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Comprehensive Clinical Evaluation, Diagnostic Imaging, and Multidisciplinary Management of Tubo-Ovarian Abscess: Implications for Family Medicine, Nursing Care, and Radiologic Practice

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Abstract

Background: A tubo-ovarian abscess (TOA) is a severe complication of pelvic inflammatory disease (PID), characterized by a purulent collection involving the fallopian tube and ovary. It poses significant risks of morbidity, infertility, chronic pelvic pain, and life-threatening sepsis if not managed promptly.

Aim: This article provides a comprehensive clinical evaluation of TOA, detailing its etiology, risk factors, pathophysiology, and diagnostic approach. It aims to outline evidence-based, multidisciplinary management strategies involving family medicine, nursing, radiology, and gynecology to optimize patient outcomes and preserve reproductive potential.

Methods: The review synthesizes current clinical guidelines and literature on TOA management. Diagnosis relies on a combination of patient history, physical examination (e.g., cervical motion tenderness, adnexal mass), laboratory tests (e.g., leukocytosis, elevated inflammatory markers, STI testing), and imaging, primarily transvaginal ultrasonography and computed tomography.

Results: Initial management with broad-spectrum intravenous antibiotics is successful in 70-87% of cases. For larger abscesses (>5.5 cm) or those not responding to antibiotics within 72 hours, image-guided drainage or surgical intervention (laparoscopy/laparotomy) is required. Multidisciplinary care is critical for success, improving survival and fertility outcomes.

Conclusion: TOA is a serious gynecologic emergency whose prognosis depends on early diagnosis and a tailored, multidisciplinary approach. Timely antibiotic therapy, coupled with appropriate drainage or surgery when indicated, is essential to reduce acute morbidity and preserve long-term reproductive health.

Keywords: Tubo-ovarian abscess, Pelvic inflammatory disease, Multidisciplinary management, Image-guided drainage, Infertility, Broad-spectrum antibiotics..

1. Introduction

Tubo-ovarian abscess (TOA) constitutes a significant clinical entity within gynecologic and reproductive health, representing a severe sequela of pelvic inflammatory disease (PID) and posing considerable morbidity risks to women of reproductive age. While predominantly associated

with sexually active women, TOA may develop independently of overt PID, reflecting the capacity of ascending infections from the lower genital tract to precipitate extensive adnexal inflammation. Pathophysiologically, the condition involves purulent accumulation within the fallopian tubes and ovarian tissue, frequently extending to adjacent pelvic

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structures, including the bladder, bowel, and peritoneum. Prior to the widespread adoption of antimicrobial therapy, TOA-related mortality rates approached 12%, illustrating the potentially lifethreatening nature of this disorder and the critical importance of timely recognition and intervention [1]. Clinically, patients often present with nonspecific symptoms such as lower abdominal pain, fever, and malodorous vaginal discharge, which necessitate a high index of suspicion for accurate diagnosis. Imaging modalities, including transvaginal ultrasonography and computed tomography, play a central role in confirming the presence of fluid-filled, inflamed adnexa or abscess formation, although clinical presentations can be highly variable and occasionally subtle.

The ramifications of TOA extend beyond acute infection, with significant implications for longterm reproductive health. Untreated or inadequately managed abscesses may result in chronic pelvic pain, tubal damage, and infertility, while the disruption of normal tubal anatomy heightens the risk of ectopic pregnancy. The potential for abscess rupture introduces an immediate threat of systemic sepsis, necessitating urgent clinical intervention to mitigate life-threatening outcomes [2][3][4]. Contemporary management protocols advocate for early initiation of broad-spectrum intravenous antibiotics, which often suffice in controlling localized infections. However, therapeutic challenges arise when abscesses exceed a critical size threshold or fail to respond to pharmacologic treatment within a 72-hour window, at which point surgical or image-guided interventions become necessary [5]. Surgical decision-making requires careful consideration of multiple patientspecific variables, including abscess dimensions, age, reproductive goals, comorbidities, and prior surgical history, with laparoscopic and open laparotomy approaches employed according to procedural risk assessment and anticipated outcomes.

Minimally invasive strategies, particularly image-guided drainage techniques, have gained prominence in contemporary practice. Transvaginal aspiration under ultrasonographic guidance offers a high success rate for smaller, well-circumscribed abscesses, presenting a fertility-sparing alternative to traditional surgical approaches [6]. Larger, multiloculated, or complex abscesses may necessitate placement of indwelling drainage catheters or definitive surgical excision. The selection of appropriate management is guided by clinical severity, imaging characteristics, and patient overall health status, with multidisciplinary input from gynecology, radiology, and nursing teams optimizing outcomes [7]. Despite these advances, delayed recognition or suboptimal treatment remains a primary contributor to adverse sequelae. Progression of untreated or inadequately treated TOA can culminate in the requirement for extensive surgical interventions, such

