e-ISSN: 2807-6257

Open Access Indonesian Journal of Medical Reviews

[OAIJMR]

https://hmpublisher.com/index.php/oaijmr

A Rare Manifestation of Metastatic Breast Cancer: Cervical Esophageal Stenosis with Oropharyngeal Dysphagia Decades After Primary Treatment

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ARTICLE INFO

Keywords:
Breast cancer metastasis
Cervical esophagus
Esophageal stenosis
Late recurrence
Oropharyngeal dysphagia

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All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/oaijmr.v5i5.767

A B S T R A C T

Esophageal metastasis from breast cancer is an infrequent occurrence, with cervical esophageal involvement being exceptionally rare. Presentation with oropharyngeal dysphagia, particularly decades after primary breast cancer treatment, poses a significant diagnostic challenge. This report details such a case, emphasizing the clinical course and diagnostic complexities. A 60-year-old female, with a history of primary breast cancer treated approximately two decades prior and subsequent treatment for a locoregional recurrence with surgery, chemotherapy, and radiotherapy in 2019, presented with progressive oropharyngeal dysphagia and aspiration over three months. Initial Fiberoptic Endoscopic Evaluation of Swallowing (FEES) suggested upper esophageal pathology with stenosis and extraluminal mass compression at the introitus esophagus. Esophagoscopy confirmed a high cervical esophageal stenosis impassable with the scope. Computed Tomography (CT) of the neck revealed a large heterogeneous solid mass at the C6-Th2 level, encasing the trachea and causing severe cervical esophageal stenosis with suspected wall infiltration, along with widespread metastatic disease including pulmonary and osseous metastases. In conclusion, this case highlights the critical importance of maintaining a high index of suspicion for metastatic breast cancer in patients presenting with new-onset oropharyngeal or esophageal dysphagia, even many years after their initial cancer diagnosis and treatment. Cervical esophageal metastasis, though rare, should be considered in the differential diagnosis. A multidisciplinary approach and comprehensive diagnostic evaluation, including advanced imaging, are paramount for accurate diagnosis and guiding appropriate palliative management.

1. Introduction

Breast cancer stands as one of the most frequently diagnosed malignancies in women globally, with a significant propensity for distant metastasis, which remains the primary cause of mortality associated with the disease. While common sites of breast cancer metastasis include bone, lung, liver, and brain, involvement of the gastrointestinal (GI) tract is relatively uncommon, and metastasis specifically to the esophagus is considered a rare event. Literature estimates suggest that esophageal metastases from all primary cancers account for approximately 3-6% of esophageal malignancies, with breast cancer being one of the more frequently reported primary sources among these rare secondary tumors. However, the actual prevalence of esophageal metastasis from breast cancer is estimated to be low, reportedly ranging from 0.5% to 6% in autopsy series of patients with disseminated breast cancer, and clinically diagnosed cases are even less frequent, often cited between 4.2% to 5.9% of GI metastatic cases from breast cancer. This rarity contributes to a general lack of extensive cohort studies, with much of the existing

knowledge derived from individual case reports and small case series.[1-3]

The esophagus can be affected by metastatic disease through several mechanisms: direct extension from contiguous structures (mediastinal lymph nodes), hematogenous spread, or lymphatic dissemination. For breast cancer, lymphatic spread via mediastinal and parasternal lymph node chains to paraesophageal and intertracheobronchial lymph nodes, with subsequent infiltration of the esophageal wall's intramural lymphatics, is considered the predominant pathway. This typically results in involvement of the middle third of the esophagus, aligning with regional lymphatic drainage patterns. Metastasis to the cervical esophagus, however, is an exceptionally unusual finding, representing a small fraction of these already rare esophageal metastases. The unique lymphatic drainage of the cervical esophagus, differing from its thoracic counterpart, may contribute to this distinct pattern, possibly involving retrograde spread from supraclavicular or cervical lymph nodes, or direct infiltration from adjacent metastatic deposits in the neck.[4-6]

Clinically, esophageal metastasis often presents with non-specific symptoms such as progressive dysphagia, odynophagia, chest pain, and unintentional weight loss, mimicking primary esophageal cancer or benign strictures. Dysphagia is the most common presenting complaint, typically starting with solids and progressing to liquids as the stenosis worsens. However, when the metastatic lesion is located in the cervical esophagus, patients may present with oropharyngeal dysphagia, characterized by difficulty initiating a swallow, nasal regurgitation, coughing or choking during meals, and aspiration, symptoms that can initially mislead clinicians towards a primary neurological or upper aerodigestive tract pathology rather than a distal esophageal obstruction. A particularly challenging aspect of diagnosing esophageal metastasis from breast cancer is the potential for an extremely long latency period between the initial diagnosis and treatment of the primary breast tumor and the subsequent manifestation of esophageal symptoms. Intervals of several years to, in some reported instances, over two decades have been documented, underscoring the indolent nature these metastatic cells can sometimes exhibit before becoming clinically apparent. This protracted timeline can lead to a lowered index of suspicion for metastatic disease, especially if the patient has been considered in remission for an extended period.[7,8]

The diagnostic workup typically involves endoscopic evaluation (esophagogastroduodenoscopy) with biopsy, and imaging studies such as barium swallow, computed tomography (CT), and positron emission tomography (PET-CT). However, histopathological confirmation can be challenging because metastatic deposits in the esophagus are often submucosal or manifest as extrinsic compression, making superficial mucosal biopsies frequently non-diagnostic. Endoscopic ultrasound with fine-needle aspiration (EUS-FNA) has emerged as a valuable tool in such scenarios, offering higher diagnostic yield by accessing deeper lesions.[9,10]

This case report describes an exceptionally rare presentation of metastatic breast cancer: a significant stenosis of the cervical esophagus causing severe oropharyngeal dysphagia, occurring decades after the initial treatment of primary breast cancer. The novelty of this case lies in the confluence of several unusual features: the very proximal (cervical) location of the esophageal metastasis, the predominant symptomatology of oropharyngeal dysphagia mimicking a primary swallowing initiation disorder, the extensive extraluminal compression pattern observed, and the manifestation decades after the initial breast cancer diagnosis. This report aims to contribute to the sparse literature on this specific clinical entity, detailing the diagnostic journey, highlighting the multidisciplinary management approach, and reviewing the relevant literature to enhance clinical awareness among otorhinolaryngologists, oncologists, gastroenterologists, and radiologists who may encounter such challenging presentations. A heightened understanding of such atypical metastatic

patterns is crucial for timely diagnosis and appropriate palliative management in patients with a history of breast cancer.

