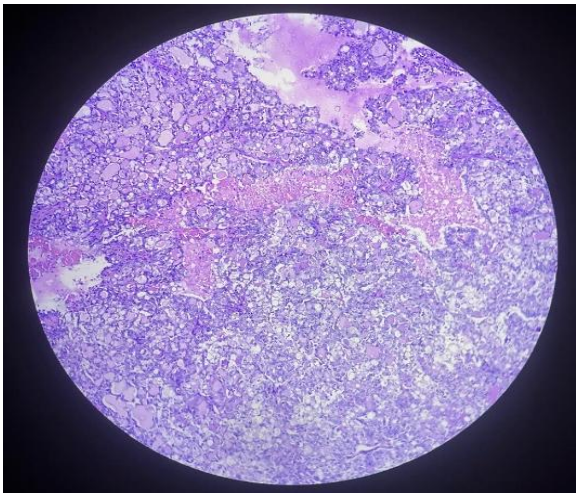


Figure 5. Low power 10x

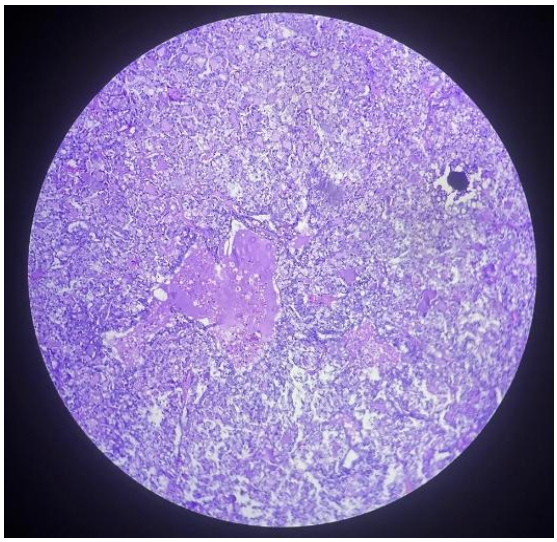


Figure 6. Low power 10x

Figure 5 & 6 Atypical ductal epithelial cells arranged as follicles with secretions mimicking thyroid tissue

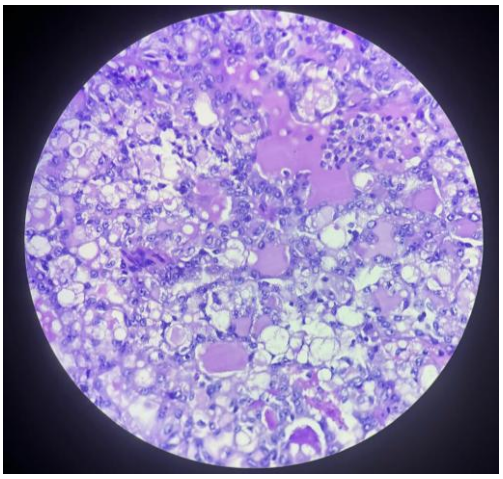


Figure 7 High power 40x

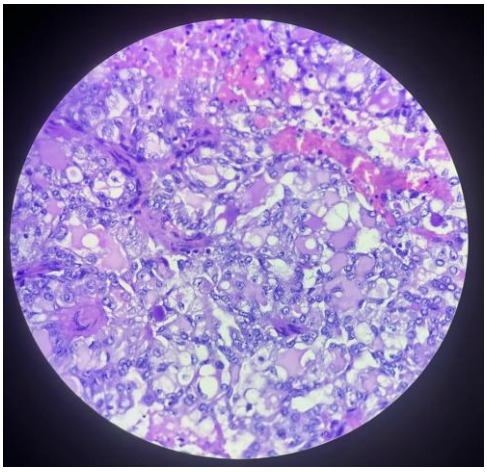


Figure 8 High power 40x

Figure 7 & 8 Tumour cells are round to oval with mild pleomorphism and vesicular nuclei and eosinophilic cytoplasm

On Immunohistochemical Examination, ER positivity was seen in 10% of tumour cells while PR and HER2 were negative.15% of Tumour cells showed positivity for KI67.