as salpingo-oophorectomy, with potential progression to hysterectomy in extreme cases. These outcomes underscore the criticality of early identification, risk stratification, and individualized treatment planning to preserve both life and reproductive function [8]. In addition to acute management considerations, the condition necessitates a comprehensive, multidisciplinary approach that integrates family medicine, nursing care, and radiologic expertise. Family physicians play a central role in early recognition and initial management, particularly in outpatient or community settings, where nonspecific symptoms may obscure the diagnosis. Nursing professionals contribute essential care coordination, patient education, and monitoring, ensuring adherence to antimicrobial regimens, timely follow-up, and early identification of clinical deterioration. Radiologists provide critical diagnostic and interventional support, facilitating precise characterization of abscess morphology and guiding minimally invasive drainage procedures when indicated. This integrative approach not only enhances patient outcomes but also mitigates long-term complications, emphasizing the importance of collaboration across healthcare disciplines in the management of TOA.

Overall, TOA exemplifies a complex gynecologic emergency with multifaceted clinical, surgical, and reproductive implications. While pharmacologic therapies remain the cornerstone of initial management, the role of surgical and imageguided interventions continues to expand, reflecting advancements in minimally invasive techniques and interdisciplinary collaboration. The prognosis of TOA heavily dependent on timely diagnosis, individualized therapeutic planning, and ongoing monitoring to prevent recurrence or irreversible reproductive harm. Recognizing the interplay between acute infection, reproductive potential, and systemic complications is essential for clinicians across family medicine, nursing, and radiologic specialties, highlighting the necessity for comprehensive, evidence-based, and patient-centered management strategies

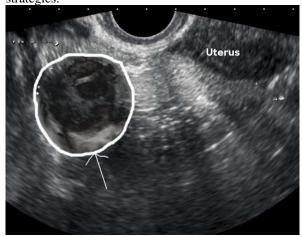


Figure-1: Tubo-Ovarian Abscess.

Etiology

Tubo-ovarian abscess (TOA) primarily arises as a complication of untreated pelvic inflammatory disease (PID), which commonly begins with infection of the lower genital tract. These infections ascend through the endocervical canal into the uterus, fallopian tubes, and ovarian tissue, leading to the development of purulent collections within the adnexa. The pathogenesis of TOA is closely linked to microbial invasion, and infectious agents represent the predominant etiologic factor. However, the spectrum of pathogens implicated in TOA differs from those typically associated with uncomplicated PID, reflecting the severity and complexity of abscess formation. In premenopausal women, sexually transmitted pathogens such as Chlamydia trachomatis and Neisseria gonorrhoeae are frequently reported and are considered significant contributors to adnexal infection [9]. Despite this, studies have demonstrated that enteric and anaerobic bacteria, including Escherichia coli, **Bacteroides** fragilis, Peptostreptococcus species, are more commonly isolated in TOA cases, particularly among postmenopausal highlighting women, the polymicrobial nature of these infections [9][10]. The microbial profile of TOA typically involves a mixture of aerobic and anaerobic organisms, encompassing enteric flora, respiratory tract bacteria, and obligate anaerobes. Polymicrobial infections account for a substantial proportion of cases, with sexually transmitted pathogens representing a smaller component compared to classic PID. Recent research has identified emerging organisms, such as Mycoplasma genitalium, as contributors to PID, raising concerns that conventional antibiotic regimens may not fully eradicate these pathogens and potentially increase the risk of progression to TOA. Although less frequent, rare organisms such as Mycobacterium species and Actinomyces have been documented in isolated TOA cases, usually in immunocompromised patients or in association with specific predisposing conditions [1][11].

Beyond ascending genital infections, TOAs can result from the direct extension of from adjacent pelvic or abdominal organs. Appendiceal infections, particularly in cases of appendicitis with perforation, represent one of the more common non-gynecologic sources of TOA. Similarly, diverticulitis or gastrointestinal perforation may contribute to adnexal abscess formation through contiguous spread. Hematogenous dissemination of pathogens from distant infection sites, although rare, is also recognized as a potential mechanism. Additionally, pelvic malignancies can create local tissue necrosis and immunocompromised microenvironments, facilitating secondary infection and abscess development [12][13]. Several risk factors influence the likelihood of TOA formation following PID. Delayed or incomplete treatment of initial genital infections significantly increases susceptibility. Other factors

include a history of multiple sexual partners, prior episodes of PID, intrauterine device use, and comorbid conditions that impair immune response. The interplay between host susceptibility, microbial virulence, and anatomical predisposition underpins the multifactorial etiology of TOA. Understanding this complex framework is critical for clinicians, as it informs both diagnostic evaluation and selection of appropriate empirical antimicrobial therapy, which must address the polymicrobial nature of these infections while considering emerging and atypical pathogens. Early recognition of potential etiologic factors allows targeted intervention, reducing the risk of abscess progression, complications, and long-term reproductive sequelae. Overall, the etiology of TOA is multifaceted, encompassing ascending infections, polymicrobial interactions, contiguous spread from adjacent organs, hematogenous and associations with dissemination, malignancy. Awareness of this diverse etiologic spectrum is essential for healthcare providers across family medicine, gynecology, nursing, and radiology to ensure accurate diagnosis, timely management, and prevention of adverse outcomes.