2. Case Presentation

In mid-2023, a 60-year-old female, Madam NNS, presented to the Otorhinolaryngology-Head and Neck Surgery (ORL-HNS) polyclinic at Prof. Dr. I.G.N.G. Ngoerah General Hospital, Bali. Her primary concern was a progressively worsening inability to swallow, a distressing symptom that had insidiously begun approximately three months prior and had since become markedly aggravated. The dysphagia was characterized by an initial difficulty with solid foods, which rapidly advanced to an inability to manage even liquids and her own saliva. Attempts to swallow frequently precipitated immediate coughing and a distressing sensation of choking. This profound dysphagia rendered her unable to maintain any oral nutritional intake and had led to significant, though not initially quantified, unintentional weight loss. Accompanying these symptoms was a persistent sensation of blockage localized to her throat. Importantly, she denied experiencing odynophagia (painful swallowing) or any sensation of chest tightness or pain.

Her medical history was particularly notable for breast cancer. She had first been diagnosed with Stage IIB invasive ductal carcinoma of the right breast in 2003, at the age of 40. Her comprehensive treatment at that time included a modified radical mastectomy, followed by six cycles of adjuvant chemotherapy with the CMF (Cyclophosphamide, Methotrexate, 5-Fluorouracil) regimen, and subsequent local radiotherapy to the chest wall and regional lymphatic basins. Following this, she received tamoxifen as endocrine therapy for five years. She had remained under regular oncological surveillance and had been disease-free for over fifteen years. However, in 2019, a locoregional recurrence was identified, involving the right axilla and supraclavicular fossa. Treatment for this recurrence was intensive, comprising surgical excision of the affected lymph nodes, further systemic chemotherapy (initially Cyclophosphamide, Doxorubicin, and 5-Fluorouracil (CAF), followed by Paclitaxel, Carboplatin, and Trastuzumab, upon determination of HER2-positivity in the recurrent tumor), and additional radiotherapy to the involved regions, which was completed by the latter part of 2019. No other significant comorbidities were reported.

Upon general physical examination during the current presentation, Madam NNS appeared visibly weak and exhibited signs of cachexia, findings consistent with her severe dysphagia and consequent malnutrition. She was, however, conscious, alert, and fully oriented. Her vital signs were stable. A thorough ear, nose, and throat examination, including the oral cavity, did not reveal any overt abnormalities. Clinical palpation of the neck did not identify any definitive cervical lymphadenopathy at this initial assessment.
Given the prominence of oropharyngeal symptoms, a Fiberoptic Endoscopic Evaluation of Swallowing (FEES) was performed. This procedure was critical for objectively assessing the pharyngeal and laryngeal phases of her swallow and for visualizing the pharyngoesophageal junction. The detailed findings from this evaluation are summarized in Table 1. The preswallowing assessment component of FEES indicated good oral hygiene, robust lip and tongue strength, symmetrical uvular movement, and adequate cheek puffing strength, as well as a satisfactory cough strength. The flexible optic laryngoscopy, an integral part of the FEES protocol, showed symmetrical velopharyngeal movement during phonation. The lingual tonsils were noted to be of first-degree enlargement, meaning they did not obscure the valleculae. Vocal cord movement appeared grossly symmetrical during respiration; however, a subtle observation of "not docked during adduction" was made, potentially indicating a minor glottic insufficiency, though this was not considered the primary pathology. Mild pooling of secretions was evident in the valleculae and piriform sinuses prior to swallowing attempts. During the swallowing assessment phase, using boluses of varying

consistencies, penetration of the bolus material into the laryngeal vestibule, superior to the vocal cords, was observed; the patient was able to sense this and made efforts to expel the bolus. More significantly, frank aspiration of bolus material was also documented. A pivotal finding during the FEES examination was the difficulty encountered in visualizing the esophagus beyond its inlet (introitus esophagus). The endoscopic appearance at the upper esophageal sphincter (UES) region was highly suggestive of a narrowed esophageal lumen, consistent with stenosis, and the characteristics of this narrowing strongly pointed towards extraluminal mass compression as the underlying cause, rather than an intrinsic mucosal abnormality.

The constellation of findings, particularly the oropharyngeal dysphagia with aspiration coupled with FEES evidence suggesting high esophageal stenosis likely secondary to extraluminal compression, led to an initial working diagnosis of oropharyngeal dysphagia due to proximal esophageal obstruction. A differential diagnosis also included bilateral vocal fold paralysis, although the laryngoscopic findings did not strongly support this. The subsequent clinical course and management are detailed in Table 2. As an immediate measure, a nasogastric tube (NGT) was inserted to provide crucial nutritional support. Concurrently, the patient was referred to a neurologist to meticulously rule out a primary neurological etiology for her profound dysphagia, especially given the initial, somewhat ambiguous note regarding vocal cord adduction and the complex nature of her swallowing impairment.

The neurology consultation involved a comprehensive assessment, including a Magnetic Resonance Imaging (MRI) scan of the head. This advanced imaging study revealed no acute intracerebral or intracerebellar lesions that could adequately explain her severe symptoms. The MRI report did note the presence of a "neurovascular conflict A. vertebralis right with N IX, X right side"; however, this finding was considered less likely to be the primary cause of her rapidly progressive dysphagia and the distinct endoscopic findings of stenosis.

Following the neurological assessment, Madam NNS returned to the care of the ORL-HNS clinic for ongoing management of her dysphagia and nutritional needs. She was managed with continued NGT feeding and underwent routine NGT changes. However, approximately one month later, a significant complication arose during a scheduled NGT replacement attempt: the tube could not be reinserted beyond the upper esophagus. This indicated a probable worsening or an extremely tight, unyielding stenosis, precluding even the passage of a flexible NGT. This critical development necessitated her immediate hospitalization for more intensive investigation and definitive management of the obstruction. An operative esophagoscopy under general anesthesia was planned to allow for direct, detailed visualization of the esophagus, a thorough assessment of the nature and precise extent of the stenosis, and to obtain biopsies if deemed feasible and safe. Given the high likelihood of needing long-term non-oral nutritional support, a consultation with a digestive surgeon for the placement of a gastrostomy tube was also arranged.