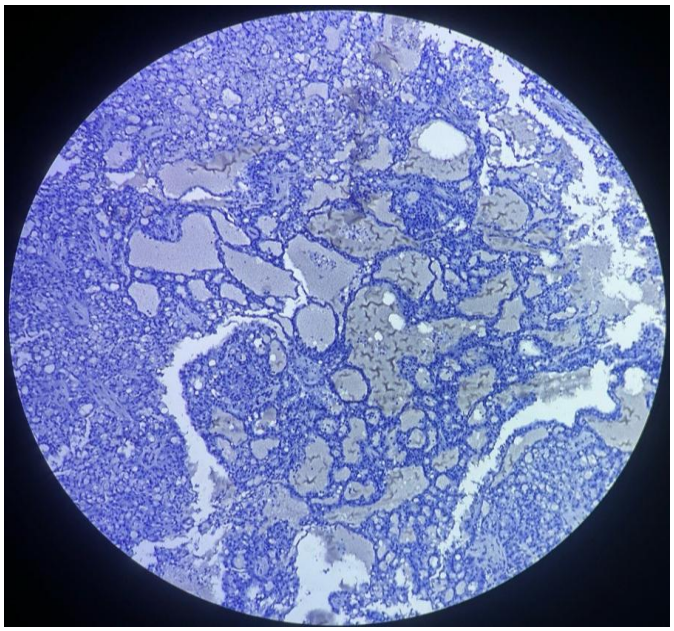


Figure 9 10% of Tumour cells showing positivity for ER indicating moderate positivity