Tubo-Ovarian Abscess Risk Factors

Tubo-ovarian abscess (TOA) develops most frequently as a complication of pelvic inflammatory disease (PID), and its occurrence is strongly influenced by the presence of specific risk factors that increase susceptibility to genital tract infections. Age represents a significant determinant, with women under 25 demonstrating a higher risk of both PID and subsequent TOA. Younger women often have an immature cervical mucosa, which is more susceptible to ascending infections, and they may also engage in behavioral patterns that increase exposure to sexually transmitted pathogens. In addition to age, sexual behavior plays a pivotal role. Having multiple sexual partners or a new sexual partner within a short period increases the likelihood of exposure to pathogens such as Chlamydia trachomatis and Neisseria gonorrhoeae, both of which are closely linked to the development of PID and its complications [6][14]. The use of intrauterine devices (IUDs) for contraception is another well-documented risk factor. Although modern IUDs carry a relatively low risk of infection, the procedures involved in device insertion or removal can transiently facilitate the introduction of bacteria into the uterine cavity, thereby increasing the potential for ascending infections and abscess formation. Similarly, invasive uterine procedures, including endometrial biopsy and assisted reproductive technologies such as in vitro fertilization (IVF), may compromise the integrity of the cervical barrier or introduce pathogens directly into the uterine environment, predisposing women to TOA. The manipulation of the endometrium or adnexa during these procedures creates opportunities for microbial colonization and infection, particularly in patients with pre-existing subclinical infections [6][14].

Early onset of sexual activity is an additional behavioral risk factor. Sexual activity beginning before the age of 15 is associated with higher rates of PID and, consequently, TOA, likely due to increased vulnerability of the developing genital tract mucosa combined with behavioral exposures. Unprotected intercourse further compounds this risk by facilitating transmission of sexually transmitted infections (STIs), including C. trachomatis and N. gonorrhoeae, which remain among the most common pathogens implicated in the initial stages of PID. A documented history of STIs significantly elevates the probability of recurrent infections and persistent inflammation, thereby increasing the risk of abscess formation. Recurrent infections can also lead to structural changes in the fallopian tubes, which promote bacterial retention and abscess development [6][14]. Additional factors influencing TOA risk include certain demographic, socioeconomic, and healthcare access variables. Limited access to routine gynecologic care, infrequent STI screening, and delayed treatment of initial infections can all contribute to the progression from uncomplicated PID to TOA. Behavioral and lifestyle factors, such as inconsistent use of barrier contraception and engagement in high-risk sexual practices, amplify exposure to pathogenic organisms. Immunocompromised status, whether due to chronic medical conditions, medications, or other factors, may also increase susceptibility to severe infections, leading to more aggressive disease progression [6][14]. Recognizing these risk factors is essential for clinicians across family medicine, nursing, and gynecology, as early identification allows for targeted prevention, patient education, and timely intervention. Preventive strategies include promoting safe sexual practices, encouraging regular STI screening, providing appropriate counseling regarding IUD use and fertility procedures, and ensuring prompt treatment of genital infections. Understanding the multifactorial nature of TOA risk not only facilitates early diagnosis but also supports multidisciplinary approaches to reduce morbidity, preserve reproductive potential, and prevent life-threatening complications associated with abscess formation. By addressing behavioral, procedural, and medical risk factors, healthcare providers can significantly mitigate the incidence and severity of TOA in vulnerable populations [6][14].

Epidemiology

Pelvic inflammatory disease (PID) represents a significant public health concern in the United States, with prevalence estimates exceeding two million cases annually among females [15]. The distribution of PID demonstrates notable demographic and geographic disparities. Non-Hispanic Black women residing in Southern states are disproportionately affected, reflecting a combination of socioeconomic factors, disparities in access to healthcare, and differences in sexual health education

and resources [15]. The microbial etiology of PID is diverse, with sexually transmitted pathogens such as Chlamydia trachomatis and Neisseria gonorrhoeae responsible for the majority of infections. Commensal vaginal organisms, including anaerobes and other bacterial species, contribute to the pathogenesis in a substantial proportion of cases. Less frequently, respiratory and enteric bacteria are implicated, reflecting the polymicrobial nature of upper genital tract infections [1]. Tubo-ovarian abscess (TOA) arises as a severe complication of PID, with its epidemiology closely tied to the incidence and underlying characteristics of PID Hospitalization data indicate that approximately 15% to 35% of patients admitted for PID develop TOA. underscoring the clinical significance of this complication [14][8]. The wide range reflects variations in study populations, diagnostic criteria, and healthcare access, as well as differences in the timing and adequacy of initial treatment. TOAs primarily affect women of reproductive age, though they can occur across the lifespan. Postmenopausal women account for a smaller proportion of TOA cases, estimated between 6% and 18% [11]. In these patients, abscess formation may be associated with factors such as underlying pelvic malignancy, altered hormonal milieu, and the presence of comorbidities that increase susceptibility to infection [1].