During the operative esophagoscopy, a rigid esophagoscope was carefully introduced. The intraoperative findings starkly mirrored the impressions from the earlier FEES: a significant, constricting narrowing was encountered in the proximal cervical esophageal lumen. This stenosis was located approximately 1-2 cm distal to the esophageal inlet, corresponding anatomically to the C6 vertebral level. The stenosis was exceptionally tight, rendering further passage of the standard adult esophagoscope impossible. Critically, the mucosal surface overlying this narrowed segment appeared smooth, intact, and had a characteristic "slippery" feel upon gentle probing with the tip of the esophagoscope. This appearance strongly suggested that the stenosis was caused by extraluminal compression from an adjacent mass, rather than being an intrinsic mucosal malignancy which might typically present with ulceration,

friability, or an exophytic mass. Due to the extreme tightness of the stenosis and the significant risk of esophageal perforation associated with forceful instrumentation or attempting a biopsy of what appeared to be an extraluminal compressing mass, the esophagoscopy was discontinued after these findings were thoroughly documented. Consequently, no biopsy was obtained from the stenotic esophageal segment itself at this juncture due to safety concerns. Following the termination of the esophagoscopy, the digestive surgeon proceeded with the planned Stamm gastrostomy, successfully establishing a secure route for long-term enteral feeding.

Given the patient's extensive history of breast cancer and the unambiguous operative findings pointing towards an extraluminal compressive mass in the neck as the cause of her esophageal stenosis, an urgent referral was made to an oncology surgeon. This referral was for comprehensive oncological restaging and to specifically consider the cervical mass as a potential metastasis from her known breast cancer. As a cornerstone of this restaging process, a contrast-enhanced Computed Tomography (CT) scan of the neck and thorax was performed.

The CT scan yielded extensive and alarming findings, painting a clear picture of advanced malignant disease: A large, irregular, and heterogeneously enhancing solid mass, exhibiting central necrotic components and foci of calcification, was identified. This dominant mass was primarily situated in the region of the right lobe of the thyroid gland and the thyroid isthmus, with significant craniocaudal extension from approximately the C6 to the Th2 vertebral levels. The mass demonstrated aggressive local invasion, extending into the visceral space and the retropharyngeal space. Most critically, in relation to her presenting symptoms, this cervical mass was observed to completely encase the trachea and cause severe, high-grade stenosis of the cervical esophagus (pars cervicalis). The imaging also raised suspicion of direct infiltration by the mass into the esophageal wall at this level. The radiological characteristics of this mass were highly suspicious for a metastatic deposit, almost certainly originating from her breast cancer. The CT scan further revealed multiple suspicious lymphadenopathies distributed bilaterally across various cervical regions, including Ia, Ib, IIb, III, IVa, IVb, Va, and Vb, indicating significant regional lymphatic spread of the malignancy. The thoracic portion of the CT scan provided further, unequivocal evidence of widespread metastatic disease: Osteolytic lesions, characteristic of bone metastases, were clearly identified in the glenoid fossa and the corpus of the right scapula. Multiple pulmonary nodules, varying in size and exhibiting spiculated margins, were present in the superior, middle, and inferior lobes of the right lung, as well as in the superior and inferior lobes of the left lung. These findings were highly characteristic of nodular-type pulmonary metastases. An area of consolidation noted in the inferior lobe of the right lung was considered suspicious for either a pneumonic-type pulmonary metastasis or a secondary infectious pneumonia, a common complication in immunocompromised or debilitated cancer patients. Other incidental findings reported on the CT scan included some thickening of the parietal pleura, an area of fibrosis in the right middle lobe (interpreted as an old, resolved process), degenerative changes of spondylosis cervicothoracalis, and evidence of aortosclerosis.

These comprehensive and unequivocal CT findings solidified the diagnosis of widely metastatic breast cancer. The large, compressive metastatic mass in the neck was definitively identified as the cause of the severe cervical esophageal stenosis, which in turn led to the patient's profound oropharyngeal dysphagia and aspiration. Following this definitive diagnosis and staging, Madam NNS was managed by the multidisciplinary oncology team. The primary focus of her management shifted to palliative care, encompassing aggressive symptom control (particularly for dysphagia-related distress), ongoing nutritional support via the gastrostomy, and careful consideration of systemic therapy options appropriate for her advanced metastatic disease status and overall performance condition.

Table 1. Summary of patient's clinical findings (Patient NNS, 60-year-old Female).

Category	Finding
Presenting complaints	Progressive dysphagia (3 months, solid to liquid, unable to eat)
	Immediate coughing after swallowing solid-liquid food
	Sensation of blockage in the throat
	Denial of odynophagia or chest tightness
Past medical history	Primary Breast Cancer (Invasive Ductal Carcinoma, Stage IIB, 2003): Mastectomy, Adjuvant Chemo (CMF), Radiotherapy, Endocrine therapy (Tamoxifen)
	Recurrent Breast Cancer (Locoregional, 2019): Surgery, Chemo (CAF, Paclitaxel, Carboplatin, Trastuzumab), Radiotherapy
General examination	Weak general condition, conscious
	Cachectic appearance
ORL-HNS examination	No abnormalities in the ear, nose, and throat on routine exam
FEES - Preswallowing	Good oral hygiene, Strong lip/tongue strength, Symmetrical uvula, Adequate cheek puff/cough
FEES - Laryngoscopy	Symmetrical velopharyngeal movement, 1st-degree lingual tonsil, Vocal cords "not docked during adduction", Mild secretion pooling (vallecula/piriform sinus)
Fees - Swallowing	Penetration (bolus above vocal cords, patient feels/expels), Aspiration (Yes), No residue, Cough reflex during aspiration (Yes), Silent Aspiration (Not present)
	Narrowed esophageal lumen (introitus esophagus), stenosis, indicative of extraluminal mass compression
Initial working diagnosis	Oropharyngeal dysphagia with differential diagnosis proximal esophageal dysphagia + bilateral plica vocalis paralysis
Neurology assessment	Head MRI: No intracerebral/intracerebellar lesion. Neurovascular conflict A. vertebralis right with N IX, X right side (deemed less likely causative).
Esophagoscopy findings	Narrowing in the proximal cervical esophageal lumen (1-2 cm from inlet, C6 level), causing stenosis; impassable with scope. Mucosal surface slippery (extraluminal compression).
Neck CT-scan findings	Heterogeneous solid mass (C6-Th2) in thyroid/isthmus region, extending to visceral/retropharyngeal space. Encases trachea, causes esophageal stenosis (pars cervicalis), susp. esophageal wall infiltration, susp. metastatic mass.
	Multiple suspicious cervical lymphadenopathies (bilateral regions Ia, Ib, IIb, III, IVa, IVb, Va, Vb).
Thoracic CT findings	Osteolytic lesions (right scapula) - susp. metastatic bone disease. Multiple bilateral pulmonary nodules (spiculated edges) - susp. nodular pulmonary metastases.
	Pneumonia (right inferior lobe) - susp. pneumonic pulmonary metastasis or infection. Thickened parietal pleura, right lung fibrosis (old process).