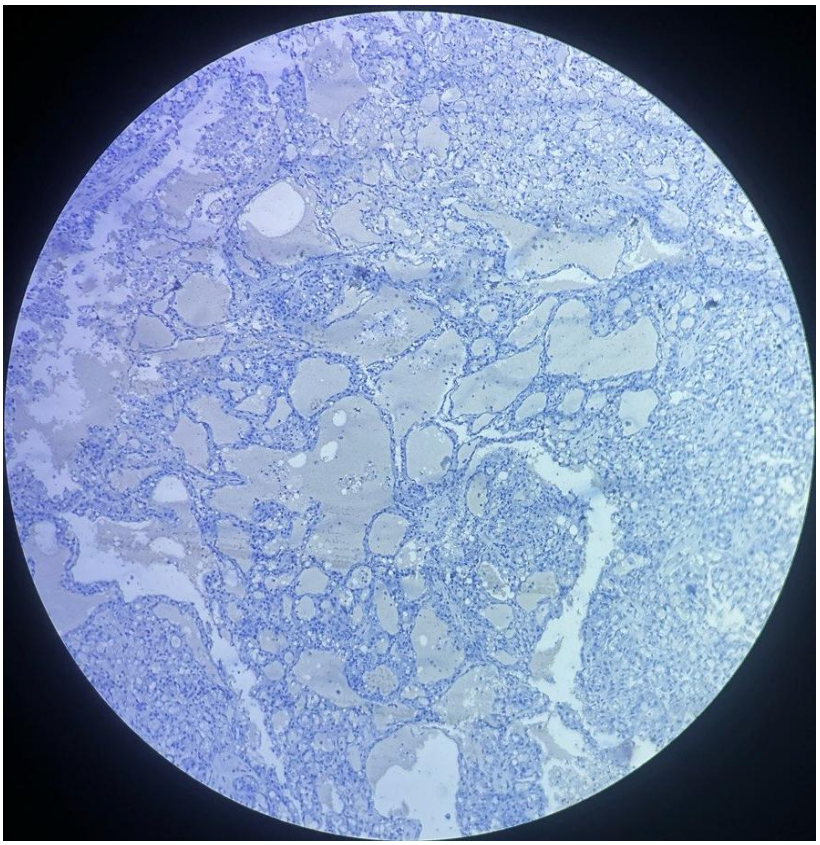


Figure 10 *Tumour cells negative for PR*

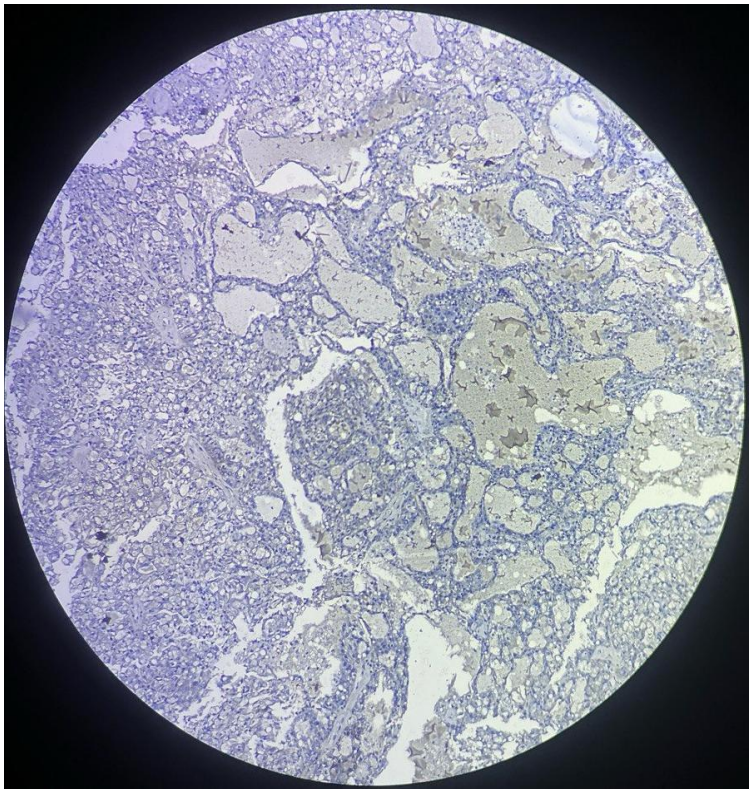


Figure 11 *Tumour cells negative for HER2*

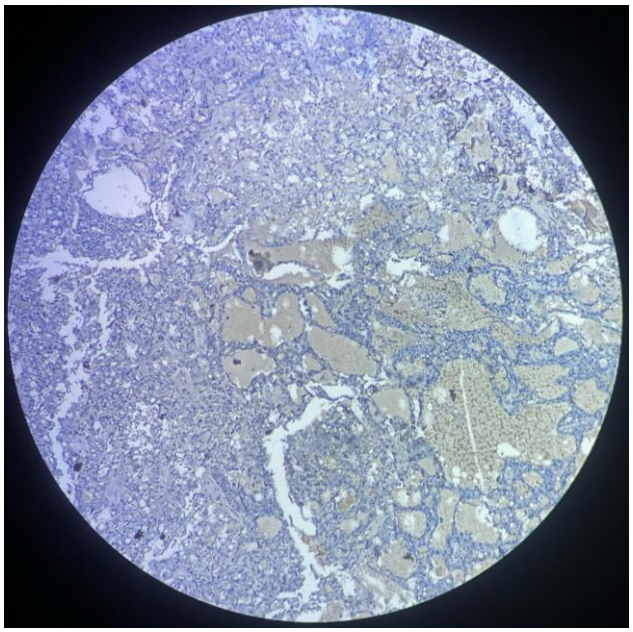


Figure 12. 15% of *Tumour cells positive for KI67*

Grossly buccal mucosa lesion/left mandibulectomy specimen was measuring 3 x 2 x 1cms. It was reported as Well Differentiated Squamous cell carcinoma (Grade 1 CAP PROTOCOL) on histopathology.