The microbial characteristics of TOA are predominantly polymicrobial, encompassing aerobic, anaerobic, and facultative bacterial species. This complexity contributes to both the severity of clinical presentations and the challenges associated with management. Polymicrobial infections are associated with increased tissue destruction, higher risk of abscess rupture, and greater likelihood of requiring surgical intervention. The contribution of enteric, respiratory, and vaginal flora highlights multifactorial etiology of TOA and underscores the need for broad-spectrum antimicrobial coverage during initial treatment [1]. Advances in diagnostic imaging, early recognition, and the implementation of effective antibiotic regimens have significantly altered the epidemiology of TOA. Mortality rates, which historically were as high as 12% prior to the availability of antibiotics, have declined dramatically and are now estimated at approximately 1 in 740 cases [1]. This decline reflects improvements in medical management, timely surgical intervention, multidisciplinary care approaches that incorporate gynecologists, radiologists, and nursing teams. Early hospitalization and treatment are critical determinants of favorable outcomes, particularly in patients presenting with large abscesses, systemic symptoms, or delayed care [1]. Despite these improvements, TOA remains a clinically important complication of PID with substantial implications for reproductive health and overall morbidity. Infertility, chronic pelvic pain, and increased risk of ectopic pregnancy are frequently

reported among survivors, emphasizing the need for prevention and early intervention. The epidemiologic patterns underscore the importance of targeted public health measures, including STI screening, education on safe sexual practices, and timely treatment of lower genital tract infections, particularly in high-risk populations. Understanding the prevalence, risk distribution, and demographic characteristics of TOA is essential for clinicians in family medicine, nursing, and radiology to optimize early recognition, implement effective treatment strategies, and reduce both short- and long-term complications [1].

Pathophysiology

Tubo-ovarian abscess (TOA) develops through a complex interplay of microbial invasion, host immune response, and anatomic factors that facilitate the spread of infection within the female reproductive tract. The process typically begins with the ascent of pathogenic organisms from the lower genital tract, including the vagina and cervix, into the endometrium, fallopian tubes, and ovarian tissue. Initial epithelial damage, often induced inflammation or prior infections, compromises the natural mucosal barrier, allowing bacteria to penetrate and colonize the upper genital tract [1][6]. Once established, the infection triggers a localized inflammatory response, resulting in the formation of a tubo-ovarian mass composed of edematous tissue, purulent exudate, and infiltrating immune cells. This inflammatory complex often extends to involve adjacent pelvic organs, such as the bladder, bowel, or peritoneum, particularly in cases of delayed treatment or highly virulent pathogens [1][6]. The microbial composition of TOA is typically polymicrobial, reflecting the mixed flora of the lower genital tract, gastrointestinal tract, and, occasionally, respiratory system. Commonly isolated organisms include Escherichia coli, **Bacteroides** Peptostreptococcus species, and, in premenopausal women, sexually transmitted pathogens such as Chlamydia trachomatis and Neisseria gonorrhoeae. Less frequently, atypical organisms, including Mycoplasma genitalium, Mycobacterium species, or Actinomyces, can contribute to infection, particularly in immunocompromised patients or those with specific predisposing conditions [1]. The presence of multiple pathogens amplifies the inflammatory response, promotes tissue necrosis, and increases the likelihood of abscess formation [1][6].

TOA can also arise through alternative pathways, including lymphatic or hematogenous dissemination. Tubercular organisms, for instance, may reach the upper genital tract via these routes, establishing a chronic inflammatory lesion that can evolve into an abscess. Contiguous spread from adjacent pelvic or abdominal infections, such as appendicitis or diverticulitis, represents another mechanism contributing to abscess development. The immune response, while protective, contributes to tissue damage through the recruitment of neutrophils

and macrophages, the release of proteolytic enzymes, and the formation of fibrinous adhesions that encapsulate the infected area, resulting in the characteristic tubo-ovarian mass [1][6]. pathophysiologic progression of TOA underscores the importance of early recognition and intervention. Ascending infections can rapidly lead to systemic involvement, with the risk of sepsis, peritonitis, and long-term reproductive complications such as tubal obstruction, chronic pelvic pain, and infertility. Understanding the mechanisms by which pathogens invade, proliferate, and elicit inflammatory responses provides clinicians with the foundation for targeted antimicrobial therapy, timely surgical or image-guided intervention, and strategies to preserve reproductive function while preventing life-threatening sequelae

