e-ISSN: 2747-2051

Archives of the Medicine and Case Reports

[AMCR]

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

When the Lungs Suffer for the Ovary: A Case of Meigs Syndrome Presenting as Severe Dyspnea and Massive Unilateral Pleural Effusion

Nadila Ayu Putri[1]*, Fadel Fikri Suharto[2]

[1]General Practitioner, BARI Regional General Hospital, Palembang, Indonesia

[2]Department of Internal Medicine, BARI Regional General Hospital, Palembang, Indonesia

ARTICLE INFO

Keywords:
Ascites
Dyspnea
Meigs syndrome
Ovarian fibroma
Pleural effusion

***Corresponding author:**
Nadila Ayu Putri

E-mail address:
nadilaayuputri8@gmail.com

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

https://doi.org/10.37275/amcr.v6i4.808

A B S T R A C T

Meigs syndrome is a rare clinical entity defined by the triad of a benign ovarian tumor, ascites, and pleural effusion. It characteristically resolves completely after the surgical removal of the primary tumor. Despite its benign nature, the clinical presentation often mimics advanced-stage ovarian malignancy, posing a significant diagnostic challenge. A 47-year-old nulligravid woman presented with a two-week history of severe dyspnea, orthopnea, and pleuritic chest pain, set against a four-month backdrop of progressive abdominal distension and a 10-kg weight loss. Physical examination revealed findings consistent with a massive right-sided pleural effusion, significant ascites, and a large, firm pelvic-abdominal mass. Imaging confirmed a massive right pleural effusion and a complex ovarian mass exceeding 16 cm. A diagnosis of Meigs syndrome was established after pleural fluid analysis revealed a cytologically negative, exudative fluid. The patient's acute respiratory distress was managed with serial therapeutic thoracentesis, with 5,000 mL of fluid removed over five days, leading to dramatic symptomatic improvement. She was subsequently stabilized for definitive surgical intervention. In conclusion, this case powerfully illustrates the critical importance of including Meigs syndrome in the differential diagnosis for women with an ovarian mass and concurrent pleuro-peritoneal effusions. Timely recognition and a staged, multidisciplinary management approach are paramount for alleviating life-threatening symptoms and achieving an excellent prognosis, reaffirming the clinician's highest duty: to diligently seek out the curable, even when faced with the seemingly incurable.

1. Introduction

Meigs syndrome, first systematically described by Joe Vincent Meigs in 1937, represents a fascinating and rare intersection of gynecology and internal medicine. It is classically defined by the triad of a benign solid ovarian tumor, accompanied by ascites and pleural effusion. The most remarkable characteristic of this syndrome is the complete and permanent resolution of the effusions and associated symptoms following the surgical resection of the ovarian tumor.[1] While the first observations were noted as early as 1887 by Demons, Meigs' comprehensive case series solidified its place in medical literature. The syndrome is most frequently diagnosed in postmenopausal women, with a peak incidence between 30 and 60 years.[2] The tumors most commonly implicated are benign fibro-thecomatous tumors of the ovarian stroma, with ovarian fibromas being the most frequent type. However, this clinical picture has also been reported with other benign tumors such as Brenner tumors, granulosa cell tumors, and struma ovarii.[3] The full triad of Meigs

syndrome occurs in only about 1-2% of cases of ovarian fibroma, typically when the tumor is larger than 10 cm in diameter. The pathophysiology remains a subject of investigation, with leading hypotheses implicating mechanical peritoneal irritation and, more compellingly, the tumor's production of potent vascular mediators like vascular endothelial growth factor (VEGF) and inflammatory cytokines, which increase capillary permeability.[4] The pleural effusion, characteristically right-sided, is thought to result from the trans-diaphragmatic passage of ascitic fluid.[5]

It is crucial to distinguish true Meigs syndrome from its mimics. "Pseudo-Meigs Syndrome" presents with an identical clinical triad, but the tumor is not a benign ovarian fibroma; it can be another pelvic tumor (benign or malignant), such as a uterine leiomyoma, or even a metastatic cancer to the ovary.[6] "Pseudo-pseudo Meigs Syndrome" (Tjalma Syndrome) involves multiple serosal effusions and an elevated CA-125 in the context of systemic lupus erythematosus.[7,8] This nosology underscores the diagnostic precision required. The clinical presentation—a middle-aged woman with weight loss, an abdominal mass, and pleuro-peritoneal effusions—is nearly indistinguishable from advanced-stage ovarian cancer, creating a significant diagnostic dilemma that often leads to extensive and anxiety-provoking investigations.[9,10] The aim of this report is to present a classic yet compelling case of Meigs syndrome where severe, life-altering respiratory distress was the dominant initial symptom. The novelty of this report lies in its adherence to the CARE guidelines to provide a detailed, methodologically rigorous account of the step-by-step diagnostic reasoning, multidisciplinary management in a resource-limited setting, and the patient's own perspective on their clinical journey. We emphasize how prompt recognition and aggressive symptomatic treatment, specifically therapeutic thoracentesis, can serve as a vital bridge to definitive surgery, offering valuable, reproducible insights for clinicians and reinforcing the tenet that in the face of apparent metastatic disease, a benign and curable diagnosis must always be considered.

2. Case Presentation

Chronological timeline of the patient's case, illustrating a decade-long period of clinical latency followed by a rapid, four-month progression to acute cardiorespiratory decompensation, Figure 1. The timeline begins approximately ten years prior to admission with the incidental ultrasonographic finding of a large, asymptomatic ovarian cyst measuring over 10 cm. This initial discovery was not pursued with further intervention. A key inflection point occurred six years prior to presentation, when the patient experienced symptomatic enlargement of the mass, characterized by abnormal uterine bleeding and severe abdominal pain, with imaging confirming an increase in size to over 13 cm. Despite these warning signs, a significant period of clinical quiescence followed, ending four months prior to admission with the insidious onset of progressive abdominal distension, signaling the development of significant ascites. This marked the beginning of a rapid decline, which accelerated dramatically in the two weeks preceding hospitalization with the onset of severe, worsening dyspnea as a massive pleural effusion accumulated. The culmination of this decade-long process was her emergency admission in a state of severe respiratory distress. The final phase of the timeline documents the critical interventions performed over five days, wherein serial therapeutic thoracentesis resulted in the drainage of five liters of pleural fluid, leading to the successful stabilization of her acute symptoms and paving the way for definitive surgical planning. This graphical representation clearly delineates the transition from a chronic, indolent gynecological condition to an acute, life-threatening medical emergency.