Figure 13. Gross Specimen of Mandibulectomy

Figure 14. Cross section of Mandibulectomy specimen

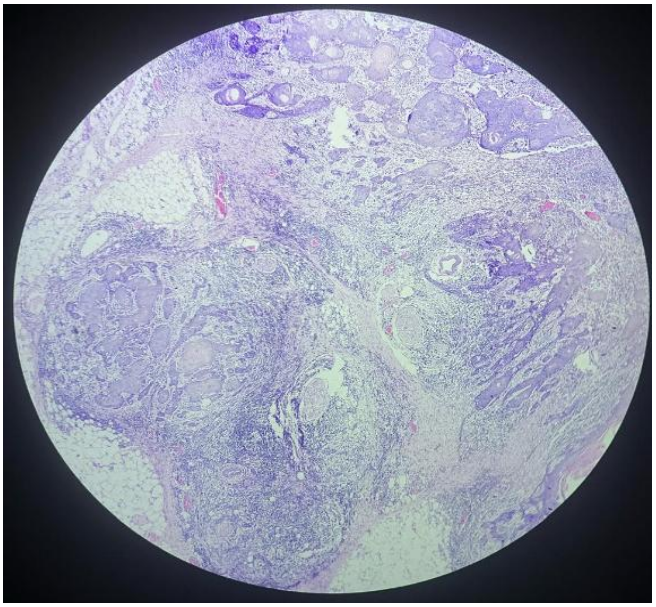


Figure 15 Scanner view 4x

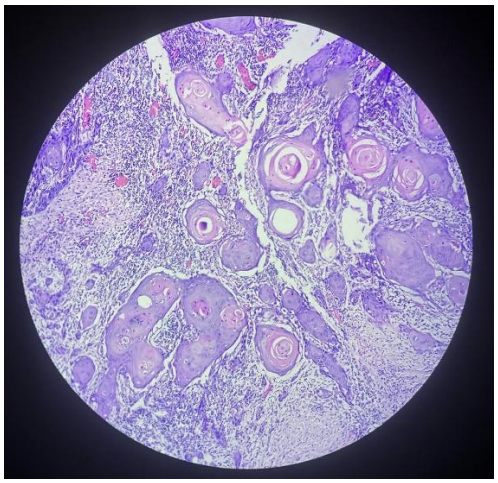


Figure 16 Low Power 10x

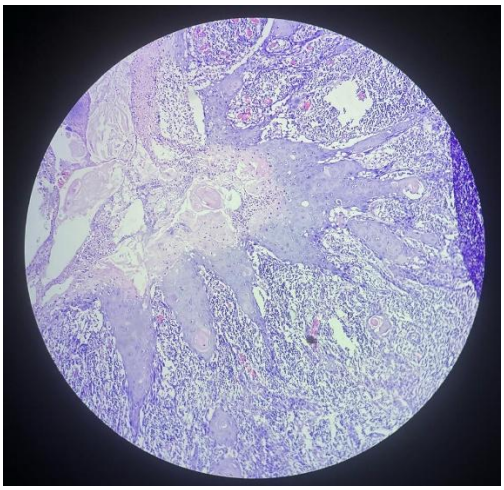


Figure 17 Low power 10x

Figures 15,16 & 17 Keratinising stratified squamous epithelium with dysplastic changes transcending into the stroma, arranged as sheets nests and clusters with intercellular bridging with focal keratin pearl formation.