History and Physical

The diagnostic evaluation of suspected tuboovarian abscess (TOA) begins with a comprehensive history that recognizes the overlap between TOA and pelvic inflammatory disease (PID) and anticipates atypical or subtle presentations. Clinicians should obtain a clear chronology of symptoms including onset, progression, and character of pelvic or lower abdominal pain, associated systemic signs such as fever or nausea, and any history of abnormal uterine bleeding or changes in vaginal discharge. Sexual history must be taken with sensitivity and precision because epidemiologic risk factors directly affect pretest probability. Record the number and timing of sexual partners, recent new partners, use and type of contraception, history of sexually transmitted infections, prior episodes of PID, and recent gynecologic procedures such as intrauterine device insertion, endometrial biopsy, or assisted reproductive techniques. Medication history and underlying comorbidities that impair host defense also require documentation because they alter both presentation and risk of complications. A history of recent appendicitis, diverticulitis, or other intra-abdominal infection should be elicited since contiguous spread from adjacent organs can produce an adnexal abscess. Because many women present with nonspecific complaints, clinicians should maintain a low diagnostic threshold and probe for subtle indicators of upper genital tract infection that patients might not volunteer spontaneously [6][5][13].

Symptom assessment informs the index of suspicion and guides immediate management decisions. Classically, patients with TOA report persistent lower abdominal or pelvic pain, often unilateral or worse on one side, accompanied by fever and systemic malaise. Vaginal discharge that is malodorous or purulent, abnormal uterine bleeding, and dyspareunia commonly coexist and point toward an infectious process in the reproductive tract. Nausea, vomiting, or right upper quadrant pain may be present in more extensive disease or when the infection irritates the peritoneal surfaces. Because clinical signs

vary widely and some patients with PID or evolving TOA manifest only mild or atypical symptoms, clinicians must weigh epidemiologic context heavily; in populations with elevated STI prevalence the positive predictive value of a clinical diagnosis increases, and empiric treatment should not be delayed when clinical criteria are met [5]. Physical examination complements the history and frequently provides the decisive evidence for initiating treatment. A general assessment should include vital signs to identify fever, tachycardia, or hypotension that suggest systemic inflammatory response or sepsis. Abdominal palpation may reveal focal tenderness, guarding, or peritoneal signs when inflammation extends beyond the adnexa. A careful pelvic examination remains central to diagnosis. Speculum inspection can demonstrate cervical discharge, cervical friability, or visible mucopurulent exudate. Bimanual examination often elicits cervical motion uterine tenderness, tenderness, and adnexal tenderness; these findings, when present in the appropriate clinical context, justify empiric therapy for PID and prompt imaging to evaluate for abscess formation. Palpable adnexal mass or fullness raises concern for TOA, particularly when accompanied by systemic signs such as fever or leukocytosis. Rectovaginal assessment may reproduce adnexal pain and can increase the sensitivity of detecting a posteriorly located collection. Abdominal rigidity, diffuse tenderness, or signs of peritonitis should prompt urgent imaging and surgical consultation given the possibility of abscess rupture [6][5][16].

Laboratory testing augments the history and physical but rarely replaces clinical judgment. A complete blood count often shows leukocytosis with left shift in active infection, although a normal white cell count does not exclude TOA. Inflammatory markers such as erythrocyte sedimentation rate and Creactive protein commonly rise and can help track response to therapy. Pregnancy testing is mandatory in all women of reproductive age to exclude ectopic pregnancy and to guide imaging and treatment choices. Cervical or vaginal nucleic acid amplification tests for Chlamydia trachomatis and Neisseria gonorrhoeae should be obtained because detecting and treating these pathogens affects partner management and may influence outcomes. Blood cultures have limited yield in non-septic patients but become important when fever is high or systemic illness is evident. Urinalysis can identify concurrent urinary tract infection or hematuria that may suggest alternate diagnoses. Microbiologic sampling from the cervix informs targeted therapy when positive, yet the polymicrobial nature of TOA often necessitates broadspectrum empirical coverage that includes anaerobic and enteric organisms [5][10].

Imaging plays a pivotal role when the clinical picture suggests TOA or when diagnostic uncertainty persists. Transvaginal ultrasonography serves as the

first-line modality because it readily identifies complex adnexal masses, fluid collections, and features suggestive of abscess such as thick-walled, septated masses containing echogenic debris. Ultrasound aids in distinguishing simple adnexal cysts from inflammatory collections and helps determine suitability for minimally invasive drainage. Computed tomography of the abdomen and pelvis provides superior delineation of gas-containing collections, involvement of adjacent bowel or urinary structures, and evaluation for other intra-abdominal sources such as appendicitis. CT is particularly useful in atypical presentations, postmenopausal women, or when a broader intra-abdominal pathology is suspected. Magnetic resonance imaging offers high soft tissue contrast and can clarify complex anatomy when surgical planning is necessary, although it is not routinely required for initial diagnosis. Imaging findings must always be correlated with clinical status; small fluid collections may respond to antibiotics alone while larger multiloculated abscesses often require drainage or operative management [6][5].