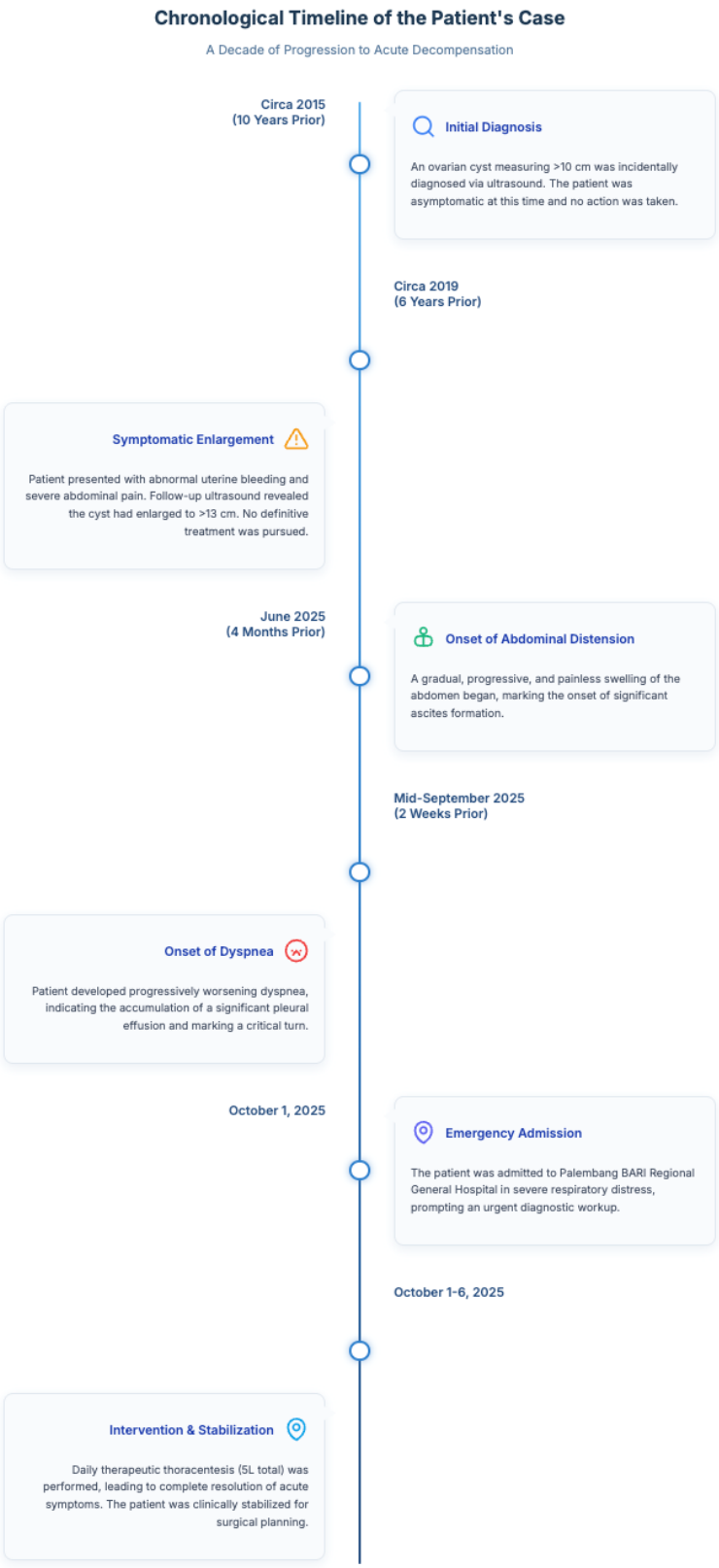

Figure 1. Chronological timeline of the patient's case.

A 47-year-old married, nulligravid woman was admitted to the emergency department with a severe and progressively worsening complaint of dyspnea over a two-week period. The patient's clinical history was significant for a long-standing ovarian mass. Figure 2 provides a comprehensive, multi-domain overview of the patient's clinical history, meticulously organized to highlight the key factors culminating in her acute presentation. The figure is structured into four distinct quadrants, each representing a critical aspect of the clinical narrative: the History of Present Illness, Past Medical History, Patient Profile, and a summary of Pertinent Negatives. This structured approach facilitates a clear understanding of the temporal evolution of her condition and the constellation of symptoms that defined her case. The central and most prominent section, the "History of Present Illness," details the acute clinical decompensation. It emphasizes the critical two-week period during which the patient's chief complaint of severe, progressive dyspnea manifested. This section further dissects the symptomatology into distinct systems. The respiratory symptoms are defined by their severity, including orthopnea and sharp, right-sided pleuritic chest pain, indicative of significant pleural inflammation and fluid accumulation. Concurrently, the insidious onset of progressive abdominal distension over four months points to the gradual development of ascites. Crucially, this section quantifies the systemic impact of her illness, noting a significant, unintentional weight loss of 10 kilograms over three months, a finding that strongly raised the initial suspicion of a malignant process. The inclusion of new-onset amenorrhea and pelvic pressure provides the vital link to a primary gynecological pathology. The "Past Medical History" quadrant provides the essential backstory, establishing the chronic nature of the underlying ovarian mass. It chronicles the initial incidental diagnosis of an asymptomatic ovarian cyst measuring over 10 centimeters a decade prior. A critical event is noted six years before presentation, when symptomatic enlargement to over 13 centimeters, accompanied by pain and abnormal bleeding, signaled a change in the tumor's behavior. The explicit notation that no consistent gynecological care was pursued underscores a significant gap in management that allowed the condition to progress unchecked for years. The "Patient Profile" section offers key demographic and social context. Her nulligravid status is a relevant detail in her gynecological history. The mention of a negative family history for ovarian or other cancers is a crucial piece of information that, while not exclusionary, slightly lowers the pre-test probability of a hereditary cancer syndrome. Finally, the "Pertinent Negatives" quadrant is of paramount importance for illustrating the process of differential diagnosis. By explicitly stating the absence of clinical or historical evidence for chronic liver disease, congestive heart failure, or thromboembolic disease, the figure demonstrates a systematic exclusion of other common causes of pleuro-peritoneal effusions. Furthermore, the lack of signs or symptoms suggestive of active tuberculosis helps to rule out another key infectious differential, particularly in a relevant geographical context. Collectively, these four quadrants create a powerful, data-rich visual narrative that encapsulates the entirety of the patient's history, effectively framing the diagnostic challenge and guiding the clinical reasoning process. The schematic serves not just as a summary of facts, but as a visual representation of the diagnostic journey itself.

Upon initial assessment, the patient was in evident respiratory distress. Her physical examination was notable for findings localized to the thoracic and abdominal systems, which strongly corroborated the symptoms reported. Figure 3 provides a detailed, top-to-bottom summary of the key clinical signs identified during the physical examination upon the patient's admission to the emergency department.