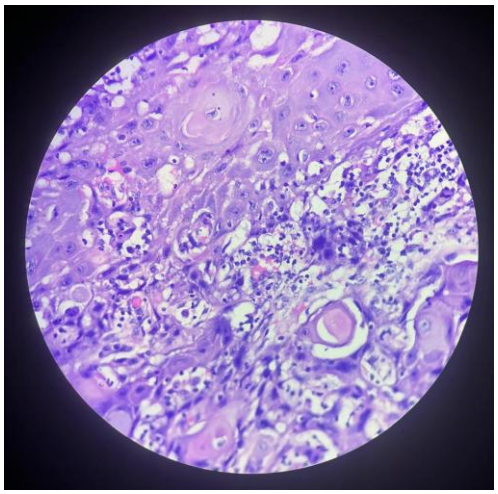
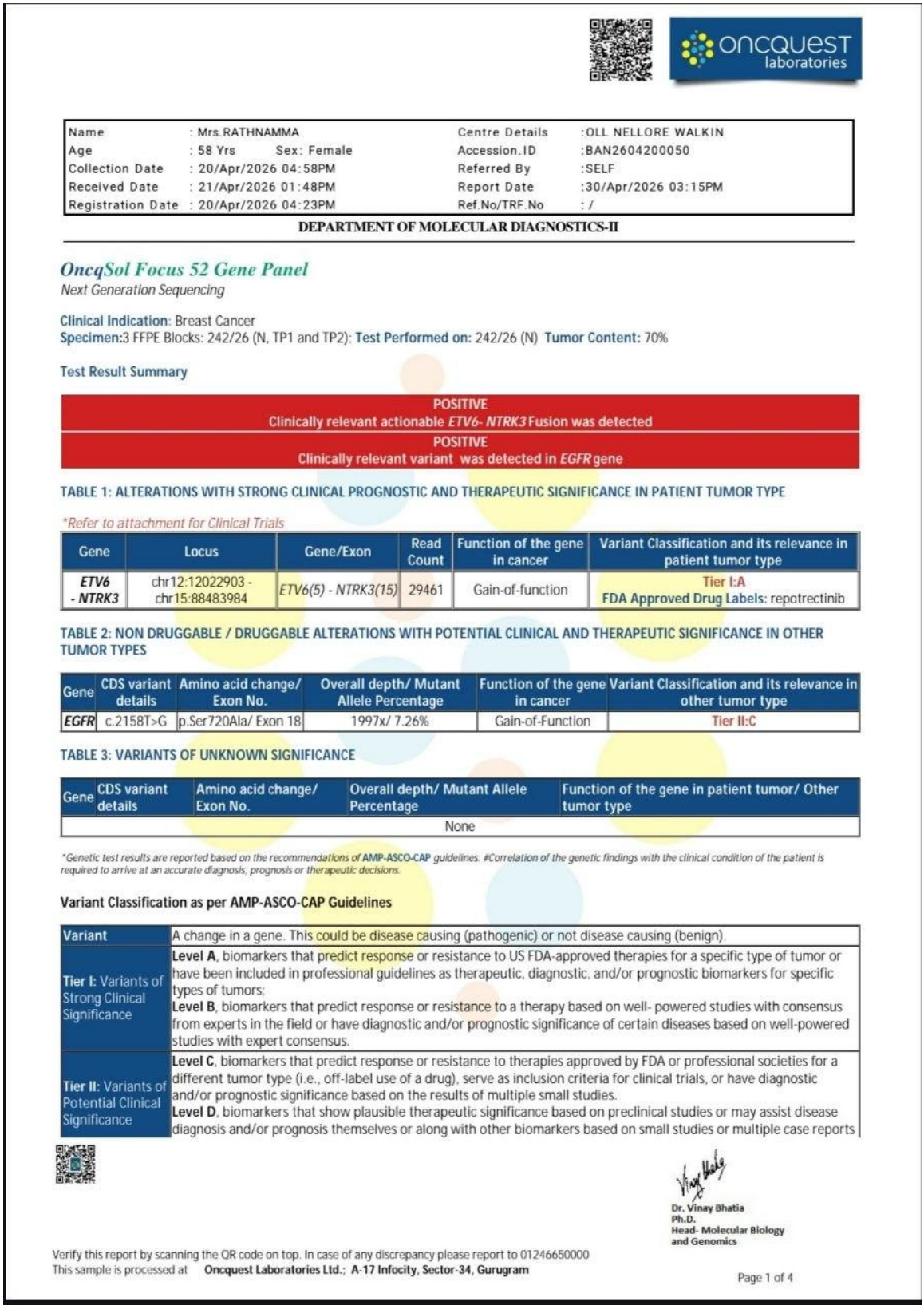


Figure18 High Power 40x

Figure 18 *Tumour cells are large round to polygonal with increased N:C ratio, prominent nucleoli and scant eosinophilic cytoplasm*

Further molecular evaluation using next-generation sequencing, performed at Onquest Laboratories, New Delhi, identified the presence of an **ETV6–NTRK3 fusion gene**, specifically involving exon 5 of *ETV6* and exon 15 of *NTRK3*. This gain-of-function fusion protein is characteristic of secretory breast carcinoma and supports the histopathological diagnosis.



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Name	: Mrs.RATHNAMMA	Centre Details	:OLL NELLORE WALKIN
Age	: 58 Yrs Sex: Female	Accession.ID	:BAN2604200050
Collection Date	: 20/Apr/2026 04:58PM	Referred By	:SELF
Received Date	: 21/Apr/2026 01:48PM	Report Date	:30/Apr/2026 03:15PM
Registration Date	: 20/Apr/2026 04:23PM	Ref.No/TRF.No	: /

DEPARTMENT OF MOLECULAR DIAGNOSTICS-II

OncqSol Focus 52 Gene Panel

Next Generation Sequencing

Clinical Indication: Breast Cancer
Specimen:3 FFPE Blocks: 242/26 (N, TP1 and TP2); **Test Performed on:** 242/26 (N) **Tumor Content:** 70%

Test Result Summary

POSITIVE
Clinically relevant actionable *ETV6- NTRK3* Fusion was detected

POSITIVE
Clinically relevant variant was detected in *EGFR* gene

TABLE 1: ALTERATIONS WITH STRONG CLINICAL PROGNOSTIC AND THERAPEUTIC SIGNIFICANCE IN PATIENT TUMOR TYPE

**Refer to attachment for Clinical Trials*

Gene	Locus	Gene/Exon	Read Count	Function of the gene in cancer	Variant Classification and its relevance in patient tumor type
ETV6 - NTRK3	chr12:12022903 - chr15:88483984	*ETV6(5) - NTRK3(15)*	29461	Gain-of-function	Tier I:A **FDA Approved Drug Labels:** repotrectinib