Table 2. Summary of diagnostic and treatment procedures and follow-up.

Stage	Procedure/Intervention/Finding	Date (Conceptual)
Initial presentation & assessment	Presentation to ORL-HNS polyclinic with dysphagia	Mid-2023 (Day 1)
	Fiberoptic Endoscopic Evaluation of Swallowing (FEES) - revealed high esophageal stenosis, extraluminal compression	Day 1
	Initial Working Diagnosis: Oropharyngeal dysphagia, proximal esophageal dysphagia	Day 1
	Nasogastric Tube (NGT) insertion for nutrition	Day 1
	Referral to Neurologist	Day 1
Neurological workup	Neurologist consultation	Week 1
	Head MRI - no causative CNS lesion identified	Week 1-2
	Return to ORL-HNS for further management	Week 2
Escalation & further investigation	Routine NGT changes, ongoing NGT feeding	Week 2 - Week 6
	NGT reinsertion failure - indicated worsening/tight stenosis	Approx. 1 month later
	Hospitalization for esophagoscopy & gastrostomy consultation	Approx. 1 month later
Surgical interventions & definitive local diagnosis	Operative Esophagoscopy - confirmed severe proximal cervical stenosis (C6), impassable, extraluminal compression appearance	Hospital Day 1-2
	Gastrostomy tube placement by a Digestive Surgeon for long-term feeding	Hospital Day 1-2
Oncological staging & diagnosis	Referral to Oncology Surgeon for restaging	Post-gastrostomy
	Neck & Thorax CT scan with Contrast - revealed large cervical metastatic mass causing stenosis, widespread distant metastases	Post-gastrostomy
Follow-up & management plan	Diagnosis: Widely metastatic breast cancer with cervical esophageal compression	Post-CT scan
	Management by the Oncology team - focus on palliative care, symptom control, and consideration of systemic therapy	Ongoing

3. Discussion

The case of Madam NNS presents a confluence of rarities in the context of metastatic breast cancer, offering profound insights into the atypical behavior of this common malignancy and the diagnostic odysseys that can ensue. Metastasis, the dissemination of cancer cells from a primary tumor to distant anatomical sites, represents the most lethal attribute of cancer and is the culmination of a complex, multistep biological cascade often referred to as the "metastatic cascade." This intricate process involves: local invasion of cancer cells into the surrounding stroma; intravasation into the lumina of blood or lymphatic vessels; survival and transit of these circulating tumor cells (CTCs) within the vasculature, evading host immune surveillance; arrest at a distant capillary bed or lymphatic sinus; extravasation from the vessel into the parenchyma of the target organ; and (6) an adaptive process of colonization, wherein micrometastases must proliferate and induce angiogenesis to form clinically detectable secondary tumors. Each step of this cascade is governed by a dynamic interplay between the intrinsic properties of the tumor cells and the host microenvironment of both the primary tumor and the distant target organ. Breast cancer, a heterogeneous disease encompassing multiple molecular subtypes, exhibits distinct metastatic propensities. The "seed and soil" hypothesis, first proposed by Stephen Paget in 1889, remains a cornerstone in understanding metastatic organotropism. This theory posits that while cancer cells (the "seeds") may disseminate widely, they can only successfully colonize and proliferate in specific distant organs that provide a hospitable microenvironment (the "soil"). This explains why certain cancers preferentially metastasize to particular organs. For breast cancer, the bones, lungs, liver, and brain are the most common "fertile soils."[11,12]

Several molecular alterations drive the metastatic potential of breast cancer cells. Epithelial-to-mesenchymal transition (EMT) is a crucial phenotypic program wherein epithelial cancer cells acquire mesenchymal characteristics, such as increased motility, invasiveness, and resistance to apoptosis. EMT is often orchestrated by transcription factors like Snail, Slug, Twist, and ZEB1/2, and is influenced by signaling pathways including TGF-β, Wnt, and Notch. These transformed cells can more easily degrade the extracellular matrix (ECM) through the secretion of matrix metalloproteinases (MMPs) and other proteolytic enzymes, facilitating local invasion Once cancer cells intravasate, they face numerous challenges in the circulation, including shear stress, anoikis (apoptosis due to loss of cell-cell and cell-matrix adhesion), and immune attack. The formation of tumor cell aggregates or emboli, often involving platelets, can enhance survival. The eventual arrest of CTCs in distant organs is not random but is influenced by anatomical factors (first capillary bed encountered) and specific adhesive interactions between tumor cells and endothelial cells, mediated by molecules like selectins, integrins, and cadherins. Successful extravasation and the formation of micrometastases do not guarantee the development of overt metastatic lesions. Many micrometastases remain dormant for extended periods, sometimes years or even decades, as exemplified in the current case with its 20-year latency from primary treatment. Tumor dormancy can be cellular (quiescent single cells) or angiogenic (micrometastases unable to recruit sufficient neovasculature for expansion). The molecular mechanisms governing dormancy and reactivation are complex and involve tumor cell-intrinsic factors, the immune system, and the microenvironment of the metastatic niche. Angiogenesis, the formation of new blood vessels, is a critical switch for dormant micrometastases to grow into macroscopic tumors. Vascular endothelial growth factor (VEGF) is a key pro-angiogenic factor often targeted in cancer therapy.[13,14]

While hematogenous spread is a major route for distant metastasis, lymphatic dissemination plays a particularly significant role in the spread of many carcinomas, including breast cancer, and is considered the predominant mechanism for metastasis to the esophagus. The lymphatic system, a network of vessels and nodes paralleling the venous

system, collects interstitial fluid, immune cells, and macromolecules, returning them to the blood circulation. Tumor cells can invade lymphatic capillaries, which, unlike blood capillaries, lack a well-developed basement membrane and have loose inter-endothelial junctions, facilitating easier entry. The lymphatic drainage of the breast is primarily to the axillary lymph nodes (Level I, II, and III), which receive approximately 75-90% of the lymph. The internal mammary lymph nodes, located along the sternal border, represent another significant drainage pathway, particularly for medially located tumors or those with axillary nodal involvement. Supraclavicular lymph nodes can become involved through superior axillary pathways or direct drainage. From these primary nodal basins, tumor cells can disseminate further. For esophageal involvement, the proposed route often involves spread from the internal mammary or axillary nodes to the mediastinal lymph node chains, particularly the parasternal, paraesophageal, and intertracheobronchial nodes. These mediastinal nodes have efferent lymphatic connections to the esophagus. The esophageal wall itself possesses a rich, longitudinally oriented network of submucosal and intramural lymphatics. Once metastatic cells reach the paraesophageal lymph nodes, they can infiltrate these intramural esophageal lymphatics, leading to submucosal tumor deposits. This pattern of spread explains why esophageal metastases from breast cancer often present as submucosal lesions causing extrinsic compression or circumferential narrowing, rather than discrete intraluminal mucosal masses. The middle third of the esophagus is most frequently affected, likely due to its anatomical proximity to the major mediastinal lymphatic drainage pathways from the breast and tracheobronchial tree.[15,16]