Differential diagnosis remains broad and includes ectopic pregnancy, complicated ovarian cysts, appendicitis, diverticulitis, endometritis without adnexal involvement, urinary tract infection, and noninfectious pelvic pain syndromes. The concurrence of fever, cervical motion tenderness, adnexal mass, and epidemiologic risk factors favors PID with TOA. Conversely, absence of cervical or uterine tenderness should prompt exploration of alternate causes. Given the potential for rapid deterioration and long term reproductive harm, clinicians should err on the side of early empiric therapy when clinical criteria for PID are met and other acute surgical causes have been reasonably excluded [5][10]. Clinical decision making must integrate the history and physical with laboratory and imaging results to stratify risk and select management. Women who are hemodynamically unstable, show peritoneal signs, or who have evidence of rupture require immediate surgical evaluation. Patients with confirmed or highly suspected TOA who are clinically stable may begin broad-spectrum intravenous antibiotics and undergo image-guided drainage if collections are sizable or fail to respond to medical therapy within an appropriate observation Through evaluation and treatment, window. comprehensive counseling, partner notification, and plans for follow up are essential to prevent recurrence and to address potential fertility consequences. Documentation should reflect the rationale for empiric therapy, the findings that supported escalation to imaging or intervention, and the plan for outpatient follow up if hospitalization is not required [5][13]. In summary, a thorough history and directed physical examination form the backbone of TOA diagnosis. High clinical suspicion, targeted laboratory tests, and timely imaging refined diagnostic accuracy. Prompt initiation of appropriate therapy preserves reproductive potential and reduces morbidity. Interdisciplinary coordination among primary care, gynecology, radiology, and nursing optimizes outcomes and ensures continuity of care in patients with this potentially complex condition.

Evaluation

Laboratory investigation represents the initial pillar of the diagnostic evaluation for suspected tuboovarian abscess (TOA) and serves to exclude competing diagnoses while identifying systemic infection. A urine pregnancy test is mandatory for all women of reproductive potential because pregnancy status determines both differential diagnosis and imaging choices, and it excludes ectopic pregnancy as a life-threatening alternative. Urinalysis complements the pregnancy test by screening for urinary tract pathology that can mimic or coexist with pelvic infection [10][6]. A complete blood count is useful to detect leukocytosis and a left shift that support an active inflammatory process but it lacks specificity for TOA. Inflammatory biomarkers such as C-reactive protein and erythrocyte sedimentation rate frequently rise in acute pelvic infection and can provide objective measures to corroborate clinical suspicion and to follow therapeutic response [5][10]. Focused microbiologic testing is essential. Nucleic acid amplification tests for Chlamydia trachomatis and Neisseria gonorrhoeae should be obtained from cervical or vaginal specimens because laboratory confirmation of these pathogens informs partner treatment and may influence management decisions [5]. A wet mount of vaginal secretions can identify large numbers of white blood cells, trichomonads, or features of bacterial vaginosis that contribute to the clinical picture. Blood cultures have limited diagnostic yield in stable outpatients but should be obtained when fever is high or when systemic sepsis is a concern. When clinical features raise concern for a noninfectious masquerader such as an adnexal malignancy, measurement of tumor markers including cancer antigen 125 and alpha-fetoprotein may assist in differentiation, although these markers lack specificity and must be interpreted in conjunction with imaging and clinical findings [7]. Overall, no single laboratory parameter definitively establishes the diagnosis of PID or TOA, and clinicians must synthesize laboratory results with history and examination to improve diagnostic accuracy [5][10].

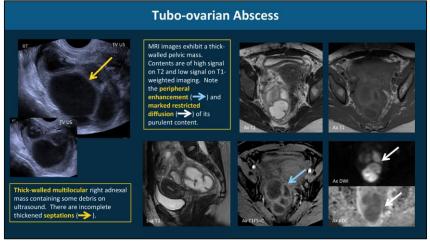


Figure-2: MRI Imaging of Tubo-Ovarian Abscess.