TABLE 2: NON DRUGGABLE / DRUGGABLE ALTERATIONS WITH POTENTIAL CLINICAL AND THERAPEUTIC SIGNIFICANCE IN OTHER TUMOR TYPES

Gene	CDS variant details	Amino acid change/ Exon No.	Overall depth/ Mutant Allele Percentage	Function of the gene in cancer	Variant Classification and its relevance in other tumor type
EGFR	c.2158T>G	p.Ser720Ala/ Exon 18	1997x/ 7.26%	Gain-of-Function	Tier II:C

TABLE 3: VARIANTS OF UNKNOWN SIGNIFICANCE

Gene	CDS variant details	Amino acid change/ Exon No.	Overall depth/ Mutant Allele Percentage	Function of the gene in patient tumor/ Other tumor type
None				

**Genetic test results are reported based on the recommendations of AMP-ASCO-CAP guidelines. #Correlation of the genetic findings with the clinical condition of the patient is required to arrive at an accurate diagnosis, prognosis or therapeutic decisions.*

Variant Classification as per AMP-ASCO-CAP Guidelines

Variant	A change in a gene. This could be disease causing (pathogenic) or not disease causing (benign).
Tier I: Variants of Strong Clinical Significance	**Level A**, biomarkers that predict response or resistance to US FDA-approved therapies for a specific type of tumor or have been included in professional guidelines as therapeutic, diagnostic, and/or prognostic biomarkers for specific types of tumors; **Level B**, biomarkers that predict response or resistance to a therapy based on well- powered studies with consensus from experts in the field or have diagnostic and/or prognostic significance of certain diseases based on well-powered studies with expert consensus.
Tier II: Variants of Potential Clinical Significance	**Level C**, biomarkers that predict response or resistance to therapies approved by FDA or professional societies for a different tumor type (i.e., off-label use of a drug), serve as inclusion criteria for clinical trials, or have diagnostic and/or prognostic significance based on the results of multiple small studies. **Level D**, biomarkers that show plausible therapeutic significance based on preclinical studies or may assist disease diagnosis and/or prognosis themselves or along with other biomarkers based on small studies or multiple case reports

Dr. Vinay Bhatia
Ph.D.
Head- Molecular Biology and Genomics

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Sample Cancer Type: Breast Cancer

Relevant Breast Cancer Findings

Gene	Finding	Gene	Finding
AKT1	None detected	FGFR3	None detected
BRAF	None detected	NTRK1	None detected
ERBB2	None detected	NTRK2	None detected
ESR1	None detected	NTRK3	***ETV6::NTRK3 fusion***
FGFR1	None detected	PIK3CA	None detected
FGFR2	None detected	RET	None detected

Relevant Biomarkers

Genomic Alteration	Relevant Therapies (In this cancer type)	Relevant Therapies (In other cancer type)	Clinical Trials
ETV6::NTRK3 fusion	**repotrectinib** [1] entrectinib larotrectinib	entrectinib larotrectinib repotrectinib	4
EGFR S720A Allele Frequency: 7.26%	None*	osimertinib	0

*** Public data sources included in relevant therapies:** NCCN, ESMO, **FDA**[1]

Biomarker Descriptions

EGFR S720A

epidermal growth factor receptor

Background: The EGFR gene encodes the epidermal growth factor receptor (EGFR), a member of the ERBB/human epidermal growth factor receptor (HER) tyrosine kinase family[1]. In addition to EGFR/ERBB1/HER1, other members of the ERBB/HER family include ERBB2/HER2, ERBB3/HER3, and ERBB4/HER4[2]. EGFR ligand-induced dimerization results in kinase activation and leads to stimulation of oncogenic signaling pathways, including the PI3K/AKT/MTOR and RAS/RAF/MEK/ERK pathways[3]. Activation of these pathways promotes cell proliferation, differentiation, and survival[4,5].