Metastasis to the cervical esophagus, as in Madam NNS's case, is considerably rarer. The lymphatic drainage of the cervical esophagus is distinct from the thoracic esophagus. It primarily drains to the paratracheal lymph nodes, the deep internal jugular chain, and potentially the supraclavicular nodes. Involvement of the cervical esophagus by metastatic breast cancer could theoretically occur through several less common pathways: extensive mediastinal lymphatic disease with retrograde spread superiorly into the cervical paraesophageal lymphatics; direct hematogenous seeding to the cervical esophagus (less likely given the typical lymphatic pattern); or metastasis to cervical lymph nodes (supraclavicular or deep jugular) with subsequent direct extension or secondary lymphatic spread to the adjacent cervical esophagus. In this patient, the CT findings of a large mass in the thyroid/isthmus region at C6-Th2, associated with extensive bilateral cervical lymphadenopathy, suggest a significant burden of metastatic disease in the neck. This could represent either primary cervical nodal metastasis with subsequent invasion of adjacent structures including the esophagus, or direct metastatic seeding to the soft tissues of the neck with secondary esophageal involvement. The encasement of the trachea and esophagus by this mass points to a locally aggressive metastatic deposit.[17,18]

A detailed understanding of esophageal anatomy and physiology is crucial to appreciate how metastatic disease, particularly from an extraluminal source, can lead to profound dysphagia. The esophagus is a muscular tube, approximately 25 cm in length, extending from the pharynx (at the level of the cricoid cartilage, C6) to the stomach (at the level of T11). It is divided into cervical, thoracic, and abdominal parts. The cervical esophagus, relevant to this case, is about 5 cm long and lies posterior to the trachea. The esophageal wall comprises four layers: mucosa, submucosa, muscularis propria, and adventitia (serosa for the intra-abdominal part). The mucosa consists of non-keratinized stratified squamous epithelium, lamina propria, and muscularis mucosae. Primary esophageal cancers typically arise from this layer. The submucosa is a layer of loose connective tissue containing blood vessels, nerves (Meissner's plexus), and, importantly, an extensive network of lymphatic vessels. This submucosal lymphatic plexus is where metastatic cells often deposit and proliferate,

as discussed. The muscularis propria consists of an inner circular and an outer longitudinal muscle layer. In the proximal third (including the cervical esophagus), these muscles are predominantly striated (voluntary), transitioning to smooth muscle (involuntary) in the distal two-thirds. Auerbach's (myenteric) plexus, which controls peristalsis, lies between these muscle layers. The adventitia is the outermost layer of connective tissue, anchoring the esophagus to surrounding structures.[19,20]

The upper esophageal sphincter (UES) is a high-pressure zone located at the pharyngoesophageal junction, primarily formed by the cricopharyngeus muscle, along with contributions from the inferior pharyngeal constrictor and the proximal cervical esophagus. The UES is tonically contracted at rest, preventing air entry into the esophagus and reflux of esophageal contents into the pharynx. During swallowing, coordinated neural reflexes cause UES relaxation and opening, allowing bolus passage. Esophageal peristalsis, a series of coordinated muscle contractions, propels the bolus distally. Primary peristalsis is initiated by swallowing, while secondary peristalsis is triggered by esophageal distension from a retained bolus or refluxed material.

Extraluminal compression or infiltration of the esophageal wall by a metastatic mass, as occurred in Madam NNS's case at the C6-Th2 level, can disrupt esophageal function in several ways. Direct narrowing of the lumen physically impedes bolus passage, leading to dysphagia. This is the most straightforward mechanism. The smooth, slippery mucosa found on esophagoscopy is a hallmark of extrinsic compression, where the overlying mucosa is stretched but not primarily diseased. A mass in the cervical esophagus can directly impinge on the UES or the neural pathways controlling its relaxation, leading to incomplete opening or premature closure. This contributes significantly to oropharyngeal dysphagia symptoms like food sticking high in the throat. Infiltration of the muscularis propria or the myenteric plexus can disrupt normal peristaltic activity in the affected segment, leading to ineffective bolus transport even if the lumen is not completely obliterated. Involvement of sensory nerves in the esophageal wall might alter the perception of bolus passage or trigger abnormal reflexes.

The symptomatology of esophageal metastasis from breast cancer is often insidious and non-specific, with dysphagia being the most common complaint. The character of dysphagia can provide clues to the level of obstruction. Typically, mid or distal esophageal obstruction leads to a sensation of food sticking retrosternally or in the lower chest. In contrast, Madam NNS presented predominantly with oropharyngeal dysphagia: difficulty initiating the swallow, immediate coughing and choking upon swallowing attempts, and a sensation of blockage high in the throat. This presentation is highly suggestive of an obstruction at or very near the pharyngoesophageal junction (UES and cervical esophagus). The mechanics of oropharyngeal swallowing are complex, involving precise coordination of the tongue, pharyngeal constrictors, larynx, and UES. A structural lesion like a high cervical esophageal stenosis, as identified by FEES and esophagoscopy in this patient, can profoundly disrupt this coordination. The narrowed inlet prevents efficient passage of the bolus from the pharynx into the esophagus. The pharynx contracts against a relatively closed UES or a stenotic segment immediately distal to it, leading to increased intrapharyngeal pressure. Incomplete bolus clearance results in significant residue in the valleculae and piriform sinuses post-swallow, as observed with the pooling of secretions in this case. This residue can subsequently spill into the larynx (penetration) or trachea (aspiration) during or after the swallow, leading to coughing and choking. The FEES findings of both penetration and aspiration directly support this mechanism. Such symptoms can easily be mistaken for primary neuromuscular disorders (stroke, motor neuron disease, myasthenia gravis) or primary laryngeal/pharyngeal pathology, delaying the consideration of an esophageal cause. The initial neurological consultation in this case reflects this diagnostic challenge.