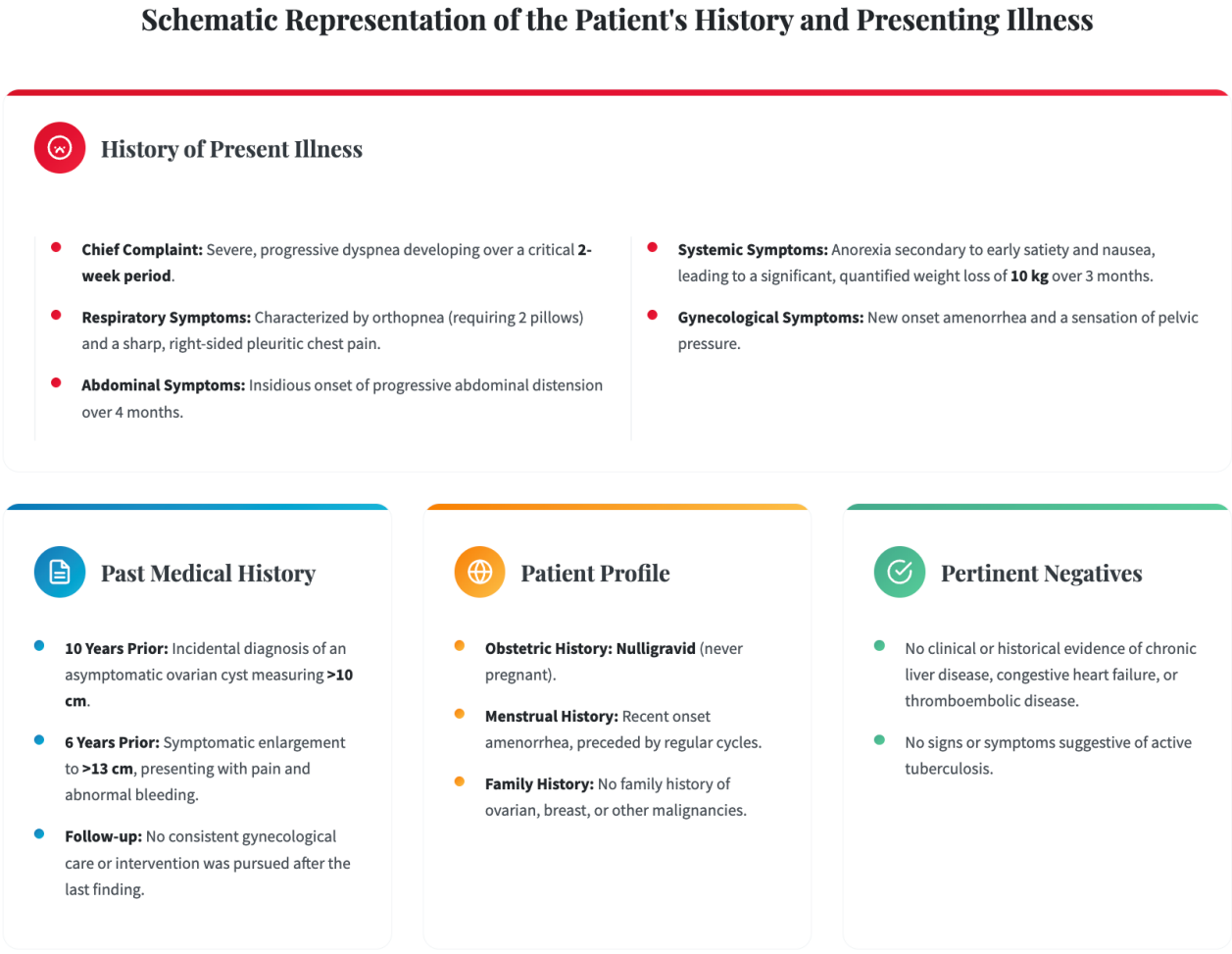

Figure 2. Patient's history and presenting illness.

The findings are systematically organized by anatomical region, collectively painting a compelling picture of a multi-system process dominated by massive fluid accumulation. The initial assessment of Vitals and General Appearance immediately highlighted the severity of the patient's condition. She was in moderate respiratory distress, evidenced by the use of accessory muscles, and appeared cachectic, consistent with her history of significant weight loss. The vital signs were notable for marked tachypnea, with a respiratory rate of 27 breaths per minute, a direct physiological response to the underlying hypoxia and mechanical impairment caused by the pleural effusion. Despite this, she was hemodynamically stable, maintaining a normal blood pressure and heart rate, with an oxygen saturation of 97% on low-flow supplemental oxygen. The Thoracic Examination revealed the classic and dramatic signs of a massive unilateral pleural effusion. Visual inspection showed asymmetrical chest wall expansion, with markedly reduced movement on the right side. This finding was corroborated by palpation, which demonstrated decreased tactile fremitus over the right hemithorax, and by percussion, which elicited stony

dullness—a flat, high-pitched sound indicative of a large fluid collection—over the entire right lung field. Auscultation confirmed these findings, with breath sounds being markedly diminished to completely absent on the right side, confirming the lack of air entry due to the compressive effect of the massive effusion. Crucially, the Cardiovascular Examination was entirely unremarkable. The jugular venous pressure (JVP) was not elevated, a key finding that argued strongly against congestive heart failure as the primary etiology of the fluid overload. Heart sounds were regular with normal S1 and S2 components, and no murmurs, gallops, or rubs were appreciated, ruling out significant valvular disease or pericardial effusion. The point of maximal impulse was non-displaced. The complete absence of any positive cardiovascular findings was a pivotal piece of the diagnostic puzzle, allowing the clinical team to confidently exclude a cardiac origin for the patient's symptoms. The Abdominal Examination revealed the second major component of the fluid overload. The abdomen was severely distended, with a measured circumference of 110 cm, and was tense on palpation. A large, firm, immobile mass with a smooth surface was easily palpable, occupying the lower and mid-abdomen. The presence of shifting dullness on percussion provided definitive clinical confirmation of significant ascites. These findings, when combined with the thoracic exam, established the presence of both pleural and peritoneal effusions in the setting of a large pelvic mass. Finally, the examination of the Extremities completed the clinical picture of systemic fluid dysregulation. There was bilateral, symmetrical, 2+ pitting edema extending to the mid-shins. This finding is consistent with the low oncotic pressure and high hydrostatic pressure state created by the underlying pathology. Importantly, there were no signs of deep vein thrombosis, such as unilateral calf swelling or tenderness, making a pulmonary embolism a less likely cause of her acute dyspnea. In aggregate, the physical examination was instrumental in establishing the clinical triad of a large pelvic mass, massive right-sided pleural effusion, and significant ascites, while simultaneously providing key negative findings that helped to systematically narrow the extensive list of differential diagnoses.

To elucidate the cause of the patient's symptoms, a series of diagnostic investigations was performed. Imaging was pivotal in confirming the physical findings and establishing the triad of mass, ascites, and pleural effusion. Laboratory analysis of blood and pleural fluid was crucial for narrowing the broad differential diagnosis. Figure 4 illustrates the systematic, multi-stage diagnostic process employed in this case, visually representing the logical pathway from a broad initial differential diagnosis to a definitive and unexpected conclusion. The figure is structured as a diagnostic funnel, demonstrating how a sequence of targeted investigations systematically narrowed the field of possibilities, ultimately isolating the correct diagnosis. The process began with the Initial Assessment & Differential Diagnosis, which was formulated based on the patient's presenting constellation of symptoms and signs: a middle-aged woman with a large pelvic mass, massive ascites, significant pleural effusion, and constitutional symptoms of cachexia. At this early stage, the clinical picture was highly alarming and necessitated the consideration of several critical possibilities. The leading and most concerning diagnosis was Advanced Ovarian Malignancy, given that this presentation is the classic triad for metastatic epithelial ovarian cancer. Concurrently, Meigs Syndrome was maintained as a crucial, albeit rare, alternative. Given the potential for infectious etiologies to mimic malignancy, especially in certain geographical locations, Tuberculous Pleurisy and Peritonitis was included as a key differential. Finally, Systemic Causes of anasarca, such as decompensated congestive heart failure (CHF) or end-stage liver disease (cirrhosis) with hepatic hydrothorax, were considered, although they were ranked lower based on

the initial clinical assessment. The second stage, Key Diagnostic Investigations, was designed to critically evaluate and differentiate between these possibilities. The investigations were bifurcated into imaging and laboratory analyses. Imaging Findings were foundational, with a chest X-ray confirming the massive, unilateral right-sided pleural effusion, and a transabdominal ultrasound corroborating the presence of a large, predominantly solid ovarian mass measuring over 16 cm, alongside a large volume of ascites.