To increase specificity for a clinical diagnosis of PID, the Centers for Disease Control and Prevention recommends incorporating supportive findings such as an oral temperature greater than 101 °F (38.3 °C), mucopurulent cervical discharge or cervical friability on examination, large numbers of white blood cells on saline microscopy of vaginal fluid, elevated ESR, elevated CRP, and laboratory confirmation of cervical infection with N. gonorrhoeae or C. trachomatis [5]. These criteria do not replace clinical judgment but provide a framework for deciding when empiric therapy is indicated and when further diagnostic evaluation is warranted. Clinicians should apply these criteria within the epidemiologic context of the patient because predictive value changes with underlying prevalence of sexually transmitted infections. Imaging is the second principal component of evaluation when TOA is suspected or when the clinical presentation is atypical. Transabdominal and transvaginal ultrasonography are the preferred initial imaging modalities in reproductive-aged women because they are widely available, rapid, and sensitive for detecting complex adnexal collections [17]. Sonographic features that suggest TOA include loss of normal anatomic boundaries due to severe inflammation, a heterogeneous complex adnexal mass, irregular fluidfilled cavities with internal septations, echogenic debris consistent with purulent content, and loculated free fluid in the rectouterine pouch. Thickened and distended fallopian tubes on ultrasound particularly suggestive of a tubo-ovarian process [17]. Ultrasonography also informs procedural planning when transvaginal drainage is contemplated because it defines size, loculation, and accessibility of the collection.

Ultrasound findings are not pathognomonic, and several common intraabdominal inflammatory

conditions, including appendicitis and infectious enterocolitis, may produce overlapping sonographic appearances, prompting the need for additional imaging in ambiguous cases [18][16]. When ultrasound is inconclusive or when clinicians require a broader view of intraabdominal pathology, computed tomography of the abdomen and pelvis is commonly employed. CT characteristically demonstrates a complex solid-cystic adnexal mass with thickened irregularly enhancing walls and septated internal fluid. Surrounding mesosalpinx thickening, dilated fallopian tubes, periovarian fat stranding, and associated findings such as peritoneal free fluid, bowel wall thickening, ileus, or hydronephrosis may also be evident and are useful for identifying contiguous or alternative sources of infection [19][16]. CT is particularly valuable in the postmenopausal population and in patients with atypical presentations in whom pelvic malignancy or non-gynecologic intraabdominal disease is a substantial concern. Magnetic resonance imaging provides superior soft tissue contrast and is recommended when ultrasonographic and CT results remain equivocal or when detailed anatomic delineation is required for surgical planning [17]. MRI better characterizes tubal and ovarian architecture and differentiates abscess from neoplasm through techniques that demonstrate septal and thick rim mucosal enhancement after intravenous gadolinium administration and restricted diffusion within purulent tubal contents on diffusionweighted imaging. These MRI features are helpful in distinguishing complex inflammatory collections from cystic neoplasms and in assessing the degree of inflammation and surrounding tissue involvement [19].

Integration of laboratory and imaging data guides clinical decision making. Imaging that documents a sizable, multiloculated, or poorly accessible collection, combined with persistent systemic inflammation despite appropriate antibiotics, supports consideration of image-guided drainage or surgical intervention. Conversely, small, wellcircumscribed collections without systemic toxicity may be managed initially with broad-spectrum antimicrobial therapy with close clinical and laboratory monitoring. In all cases, diagnostic evaluation must proceed concurrently with empiric therapy when the pretest probability of severe pelvic infection is high, because delay in treatment increases the risk of abscess enlargement, rupture, and long-term reproductive morbidity. Multidisciplinary coordination among primary care, gynecology, radiology, and nursing staff optimizes the interpretation of laboratory and imaging results and aligns diagnostic steps with timely therapeutic actions.

Treatment / Management

Management of tubo-ovarian abscess (TOA) has evolved from radical surgical removal toward a spectrum of conservative, image-guided, and surgical

approaches tailored to disease severity, abscess morphology, patient reproductive wishes, and clinical response to therapy. Initial treatment principles prioritize stabilization, broad-spectrum antimicrobial coverage, and timely reassessment. Hospitalization and prompt gynecologic consultation remain the standard of care for women with a confirmed or strongly suspected TOA because inpatient monitoring allows for serial clinical assessment, laboratory surveillance, and rapid escalation to drainage or operative management when indicated [20]. Daily monitoring of leukocytosis with complete blood counts and serial assessment of inflammatory markers provide objective measures of treatment response and help identify clinical deterioration early [21][22][23].