The long latency period between the primary breast cancer treatment and the onset of esophageal symptoms is another hallmark that can complicate diagnosis. Madam NNS developed dysphagia approximately 20 years after her initial breast cancer diagnosis and treatment (and 4 years after treatment for a recurrence). Such prolonged disease-free intervals can lead to a decreased index of suspicion for metastatic disease. Breast cancer cells are known for their ability to enter a state of dormancy, sometimes for many decades, before reactivating to cause overt metastases. The triggers for this reactivation are not fully understood but may involve changes in the host immune system, hormonal milieu, or the microenvironment of the dormant cells. This phenomenon of late recurrence is well-documented in breast cancer literature, with esophageal metastases reported 10, 15, or even more than 20 years after primary diagnosis. This case adds to the body of evidence supporting the need for lifelong vigilance in breast cancer survivors who develop new, unexplained symptoms.

The diagnostic journey in cases of suspected esophageal metastasis, particularly with atypical presentations, requires a systematic approach leveraging multiple modalities. Fiberoptic Endoscopic Evaluation of Swallowing (FEES) was a crucial initial investigation in Madam NNS's case, performed by the ORL-HNS team. FEES allows direct visualization of the pharynx and larynx during the act of swallowing various food consistencies. Its strengths include portability, repeatability, lack of radiation exposure, and excellent visualization of upper airway anatomy and physiology. In this patient, FEES provided several key pieces of information:Objective confirmation of oropharyngeal dysphagia with penetration and aspiration; Identification of pooling of secretions in the hypopharynx, indicative of impaired clearance; Most importantly, direct visualization of a narrowed esophageal inlet (introitus esophagus) with features suggestive of stenosis and extraluminal compression. This finding immediately shifted the diagnostic focus towards a structural lesion at the UES or proximal cervical esophagus, rather than a purely neuromuscular issue; The observation of "vocal cords not docked during adduction", while subtle, warranted further investigation to rule out any neurological component affecting laryngeal function, although the dominant finding was clearly the stenosis. Following the FEES findings and the inability to re-pass an NGT, operative esophagoscopy under general anesthesia was the logical next step. Rigid esophagoscopy offers excellent visualization of the proximal esophagus and allows for therapeutic interventions like dilation or biopsy under direct vision. It confirmed the presence of a severe, high-grade stenosis in the proximal cervical esophagus, located at C6, approximately 1-2 cm from the inlet. The stenosis was impassable with the standard adult esophagoscope, underscoring its severity. The "slippery mucosal surface" was a critical observation. This appearance is characteristic of extrinsic compression, where the underlying mucosa is stretched taut over a compressing mass, rather than being primarily neoplastic itself. Intrinsic mucosal cancers typically present with ulceration, friability, an exophytic mass, or irregular nodularity. The decision not to biopsy was a sound clinical judgment based on the risk of perforation when dealing with a tight, extrinsically compressing lesion, especially without definitive knowledge of its vascularity or precise relationship to adjacent vital structures prior to cross-sectional imaging.

The contrast-enhanced CT scan was pivotal in establishing the definitive diagnosis and staging the disease. CT provides excellent anatomical detail of soft tissues, bone, and vasculature. The CT delineated a large, aggressive-appearing heterogeneous mass in the lower neck (C6-Th2), involving the thyroid region and extending into surrounding visceral and retropharyngeal spaces. Crucially, it demonstrated the mass encasing the trachea and, most relevant to the patient's symptoms, causing severe stenosis of the cervical esophagus with suspected infiltration of the esophageal wall. The presence of extensive bilateral cervical lymphadenopathy further characterized the locoregional extent of the metastatic disease. The term

"encasement" implies that the tumor surrounds more than 270 degrees of the circumference of a structure, a feature highly suggestive of malignancy. The heterogeneity and central necrosis within the mass are also common features of aggressive tumors that have outgrown their blood supply in certain areas. The CT scan of the thorax revealed widespread distant metastases, including characteristic osteolytic lesions in the right scapula (indicative of bone metastasis) and multiple spiculated pulmonary nodules in both lungs (classic for pulmonary metastases). These findings confirmed that the cervical esophageal compression was not an isolated metastatic event but part of a disseminated systemic recurrence of her breast cancer. The identification of these distant metastases was critical for determining the overall prognosis and guiding the shift towards palliative management. CT's ability to simultaneously assess locoregional disease and distant metastatic sites makes it an indispensable tool in oncological staging. Performed during the neurological workup, the head MRI served to rule out a primary central nervous system cause for the dysphagia, such as a stroke or brain tumor affecting swallowing centers. The reported neurovascular conflict involving cranial nerves IX and X was noted but considered less likely to explain the mechanical obstruction seen on FEES and esophagoscopy. While MRI of the neck can be very useful for soft tissue delineation, in this case, the subsequent neck CT provided the necessary information regarding the cervical mass. Although not performed in this case due to anatomical limitations (likely the very high and tight nature of the stenosis making safe passage and manipulation of the EUS scope impossible), EUS-FNA warrants discussion as it is often a key diagnostic tool for esophageal submucosal lesions. EUS provides high-resolution imaging of the esophageal wall layers and adjacent structures (mediastinum, lymph nodes). It can accurately define the depth of tumor invasion and the relationship of an extraluminal mass to the esophageal wall. FNA performed under EUS guidance allows for targeted tissue acquisition from submucosal lesions or extrinsic masses with a high diagnostic yield, often exceeding that of conventional endoscopic biopsies for such lesions. Had it been feasible, EUS-FNA could have potentially provided a direct cytopathological confirmation from the cervical mass compressing the esophagus. Its absence in this case highlights that even advanced diagnostic techniques have practical limitations dictated by individual patient anatomy and lesion characteristics.