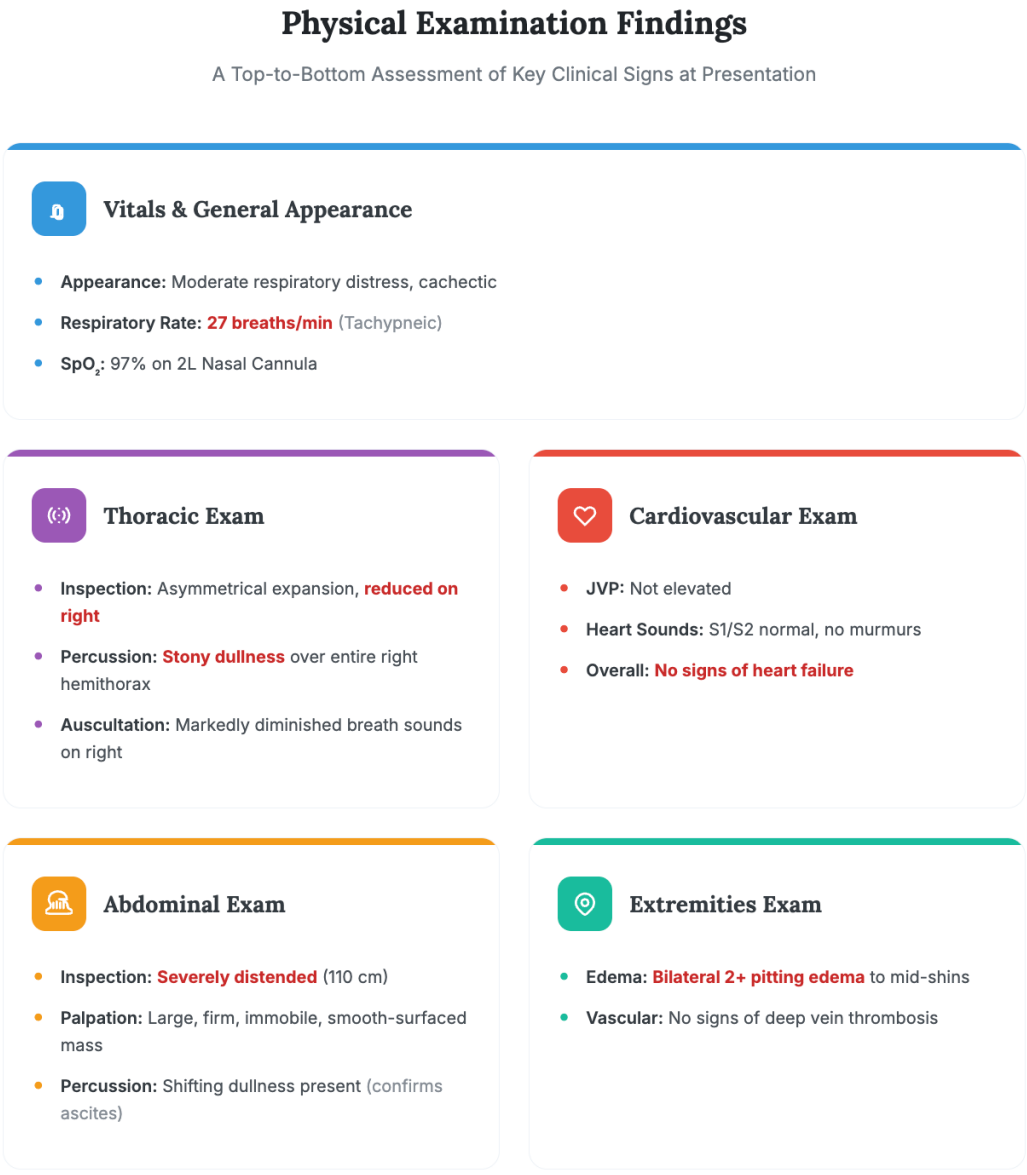

Figure 3. Physical examination findings.

These findings confirmed the physical triad but could not, on their own, distinguish between a benign or malignant etiology. The truly pivotal information was derived from the Laboratory & Fluid Analysis. The analysis of the pleural fluid, obtained via therapeutic and diagnostic thoracentesis, yielded three critical

results that decisively altered the diagnostic trajectory. First, the fluid was biochemically classified as an Exudate, a finding consistent with both malignancy and Meigs syndrome but less typical for systemic causes like CHF or cirrhosis, which usually produce transudates. Second, and most importantly, the Cytology of the fluid was unequivocally Negative for Malignant Cells. This result was the single most powerful piece of evidence against the leading diagnosis of metastatic ovarian cancer. Third, the Microbiology workup, specifically a nucleic acid amplification test for *Mycobacterium tuberculosis*, was Negative, effectively ruling out an active tuberculous infection as the cause of the effusions. The synthesis of these findings led to the final stage, the Final Diagnosis. With metastatic malignancy and tuberculosis excluded by the fluid analysis, and with systemic causes being inconsistent with the overall clinical picture, the diagnosis was distilled down to the one condition that perfectly fit the entire constellation of findings: the clinical triad of a benign ovarian tumor, ascites, and pleural effusion, with cytologically benign fluid. The final, definitive diagnosis was therefore confirmed to be Meigs Syndrome, transforming the patient's prognosis from one of presumed terminal illness to that of a completely curable condition.