Alterations and prevalence: Recurrent somatic mutations in the tyrosine kinase domain (TKD) of EGFR are observed in approximately 10-20% of lung adenocarcinoma, and at higher frequencies in never-smoker, female, and Asian populations[6,7,8,9]. The most common mutations occur near the ATP-binding pocket of the TKD and include short in-frame deletions in exon 19 (EGFR exon 19 deletion) and the L858R amino acid substitution in exon 21[10]. These mutations constitutively activate EGFR resulting in downstream signaling, and represent 80% of the EGFR mutations observed in lung cancer[10]. A second group of less prevalent activating mutations includes E709K, G719X, S768I, L861Q, and short in-frame insertion mutations in exon 20[11,12,13,14]. EGFR activating mutations in lung cancer tend to be mutually exclusive to KRAS activating mutations[15]. In contrast, a different set of recurrent activating EGFR mutations in the extracellular domain includes R108K, A289V and G598V and are primarily observed in glioblastoma[10,16]. Amplification of EGFR is observed in several cancer types including 44% of glioblastoma multiforme, 12% of esophageal adenocarcinoma, 10% of head and neck squamous cell carcinoma, 8% of brain lower grade glioma, 6% of lung squamous cell carcinoma, 5% of bladder urothelial carcinoma cancer, lung adenocarcinoma, and stomach adenocarcinoma, 3% of cholangiocarcinoma, and 2% of cervical squamous cell carcinoma,

Disclaimer: The data presented here are from a curated knowledge base of publicly available information, but may not be exhaustive. The data version is 2026.04(006). The content of this report has not been evaluated or approved by the FDA, EMA or other regulatory agencies.

DISCUSSION:

By definition synchronous tumours are those tumours occurring simultaneously or within 6 months of the diagnosis of the first primary tumour [(4)]. Billroth was the first person to introduce Multiple Primary Malignant Tumours (MPMTs) into the literature. [(5)]

Subsequently, Warren and Gates in their research in 1932 set-up certain criteria [(6)] which should be fulfilled before stating that multiple co-occurring neoplasia are MPMTs.

1. Both tumours should be histopathologically dissimilar.
2. The possibility of co-occurrence is due to metastases should be excluded.

3. Vivid characteristics of malignant transformation should be present.

Our case fully satisfies these criteria.
Later Gluckman defined "Synchronous malignancy" as any malignant tumours either presenting simultaneously or within the first 6 months of the time of diagnosis of the first primary malignancy. Any time beyond those 6 months milestone, those tumours would be considered "Metachronous malignancy" [7].

In our case both the tumours were diagnosed incidentally at the same time upon our patient's clinical presentation.

Based on the sites of occurrences, tumours can be categorized into four different types [8].

1. The multicentric type, if two histopathological different malignancies originate from the same organ.
2. The systemic type if both the malignancies originate from functionally or anatomically related organ tissues of the same organ system (i.e., rectal and colon neoplasia).
3. The paired organ neoplasia, as in the bilateral breast tissues.
4. The random type if the distinct malignancies are found incidentally in functionally and anatomically unrelated organs.

Our case is of the random type (4th type) because the Secretary carcinoma is in breast while the Squamous cell carcinoma originated in the buccal mucosa, two unrelated organ sites and histopathologically entirely indistinct from one another but arising almost in the same period of time.

The right breast showed Secretary Carcinoma which is a very rare variant whose incidence is less than 0.15% of all breast cancers [9] and the buccal mucosa showed Well differentiated squamous cell carcinoma whose incidence is 31.7% in all the oral cancers. [10]

Secretory Breast Carcinoma is usually negative for estrogen and progesterone receptor and negative HER-2 (triple-negative breast cancer). Yet it has a good prognosis.[11] Lijaun Li *et al* reported 21 ER-positive cases out of 44 total cases of SBC.[12] Our case is also of intermediate ER positivity with 10% of cells with nuclear positivity and 15% of cells positive for KI67. On the other hand the squamous cell carcinoma of the buccal mucosa has a poor prognosis [13].

On Histopathology, Secretory carcinoma of breast is a circumscribed lesion with pushing borders but may be infiltrative at periphery with microcystic, solid or tubular or admixture of all 3 patterns or sometimes can display peripheral papillary architecture with a Low grade cytologic atypia, bland uniform nuclei, low mitotic rate with vacuolated, foamy cytoplasm and abundant intracellular and extracellular pale blue to dense pink secretions, which are periodic acid Schiff (PAS) positive and diastase resistant.