Direct histopathological confirmation from the esophageal lesion itself was not obtained in Madam NNS's case due to the high risk of perforation associated with biopsying a tight, extrinsically compressing stenosis. This is not an uncommon scenario in cases of esophageal metastasis. Because these metastases are often submucosal or adventitial, standard mucosal "pinch" biopsies taken during esophagoscopy frequently yield only normal overlying squamous epithelium or non-specific inflammatory changes, leading to false-negative results. This is a major reason why EUS-FNA has become so valuable, as it can target the deeper pathological tissue. In the absence of a direct biopsy from the esophageal lesion, the diagnosis of metastatic breast cancer was established with a very high degree of certainty based on: The patient's unequivocal history of invasive breast cancer with a prior recurrence, The characteristic radiological appearance of the cervical mass on CT (aggressive, infiltrative, causing extrinsic compression), The presence of extensive, unequivocally metastatic disease in cervical lymph nodes, lungs, and bone, all confirmed by CT. The patterns of these distant metastases were entirely consistent with dissemination from breast cancer. Had a biopsy been obtainable, immunohistochemical staining would have been crucial for confirming breast origin. Markers such as GATA-3 (a transcription factor highly specific for breast and urothelial tissues), mammaglobin, and gross cystic disease fluid protein-15 (GCDFP-15) are commonly used. Additionally, assessment of estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor 2 (HER2) status on the metastatic tissue is vital for guiding systemic therapy, as these can

sometimes differ from the primary tumor. The patient's recurrent tumor in 2019 was HER2-positive, which guided her chemotherapy at that time. Different histological subtypes of breast cancer have varying metastatic patterns. Invasive lobular carcinoma (ILC), for instance, is known to have a higher propensity for metastasizing to the GI tract, including the esophagus and stomach, often in an infiltrative, linitis plastica-like pattern that can be particularly difficult to diagnose endoscopically. Invasive ductal carcinoma (IDC), the most common type (and the patient's primary histology), can also metastasize to the esophagus, typically forming discrete masses or causing extrinsic compression as seen in this case.

When a patient with a history of breast cancer presents with esophageal stenosis and dysphagia, metastatic recurrence is naturally high on the list of differentials. However, a thorough clinician must consider other possibilities. This includes squamous cell carcinoma (more common in the upper and mid-esophagus) and adenocarcinoma (more common in the distal esophagus, often associated with Barrett's esophagus). Primary esophageal cancers typically arise from the mucosa and often present with ulceration, friability, or an exophytic intraluminal mass, features not prominent in this case, where the mucosa was described as smooth and slippery. However, infiltrative primary tumors can occur. Peptic Stricture caused by chronic gastroesophageal reflux disease (GERD), usually affecting the distal esophagus. Highly relevant in this patient given her history of radiotherapy to the chest wall/axilla/supraclavicular fossa for her primary cancer and subsequent recurrence. Radiation can cause acute esophagitis followed by chronic fibrosis and stricture formation, often months to years after treatment. Differentiating radiation fibrosis from metastatic recurrence can be challenging. However, the CT appearance of a large, enhancing, necrotic mass is far more suggestive of active malignancy than bland fibrotic change. Other study also described esophageal stenosis post-chemotherapy, but the imaging here was not consistent with a simple fibrotic or inflammatory stricture. Eosinophilic esophagitis, caustic ingestion, post-surgical anastomotic strictures (not applicable here). Lung cancer, for example, can metastasize to the esophagus or mediastinal lymph nodes causing esophageal compression. Given the location of the mass in the thyroid/isthmus region, an aggressive primary thyroid cancer (anaplastic thyroid cancer) invading the esophagus would be a consideration in a patient without a history of breast cancer. Esophageal involvement by lymphoma can occur, either primary or secondary. As initially considered, various neurological disorders can cause oropharyngeal dysphagia. The negative head MRI and the clear structural obstruction identified endoscopically and radiologically made a primary neuromuscular cause unlikely, although secondary dysmotility from tumor infiltration of nerves or muscle cannot be entirely excluded as a contributing factor. The comprehensive diagnostic workup, especially the CT scan revealing the aggressive cervical mass and widespread systemic metastases characteristic of breast cancer, alongside the patient's history, overwhelmingly favored metastatic breast cancer as the unifying diagnosis.

Once widespread metastatic disease is confirmed, as in Madam NNS's case, the primary goals of treatment shift from curative intent to palliation. This involves alleviating symptoms, maintaining or improving quality of life, and potentially prolonging survival with systemic therapies. A multidisciplinary team approach is indispensable. This was the most pressing issue for Madam NNS. The placement of a Stamm gastrostomy provided a secure and durable means of enteral nutrition, bypassing the esophageal obstruction and ensuring adequate caloric and fluid intake. This is a cornerstone of palliative care for malignant dysphagia. Self-expandable metallic stents (SEMS) are often used to palliate malignant esophageal obstruction. They can be placed endoscopically and provide rapid relief of dysphagia. However, stenting in the cervical esophagus is more complex than in the mid or distal esophagus. Potential complications include foreign body sensation, pain,

stent migration, tumor ingrowth or overgrowth, and increased risk of aspiration if the stent interferes with UES function or laryngeal protection. Given the very high location (C6) and the nature of the extrinsic compression, stenting might have been considered but deemed less optimal or more risky than primary gastrostomy in this debilitated patient. Palliative radiotherapy to the obstructing cervical mass could be considered to shrink the tumor and relieve compression, but its effectiveness depends on tumor radiosensitivity and the patient's overall condition. Ablative therapies (laser, argon plasma coagulation) are generally not suitable for extrinsic compressive lesions. The choice of systemic therapy depends on several factors, including tumor hormone receptor status (ER/PR), HER2 status, prior treatments, extent and location of metastases, and patient performance status. Options include: Chemotherapy: Various cytotoxic agents can be used; Endocrine Therapy: For ER/PR-positive tumors; HER2-Targeted Therapy: For HER2-positive tumors (Trastuzumab, Pertuzumab, ado-trastuzumab emtansine, etc.). Madam NNS's recurrence in 2019 was HER2-positive, and she received Trastuzumab. Re-biopsy of a metastatic site, if feasible, can sometimes reveal changes in receptor status; The oncology team would consider these options in the context of her extensive prior treatment and current widespread disease. The prognosis for patients with esophageal metastasis from breast cancer is generally poor, reflecting Stage IV disease. Survival is typically measured in months, though some patients may live longer with effective systemic therapy and good performance status. The primary aim is to optimize the remaining quality of life. This case, with its intricate diagnostic pathway and convergence of rare clinical features—cervical esophageal metastasis from breast cancer presenting with severe oropharyngeal dysphagia decades after initial treatment—serves as a potent reminder of the complex and often unpredictable nature of metastatic cancer. It underscores the necessity for clinicians to maintain a broad differential diagnosis, employ a systematic and multidisciplinary approach to investigation, and always consider the possibility of late recurrence in cancer survivors presenting with new, unexplained symptoms. The detailed journey of Madam NNS from initial symptom to definitive diagnosis and palliative care planning provides invaluable lessons for otorhinolaryngologists, oncologists, gastroenterologists, and all healthcare professionals involved in the care of cancer patients. Her story emphasizes the importance of careful listening to the patient's narrative, meticulous clinical examination, and the judicious use of modern diagnostic technologies to unravel even the most perplexing clinical presentations.