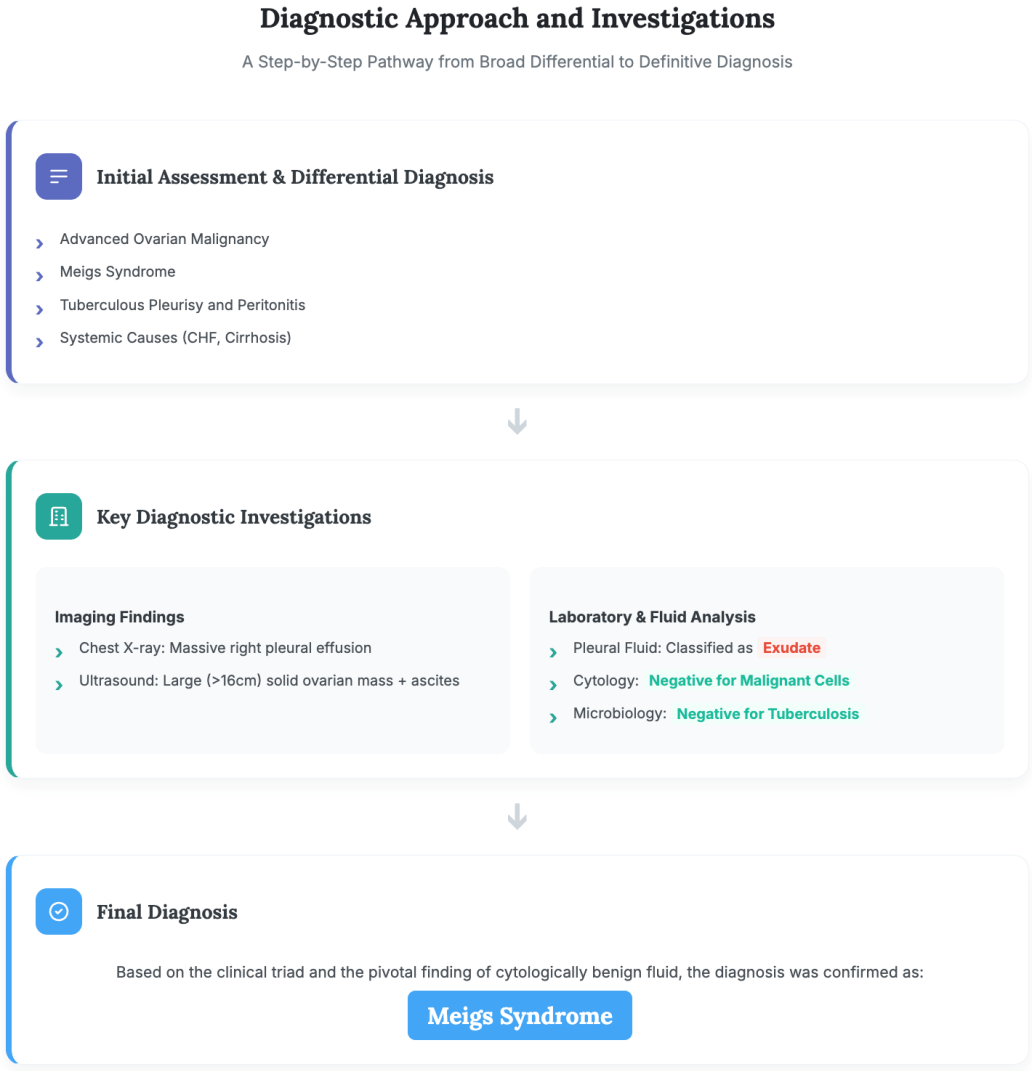

Figure 4. Diagnostic approach and investigations.

Based on the diagnosis, a multidisciplinary management plan was initiated. The immediate priority was alleviating the patient's severe respiratory distress. This was achieved through serial therapeutic thoracentesis. This intervention served as a critical bridge, stabilizing the patient for the definitive surgical treatment. Figure 5 provides a comprehensive overview of the therapeutic pathway and resultant clinical evolution of the patient over a critical five-day period of hospitalization. The figure is bifurcated into two main sections: an initial summary of the primary intervention and its overall outcome, followed by a detailed day-by-day timeline of the treatment course. This visual narrative effectively documents the patient's rapid transition from a state of acute, life-threatening respiratory distress to a stable, pre-surgical condition. The upper section serves as a synopsis, immediately identifying the Primary Intervention as Therapeutic Thoracentesis. It clarifies that the management strategy involved a staged, five-day course of ultrasound-guided pleural fluid drainage. The rationale for this approach is explicitly stated: to safely decompress the massive pleural effusion and facilitate a gradual, controlled re-expansion of the severely compressed right lung, thereby minimizing the risk of iatrogenic complications such as re-expansion pulmonary edema. The key quantitative and qualitative outcomes of this intervention are highlighted in distinct statistical boxes. Quantitatively, a total of 5,000 mL of pleural fluid was drained over the five-day period. Qualitatively, the definitive outcome was successful Stabilization of the patient, a critical prerequisite for proceeding with definitive surgical management. The central graphic, a pie chart visually representing the total volume drained, serves as an immediate and impactful anchor for this summary. The lower section of the figure delineates the granular details of this process in a sequential, day-by-day timeline. Each day is presented as a discrete card, detailing the specific volume of fluid drained and the corresponding, observable clinical response. Day 1: The intervention began with the removal of 1,000 mL of fluid, which yielded an immediate and significant clinical benefit, evidenced by an objective decrease in the patient's respiratory rate from 27 to 24 breaths per minute and a subjective initial improvement in her dyspnea. Day 2: A further 1,100 mL was drained. The clinical progress was substantial, allowing the patient to be completely weaned off supplemental oxygen and maintain adequate saturation on ambient air. Day 3: Another 1,100 mL was removed. This achieved a major clinical milestone: the resolution of orthopnea, which enabled the patient to lie flat comfortably for the first time since the onset of her severe symptoms. Day 4: An additional 1,000 mL was aspirated. By this stage, the patient reported a complete resolution of her subjective sensation of dyspnea while at rest, indicating that the mechanical load on her respiratory system had been significantly alleviated. Day 5: The final drainage of 800 mL completed the acute phase of treatment. This last intervention was associated with the cessation of her pleuritic chest pain. At the conclusion of this fifth day, the patient was deemed fully stabilized from a cardiorespiratory standpoint and was formally cleared for surgical planning. Collectively, this timeline illustrates a carefully titrated and highly effective therapeutic strategy. The final card in the sequence outlines the Definitive Treatment Plan, confirming that following this successful stabilization, the multidisciplinary team scheduled the patient for an exploratory laparotomy, the curative step in the management of Meigs syndrome.

The patient described her experience as overwhelming and frightening. For years, she had been aware of the ovarian cyst but had avoided follow-up due to fear and financial worries. The rapid onset of severe shortness of breath created a sense of panic and a fear of impending death. The initial discussions in the hospital, where cancer was mentioned as a primary concern, caused her and her family immense psychological distress.