On the other hand, Well differentiated squamous cell carcinoma has an entirely different histopathological picture. It appears as dysplastic stratified squamous epithelium extending through the basement membrane into the underlying fibrous connective tissue and is arranged as islands, nests, cords or dispersed individual cells without attachment to the surface. The malignant epithelial cells show hyperchromatic nuclei, pleomorphism, mitotic activity, individual cell keratinization, intercellular bridging and eosinophilic cytoplasm. Round, eosinophilic, concentric layers of keratin fancily called Keratin pearls are a feature of well differentiated squamous cell carcinoma.

Genetics: Apart from the regular etiological risk factors for all cancers, the pathogenesis of Secretory carcinoma breast is attributed to a specific genetic alteration, a t(12;15)(p13;q25) translocation resulting in an ETV6-NTRK3 fusion gene in both the in situ and invasive components [14,15,16]. By RNA analysis, the most frequent fusion breakpoints are between exon 5 of *ETV6* and exon 15 of *NTRK3*[17]. The pathogenesis of squamous cell carcinoma of buccal mucosa is multifactorial with accumulation of genetic alterations such as Loss of function of *TP53* mutations and *CDKN2A* inactivation.[18]

While breast cancer as primary tumour is the most common source of metastasis to the oral cavity in women, metastasis to the buccal mucosa is uncommon.[19] This present case of Buccal mucosal Squamous cell Carcinoma cannot be considered at any stage as a metastasis from the secretory breast carcinoma as both are morphologically different variants. With the available literature, there is no evidence of coexistence of both these tumours in a single patient.

Treatment: The increased incidence of multiple malignant tumours is a real challenge to the clinician and clinical attention should be made to avoid a misdiagnosis. Synchronous cancers that required no modification of the treatment, the prognosis was better and the survival rate was 28%[20]. Treatment modality should be carefully made and tailored on the individual patient suffering from this disease[21]. Despite favourable Disease Specific Survival outcomes in early-stage oropharyngeal and hypopharyngeal cancers, Overall Survival remains compromised due to secondary malignancies[22].

Secretory Ca of Breast being a very rare tumour in spite of being Triple negative phenotype yet carries good prognosis[23]. It is a relatively indolent tumour with an excellent overall prognosis. Despite a 30% incidence of axillary lymph node metastases, the 5-year survival rate is almost 100%. Surgery, whether conservative or radical, is the preferred treatment. The prognosis remains good. Simple mastectomy cures in most cases and axillary dissection to be done in LN+ on imaging or clinical positive nodes. The benefits of chemotherapy and radiotherapy remain debatable. Management is not codified, given the rarity of this subtype, so further studies are needed in order to establish appropriate management [22].

As our case has synchronous Squamous cell Carcinoma of oral cavity, this poses a complex scenario to navigate. Firstly, MRM breast surgery followed by Mandibulectomy was done. Neoadjuvant chemo with 6 cycles of Pacli Carbo was suggested for both breast and oral cavity lesions. Opinion for Radiation is required for both the tumours.

CONCLUSION:

The occurrences of synchronous tumours is an alarming situation to the patients and their attendants and must drive the medical fraternity into finding new regimens and methods in treating these cases without any significant side effects or toxicity to the patients while treating both the tumours simultaneously. To the best of our knowledge and based on the currently available literature, this combination has not been previously reported. This rarity emphasizes the importance of thorough clinical evaluation and a high index of suspicion for additional primary malignancies.

The motive behind presenting this case report is only to bring to light that a very rare tumour secretory carcinoma of breast is coexisting with squamous cell carcinoma of the buccal mucosa. With the available literature, there is no evidence of these two tumours coexisting in a single patient and probably we are the first to report this combination. This poses a challenge for the treating clinician in planning the best treatment. Hence, more case studies must be reported in the future to evaluate the association between secretory carcinoma of breast and squamous cell carcinoma of the buccal mucosa to develop proper treatment protocols.

Financial support and sponsorship: Nil.

Conflicts of interest: There are no conflicts of interest.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Ethics statement by all authors

As this is a case report without identifiers, approval from Institutional Review Board is not required.

Data Access Statement: All relevant data are within the paper and its supporting information files.

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Onquest Laboratories New Delhi

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