4. Conclusion

This compelling case of a 60-year-old woman, manifesting severe oropharyngeal dysphagia from a compressive cervical esophageal stenosis due to metastatic breast cancer two decades post-primary diagnosis, vividly underscores critical oncological lessons. The unique presentation—marked by the exceptionally proximal esophageal metastatic site and the dominant oropharyngeal symptoms—mandates a heightened clinical suspicion for metastatic recurrence in breast cancer survivors, even with atypical manifestations or after extended disease-free periods. The diagnostic journey highlighted the indispensable synergy of meticulous clinical acumen with advanced modalities like FEES and CT imaging in unraveling this complex scenario. Ultimately, this rare case reinforces that while cervical esophageal metastasis from breast cancer is uncommon, its possibility demands consideration, with a multidisciplinary, palliative-focused approach being paramount for managing such advanced presentations and optimizing patient quality of life.

5. References

1. Sakanaka K, Fujii K, Kokubo M, Ogura M, Itasaka S, Sakamoto T, et al. Improvement in Quality of Life and dysphagia after palliative external beam radiotherapy for malignant

esophageal stenosis of esophageal cancer. JCO Oncol Pract. 2025; OP2400429.
2. Nagayabu K, Sumida W, Ota K, Ono Y. Thoracoscopic congenital esophageal stenosis repair and Thal fundoplication by right thoracic approach: a case report. Surg Case Rep. 2025; 11(1).
3. Tanaka I, Boldbaatar G, Ushikubo K, Yamamoto K, Nishikawa Y, Tanabe M, et al. Correction: Longitudinal mucosal incision prior to balloon dilation: Novel and advanced approach for severe esophageal stenosis. Endosc Int Open. 2025; 13(CP): a26162611.
4. Mou T-T, Xu Y, Qian L-Z, Mao X-L, Zheng H-H, Zhang Y. Esophageal mucosal auto-transplantation versus acellular dermal matrix transplantation for preventing esophageal stenosis after endoscopic resection in patients with superficial esophageal mucosal lesions. Surg Endosc. 2025.
5. Liu X, Li C, Zhao F, Guidoin R, Liu K, Wang F, et al. A functional stent with near-infrared light triggered localized photothermal-chemo synergistic therapy for malignant stenosis of esophageal cancer. Colloids Surf B Biointerfaces. 2025; 251(114634): 114634.
6. Yano H, Sasaki F, Maeda H, Uehara S, Kabayama M, Fujino Y, et al. Effect of sprayable, highly adhesive hydrophobized gelatin microparticles on esophageal stenosis after endoscopic submucosal dissection: an experimental study in a swine model. Esophagus. 2025; 22(1): 95–104.
7. Karapetyan L, Laird-Fick H, Cuison R. Metastatic invasive lobular breast cancer presenting clinically with esophageal dysphagia. Case Rep Oncol Med. 2017; 2017: 7065674.
8. Saldi S, Perrucci E, Lancellotta V, Palumbo I, Falcinelli L, Mariucci C, et al. EP-1292 Zinc-L-Carnosine prevents dysphagia in breast cancer patients treated with adjuvant radiotherapy. Radiother Oncol. 2019; 133: S707–8.
9. Alfian Sulai DD, Krishnasamy T, Nik Mahmood NRK. Diagnostic challenge in silent metastatic invasive breast carcinoma: dysphagia as the only symptom. BMJ Case Rep. 2021; 14(7): e239997.
10. Kim MS, Huh S, Min JH, Shin Y-I, Kim S, Ko S-H. Oropharyngeal dysphagia detected on VFSS in a patient with Pancoast tumor: a case report. J Korean Dysphagia Soc. 2023; 13(2): 149–54.
11. Lee R, Lee CH, Yun YJ, Seo HG, Park S-H, Oh B-M. Dysphagia caused by dermatomyositis misdiagnosed as tumor metastasis: a case report. J Korean Dysphagia Soc. 2024; 14(1): 71–9.
12. Gómez Á, García-Chabur MA, Peñaranda D, Gómez-Mendoza A, Forero JC. Chemotherapy/radiotherapy-induced dysphagia in head and neck tumors: a challenge for otolaryngologists in low- to middle-income countries. Dysphagia. 2024.
13. Liu A, Feng Y, Chen B, Li L, Wu D, Qian J, et al. A case report of metastatic breast cancer initially presenting with esophageal dysphagia. Medicine (Baltimore). 2018; 97(45): e13184.
14. El Mobadder M, Farhat F, Nammour S. Photobiomodulation therapy in the treatment of chronic dysphagia post hormonal therapy in a breast cancer patient. Dent J. 2019; 7(2): 53.
15. Case Medical Research. Zinc-L-carnosine prevents dysphagia in breast cancer patients undergoing adjuvant radiotherapy. Case Medical Research. 2019.
16. Khanam R, Fanous IS, Fadhel EN, Hyder T, Brufsky A. Voltage-gated calcium channel antibody-induced oropharyngeal dysphagia presenting as a paraneoplastic neurological complication in breast cancer. Cureus. 2021; 13(3): e13677.

17. McNally B, Suchartlikitwong S, Haddad N, Drewitz D, Pitea TC. S2269 malignant dysphagia in latent triple negative metastatic breast cancer. Am J Gastroenterol. 2022; 117(10S): e1530–1.
18. Kodera A, Inoue H, Ogura K, Sakaguchi S, Yukawa H, Matsuoka A, et al. Bevacizumab plus paclitaxel therapy was effective for metastatic breast cancer with dysphagia due to mediastinal lymph node metastasis-A case report. Gan To Kagaku Ryoho. 2018; 45(13): 2276–8.
19. Saldi S, Perrucci E, Fulcheri CPL, Mariucci C, Chierchini S, Ingrosso G, et al. Zinc-L-carnosine prevented dysphagia in breast cancer patients undergoing adjuvant radiotherapy: Results of a phase III randomized trial. Breast J. 2020; 26(9): 1882–4.
20. Fousekis FS, Tepelenis K, Stefanou SK, Stefanou CK, Pappas-Gogos G, Theopistos V, et al. Gastric metastasis from breast cancer presenting as dysphagia. J Surg Case Rep. 2022; 2022(3): rjac080.