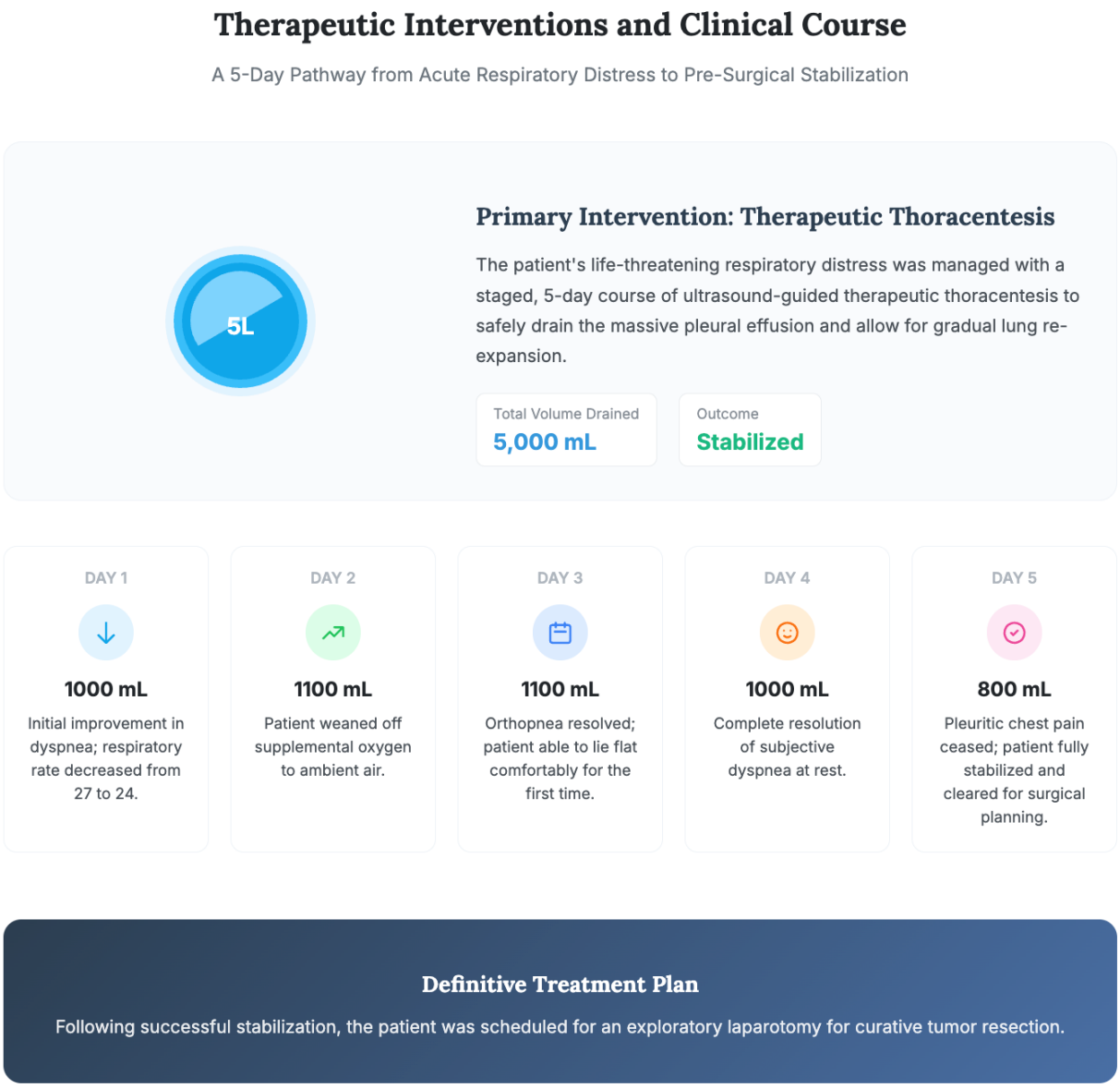

Figure 5. Therapeutic interventions and clinical course.

She expressed profound relief when the fluid drainage provided immediate ease in her breathing, describing it as "being able to live again." Learning that the condition was likely benign and curable was a moment of immense emotional release. She expressed gratitude for the multidisciplinary care and was hopeful about the upcoming surgery. Figure 6 provides a compelling visual narrative of the patient's emotional and psychological journey, mapping her experience from the initial phase of passive awareness to the final stage of hopeful resolution. The journey begins with the "Fear & Avoidance" stage, which encapsulates a multi-year period of latent anxiety. Having been aware of her large ovarian cyst for a decade, the patient's inaction was not due to ignorance but was rooted in a powerful combination of fear of a potential diagnosis and the practical barrier of financial worries. This stage highlights a common but

often under-reported aspect of patient behavior, where the psychological burden of a known but unaddressed medical issue leads to avoidance, allowing the underlying pathology to progress silently. The second stage, "Crisis & Uncertainty," marks the dramatic transition from chronic worry to acute medical emergency. The rapid onset of severe dyspnea precipitated a crisis, creating a visceral sense of panic and a fear of impending death. This physical crisis was immediately compounded by a profound psychological distress, as the clinical presentation was highly suggestive of a terminal illness. The discussions with the medical team, which necessarily included the possibility of advanced-stage cancer, plunged the patient and her family into a state of intense uncertainty and anguish. This stage underscores the immense psychological impact of a diagnostic workup for conditions that mimic malignancy. The third stage, "Relief & Intervention," represents a pivotal turning point in the patient's journey. The therapeutic thoracentesis provided not just physiological but also profound psychological relief. The ability to breathe comfortably again was described by the patient in deeply resonant terms as "being able to live again." This immediate, tangible improvement from the intervention served as the first concrete evidence that her condition was manageable, directly counteracting the sense of hopelessness that had characterized the preceding stage. This moment highlights the powerful therapeutic value of symptomatic relief in restoring a patient's sense of control and well-being. The final stage, "Hope & Resolution," documents the emotional climax of the diagnostic process. Learning that the definitive diagnosis was a benign, curable condition, rather than a terminal malignancy, triggered a moment of immense emotional release. The years of underlying fear and the acute terror of the preceding days were replaced by a powerful sense of gratitude for the care received and a newfound hopefulness for the future. This stage encapsulates the profound positive impact of receiving a good prognosis after bracing for the worst, and it reinforces the ultimate goal of the diagnostic endeavor: to replace uncertainty with clarity and, whenever possible, to replace fear with hope.

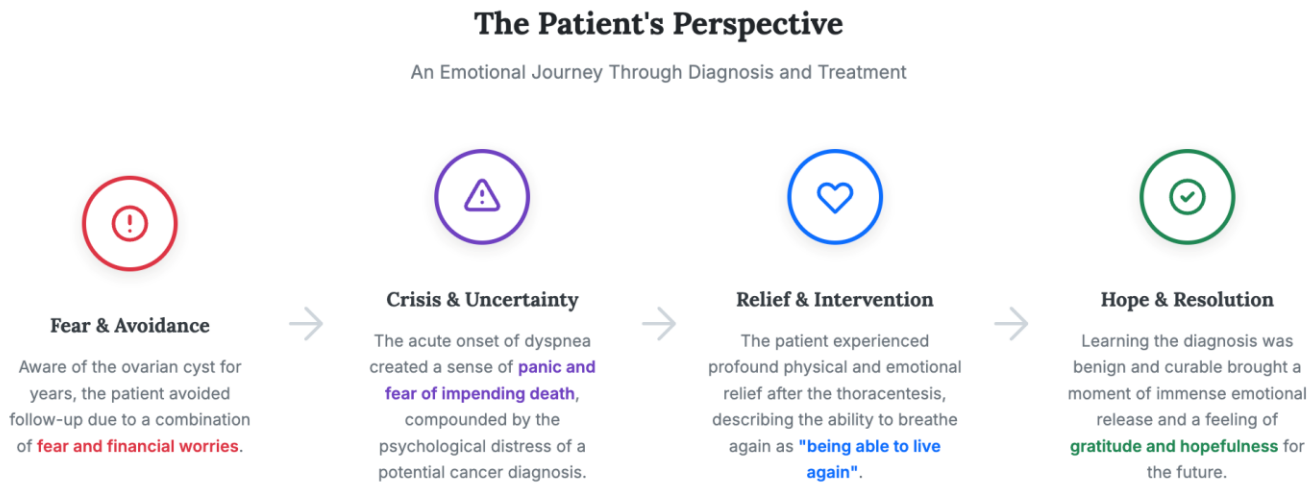

Figure 6. The patient's perspective.

3. Discussion

The presentation of this 47-year-old woman encapsulates the profound clinical drama of Meigs syndrome. This case provides a rich foundation for an exhaustive exploration of the intricate pathophysiology, the nuanced diagnostic challenges, and the logical, stepwise management that defines this rare syndrome, all viewed through the lens of this specific patient's journey.[11] Figure 7 provides a comprehensive, step-by-step deconstruction of the pathophysiological cascade responsible for the clinical presentation of Meigs syndrome, directly linking the underlying biological mechanisms to the specific findings observed in this case. The figure is structured as a top-down flowchart, illustrating a clear cause-and-effect progression from the primary etiology to the terminal clinical symptoms. This visual framework serves to elucidate how a single, benign gynecological tumor can precipitate a complex, multi-systemic syndrome that mimics advanced malignancy. The Benign Ovarian Tumor. The entire pathophysiological sequence is initiated by the presence of a large, solid, benign ovarian tumor. In this patient, this was a fibroma measuring over 16 cm in diameter.[12] Ovarian fibromas are sex cord-stromal tumors composed of spindle-shaped cells that produce a dense collagenous matrix, giving them their characteristic firm and solid structure. Crucially, these tumors are not inert. Despite their benign histology, they are both mechanically significant due to their size and biochemically active, serving as the engine for the entire syndrome. Their large mass exerts direct physical pressure on adjacent structures and the peritoneal lining, while their cellular components are capable of secreting a host of potent vasoactive and inflammatory mediators.[13] This dual nature—mechanical and biochemical—is the fundamental driver of the subsequent pathological events. Divergent Initiation Pathways From the primary tumor, the pathophysiology diverges into two synergistic pathways that ultimately converge to produce the syndrome's characteristic effusions: Pathway A (Mechanical & Inflammatory): This pathway is rooted in the physical properties of the tumor. The sheer size and weight of the >16 cm mass, situated within the dynamic pelvic environment, lead to chronic mechanical irritation of the exquisitely sensitive peritoneal surface. This constant friction provokes a sterile, localized inflammatory response. Furthermore, the tumor's bulk can compress and obstruct the delicate network of lymphatic vessels that line the peritoneum and omentum, impairing their ability to drain physiological fluid from the abdominal cavity. The combination of this sterile inflammation and lymphatic obstruction leads to the slow, steady leakage of fluid into the peritoneal space. Pathway B (Biochemical Mediators): This pathway represents the more modern and molecular understanding of the syndrome. The tumor cells are not biochemically silent; they actively secrete potent signaling molecules into the local microenvironment.[14] The most critical of these is vascular endothelial growth factor (VEGF), a powerful protein that stimulates angiogenesis and, most importantly, dramatically increases the permeability of capillaries. VEGF achieves this by binding to its receptor (VEGFR-2) on endothelial cells, triggering a signaling cascade that disrupts the integrity of the endothelial barrier. This allows not just water but also large proteins like albumin to escape from the bloodstream into the peritoneal cavity. Concurrently, the tumor also releases pro-inflammatory cytokines, such as Interleukin-6 (IL-6). IL-6 further amplifies the inflammatory response and contributes to the increased capillary leakage, while also exerting systemic effects. This biochemical pathway is responsible for the characteristically high protein content of the effusions and the rapid rate at which they can accumulate.[15] Ascites Formation Both the mechanical and biochemical pathways converge on a single, critical outcome: the formation of ascites. The fluid accumulating in the peritoneal cavity is a direct result of both the leakage from irritated surfaces

and obstructed lymphatics (Pathway A) and the high-volume, protein-rich extravasation driven by VEGF and cytokines (Pathway B).[16] In our patient, this manifested as severe abdominal distension, with a measured circumference of 110 cm, and the classic physical sign of shifting dullness on percussion. The exudative nature of her pleural fluid, rich in protein, strongly suggests that the biochemical pathway (Pathway B) was the dominant mechanism driving the fluid accumulation. The Pleural Effusion The development of the pleural effusion is a secondary event, a direct consequence of the pre-existing, large-volume ascites. The diaphragm is not an impermeable barrier but a porous muscular sheet containing microscopic fenestrations and a rich network of lymphatic channels (lacunae) on its peritoneal surface.[17] The massive volume of ascitic fluid dramatically increases the intra-abdominal pressure. This elevated pressure creates a significant hydrodynamic gradient, forcing the ascitic fluid superiorly into these diaphragmatic lymphatics and through the fenestrations. The normal negative pressure within the pleural cavity during inspiration further facilitates this unidirectional flow of fluid from the abdomen into the chest. This mechanism is analogous to that of a hepatic hydrothorax. The characteristic right-sided predominance of the effusion, as seen in this patient, is explained by the greater density and surface area of these lymphatic connections on the right hemidiaphragm.[18] The final stage of the cascade is the presentation of overt clinical symptoms, which are the direct result of the fluid collections and systemic inflammation: Severe Dyspnea: This was the patient's chief complaint, driven by the massive right-sided pleural effusion. The five liters of fluid severely compressed her right lung, causing atelectasis and preventing effective gas exchange. This led to a profound ventilation-perfusion mismatch and the subjective sensation of breathlessness, orthopnea, and pleuritic chest pain. Abdominal Distension: This symptom was a direct result of the massive ascites, which not only caused physical discomfort and a feeling of fullness but also contributed to the patient's early satiety and nausea by compressing the stomach and other abdominal viscera. Weight Loss: The patient's significant, quantified 10 kg weight loss can be attributed to the systemic effects of the inflammatory cytokines, particularly IL-6. IL-6 is a potent mediator of cachexia, promoting a catabolic state characterized by muscle wasting and anorexia. This systemic effect is a crucial element that contributes to the syndrome's mimicry of advanced malignancy.

The pathophysiology of Meigs syndrome is a multifactorial process with the benign ovarian tumor as the primary instigator. The patient's tumor was massive (>16 cm), solid, and had been present for over a decade, providing ample opportunity for both mechanical and biochemical effects to manifest. Ovarian fibromas originate from the sex cord-stromal cells of the ovary, specifically the spindle cells resembling fibroblasts, which are responsible for producing the dense collagenous matrix that gives these tumors their firm consistency. The markedly exudative nature of our patient's pleural fluid and its rapid re-accumulation strongly support the VEGF/cytokine-mediated vasopermeability hypothesis. VEGF-A, a potent signaling protein, is thought to be secreted by the tumor cells. It binds to its receptor, VEGFR-2, on endothelial cells, triggering a signaling cascade that leads to the phosphorylation of junctional proteins like VE-cadherin. This disrupts the integrity of the endothelial barrier, increasing microvascular permeability and allowing protein-rich fluid to extravasate into the peritoneal cavity. This molecular mechanism explains the high protein content of the fluid and the high-flux state leading to rapid ascites buildup. Furthermore, inflammatory cytokines like IL-6 are implicated. IL-6, acting through the JAK-STAT signaling pathway, can further amplify the inflammatory response and contribute to capillary leakage.

Figure 7. Pathophysiological cascade of Meigs syndrome.

Critically, IL-6 is a known mediator of the systemic inflammatory response and cachexia. This provides a direct pathophysiological link to our patient's anorexia and significant 10-kg weight loss. Her cachectic state, therefore, was not necessarily a sign of malignancy but could be explained by the inflammatory milieu created by the biochemically active benign tumor. The development of the massive, right-sided pleural effusion is a direct consequence of the ascites, governed by principles of fluid dynamics. The Starling's forces within the peritoneal cavity were severely altered. The increased intra-abdominal pressure from the mass and ascites significantly raised the hydrostatic pressure, while the high protein content of the ascitic fluid (due to VEGF-mediated leakage) increased its oncotic pressure. This created a powerful gradient driving fluid into the diaphragmatic lymphatics. The negative intrapleural pressure during inspiration further facilitates this unidirectional flow from the abdomen to the chest, a mechanism analogous to that seen in hepatic hydrothorax. The right-sided predominance is due to the greater density of lymphatic lacunae on the right hemidiaphragm. The single greatest clinical challenge is distinguishing Meigs syndrome from its malignant mimic, advanced ovarian carcinoma. However, a robust diagnostic process must consider a broader differential.[19] The patient's age, weight loss, ascites, and pleural effusion are classic for ovarian cancer. On imaging, while our patient's ultrasound showed a 'complex, predominantly solid' mass, the gold standard for characterization is often MRI. On MRI, a benign fibroma classically exhibits very low signal intensity on T2-weighted images due to its dense fibrous content, a feature that distinguishes it from most epithelial ovarian carcinomas (like high-grade serous carcinoma), which are typically more heterogeneous and T2-hyperintense. While the tumor marker CA-125 was not measured, it is a known confounder, often elevated in Meigs syndrome due to peritoneal irritation. Other markers like HE4 are more specific for epithelial cancer, but the definitive test, as in our case, was the negative cytology of the pleural fluid. In many regions, including Indonesia, TB is a critical differential. TB can cause exudative ascites, pleural effusion, and elevated CA-125. Another useful marker in this context is Adenosine Deaminase (ADA), which is typically elevated in TB effusions.[20] However, our patient lacked classic TB symptoms like fever or night sweats, and her pleural fluid was neutrophilic-predominant, whereas TB is classically lymphocytic. The negative GeneXpert NAAT provided strong evidence against this diagnosis.

The immediate, life-threatening problem was respiratory compromise. The cornerstone of management was ultrasound-guided therapeutic thoracentesis. The staged approach, removing 800-1100 mL daily, was deliberately chosen to mitigate the risk of re-expansion pulmonary edema. The use of a diuretic, Furosemide, was adjunctive. Furosemide acts on the Na-K-2Cl cotransporter in the thick ascending limb of the loop of Henle to induce potent diuresis. While not curative for the underlying cause of the effusions, it helped manage the total body volume and peripheral edema, reducing hydrostatic pressure. This required careful monitoring of potential side effects like hypokalemia and hyponatremia. The curative treatment is the surgical removal of the benign ovarian tumor. An exploratory laparotomy was the only appropriate surgical approach for a tumor exceeding 16 cm. The procedure involves a vertical midline incision to allow adequate exposure, aspiration of ascitic fluid for cytology, careful inspection of all peritoneal surfaces, and then resection of the tumor, often via a total abdominal hysterectomy and bilateral salpingo-oophorectomy (TAH-BSO) in a perimenopausal woman. The prognosis following complete resection is excellent, with the ascites and pleural effusion expected to resolve completely and permanently.

4. Conclusion

This case of a 47-year-old woman with Meigs syndrome serves as a powerful and essential clinical reminder. It underscores the principle that even in the presence of a clinical picture suggestive of advanced malignancy—an abdominal mass, massive effusions, and cachexia—a benign and completely curable etiology must be rigorously considered and pursued. The journey of this patient from acute respiratory failure to a stable condition awaiting definitive cure highlights the triumph of careful, staged, multidisciplinary management. The importance of cytological fluid analysis cannot be overstated, as it is often the critical piece of evidence that turns the tide against a presumptive diagnosis of cancer. Meigs syndrome is a testament to the fact that in medicine, the most alarming presentations can sometimes have the most hopeful outcomes, reaffirming the clinician's highest duty: to diligently seek out the curable, even when faced with the seemingly incurable.

5. References

1. Kortekaas KE, Pelikan HM. Hydrothorax, ascites and an abdominal mass: not always signs of a malignancy - Three cases of Meigs' syndrome. J Radiol Case Rep. 2018; 12(1): 17–26.
2. Carver C, Albert B. Massive ascites in the absence of liver disease: a rare case of pseudo-pseudo Meigs' syndrome. Am J Gastroenterol. 2017; 112: S1202–3.
3. Kortekaas KE, Pelikan HM. Hydrothorax, ascites and an abdominal mass: not always signs of a malignancy - Three cases of Meigs' syndrome. J Radiol Case Rep. 2018; 12(1): 17–26.
4. Stabile G, Zinicola G, Romano F, Laganà AS, Pozzolo CD, Ricci G. Pelvic mass, ascites, hydrothorax: a malignant or benign condition? Meigs syndrome with high levels of CA 125. Prz Menopauzalny. 2021; 20(2): 103–7.
5. Li Q, Tian B. Case report: Three cases of systemic lupus erythematosus presenting primarily with massive ascites and significantly elevated CA-125 levels and a review of pseudo-pseudo Meigs' syndrome in literature. Front Immunol. 2024; 15: 1423631.
6. Yıldırım R, Oruç H, Dinler M, Üsküdar Cansu D, Korkmaz C. A challenging diagnosis in a male presented with ascites: Pseudo-pseudo Meigs syndrome and systemic lupus erythematosus. J Turk Soc Rheumatol. 2024; 77–81.
7. Hirata T, Yamada H, Kimura S, Yanai H, Daifu T. Gynandroblastoma with massive ascites as atypical Meigs syndrome. Pediatr Int. 2025; 67(1): e70067.
8. Awad A, Essam M, Ezzat A, El Menyawi M. Systemic lupus erythematosus with lupus nephritis presented with recurrent massive ascites: a case of pseudo-pseudo Meigs syndrome. Arch Rheumatol. 2019; 34(2): 243–4.
9. Compton S, Luebker S, Baker Frost D. Recurrent ascites in systemic lupus erythematosus treated with rituximab - a case report and review of pseudo-pseudo Meigs' syndrome. Beyond Rheumatol. 2021; 3(1).
10. Laan BJ, van den Heiligenberg SM, Hemelaar M. Ascites, pleural effusion and a benign ovarian tumour; the triad of Meigs' syndrome. Ned Tijdschr Geneeskd. 2016; 160: D480.
11. Okuda K, Noguchi S, Narumoto O, Ikemura M, Yamauchi Y, Tanaka G, et al. A case of Meigs' syndrome with preceding pericardial effusion in advance of pleural effusion. BMC Pulm Med. 2016; 16(1): 71.
12. Singh S, Mishra D, Peter DK, Sood M. Anesthetic consideration in Meigs syndrome

with large pleural effusion and lung collapse. J Acute Dis. 2024; 13(1): 43–4.

13. Deniz R, Hacımurtazaoğlu-Demir G, Karaalioğlu B, Özgür DS, Akkuzu G, Yıldırım F, et al. Lupus nephritis presenting with massive ascites and pleural effusion (pseudo-pseudo Meigs' syndrome). J Rheum Dis. 2024; 31(2): 116–9.
14. Vagts C, Tole M, Kovitz K. Meigs syndrome presenting as a Lymphocytic exudative pleural effusion. Chest. 2019; 156(4): A1190.
15. Taniguchi Y, Nishikawa H, Maeda N, Terada Y. Breathlessness, pleural effusions, fibromas, and Meigs syndrome: look beyond the chest and don't delay! Lancet. 2020; 395(10223): e32.
16. Nguyen P, Yazdanpanah O, Schumaker B. Meigs' versus pseudo-Meigs' syndrome: a case of pleural effusion, ascites, and ovarian mass. Cureus. 2020.
17. Hou Y-Y, Peng L, Zhou M. Meigs syndrome with pleural effusion as initial manifestation: a case report. World J Clin Cases. 2021; 9(21): 5972–9.
18. Raghu S, Natarajan U, Shanmuganandavadivel K, Ratnam DB, Kumar PVK, Nikhita AD, et al. Meigs syndrome presenting as bilateral pleural effusion in the reproductive age-group: a rare case report. Indian J Chest Dis Allied Sci. 2023; 65(2): 114–8.
19. Ng BH, Johari How SH, Abeed NNN, Low HJ, Osman RA, Yu-Lin AB. Meigs syndrome presenting with recurrent unilateral pleural effusion. Respirol Case Rep. 2024; 12(7): e01421.
20. Etman A, Aujayeb A. Bilateral pleural effusions, ascites and pulmonary emboli: Meigs syndrome. Breathe (Sheff). 2025; 21(3): 240289.