Intravenous antibiotics comprise the first-line therapy for unruptured TOAs and achieve clinical resolution in the majority of cases when instituted promptly and combined with close observation. Reported effectiveness ranges from approximately 70% to 87% in contemporary series, reflecting advances in antimicrobial regimens and supportive care [8]. The Centers for Disease Control and Prevention (CDC) recommends parenteral regimens that cover enteric gram-negative rods, anaerobes, and likely sexually transmitted pathogens. Preferred intravenous options include ceftriaxone 1 g IV every 24 hours combined with doxycycline 100 mg orally or IV every 12 hours and metronidazole 500 mg orally or IV every 12 hours; cefotetan 2 g IV every 12 hours plus doxycycline 100 mg every 12 hours; and cefoxitin 2 g IV every 6 hours plus doxycycline 100 mg every 12 hours [5]. Doxycycline should be given orally whenever feasible because oral doxycycline offers comparable absorption to the IV formulation and reduces the need for prolonged intravenous access. Transition from parenteral to oral therapy is appropriate once the patient demonstrates clinical improvement, typically within 24 to 48 hours, and completed therapy should extend to at least 14 days in total, with oral doxycycline and metronidazole commonly used to ensure anaerobic coverage [5]. Alternative parenteral regimens, such as ampicillinsulbactam 3 g IV every 6 hours with doxycycline or clindamycin 900 mg IV every 8 hours combined with gentamicin in appropriate dosing strategies, are reserved for patients with contraindications or allergies to first-line agents and are supported by more limited data [5].

A low threshold for invasive intervention must accompany antibiotic therapy. Clinical criteria that prompt consideration of drainage or surgery include failure to improve after 72 hours of appropriate antimicrobial therapy, increasing pain, persistent fever, rising inflammatory markers, hemodynamic instability, or signs that suggest impending or actual abscess rupture [24][8][13]. Approximately one quarter to one third of patients will not respond to antibiotics alone and will require image-guided

drainage or operative management [13]. Predictors of antibiotic failure include larger abscess size, older patient age, marked leukocytosis over $16,000/\mu L$, elevated CRP and ESR, and higher presenting temperature above 38 °C [13][8]. Studies indicate that abscesses with a diameter greater than or equal to 5.5 cm are more likely to require invasive therapy compared with smaller collections, and this dimensional threshold frequently informs clinical decision making when combined with the overall clinical context [8][25].

Image-guided drainage is an effective, fertility-preserving alternative to immediate surgery for many patients. Percutaneous catheter drainage can be performed via multiple routes depending on abscess location, patient anatomy, and the preference and expertise of the interventionalist. Transvaginal aspiration under ultrasound guidance is commonly employed for accessible collections and provides minimal invasiveness with rapid symptom relief and high success rates for appropriately selected abscesses. Transabdominal and transgluteal approaches are frequently recommended because they permit sterile access and facilitate catheter placement for continuous drainage [26]. Other routes, including transrectal, transperineal, and transvesical, are utilized when dictated by abscess position or when more direct access yields better technical success. Choice of route should consider sterility, potential for organ injury, patient comfort, and future fertility considerations. Image-guided drainage allows aspiration microbiologic culture, which can refine antimicrobial therapy when culture results are available, and often shortens hospital stay and avoids the need for major laparotomy in many patients [26].

Surgical intervention remains essential in cases of rupture, refractory sepsis, or when malignancy cannot be excluded. A ruptured TOA or clinical deterioration with peritoneal signs mandates emergent operative management for peritoneal washout, debridement, and control of ongoing infection [20]. The operative approach is determined by disease extent, adhesions, and surgeon expertise. Laparoscopy offers diagnostic confirmation and potential therapeutic drainage or resection with less morbidity than open laparotomy when performed by skilled minimally invasive surgeons. Nevertheless, extensive adhesions, distorted anatomy, and dense inflammatory changes often favour an open approach, and laparotomy may be preferred in many settings to permit safe dissection and thorough washout [25][27][8][26]. For postmenopausal women, surgical exploration is frequently recommended because the risk of an occult pelvic malignancy is higher, and histologic diagnosis may be necessary [7].

The goal of surgery in women of reproductive age is to preserve fertility when feasible. Surgical options include abscess drainage, excision of infected adnexa, or salpingo-oophorectomy when tissue is nonviable, or bleeding persists. For women

who have completed childbearing or when malignancy is suspected, total abdominal hysterectomy with bilateral salpingo-oophorectomy may be considered. Placement of closed suction drains at the conclusion of surgery facilitates postoperative monitoring of ongoing infection and guides the need for further intervention [26]. Intraoperative cultures should be obtained to tailor postoperative antimicrobial therapy, and copious irrigation of the peritoneal cavity reduces residual contamination. Postoperative care includes continued broad-spectrum antibiotics guided by culture results, thromboprophylaxis when indicated, hemodynamic support for septic patients, and early mobilization and physiologic support to reduce morbidity. Decisions concerning fertility preservation require patient-centered discussion that balances the risks of leaving infected tissue in situ against the potential for future childbearing. When feasible, less radical approaches that spare ovarian tissue and preserve tubal anatomy are preferred, recognizing that residual tubal damage may still impair fertility and that patients require counseling about long-term reproductive risks including infertility and ectopic pregnancy. Reproductive endocrinology consultation may be appropriate for women with residual tubal disease who desire fertility.

Adjunctive management includes counseling regarding sexual abstinence until completion of therapy, symptom resolution, and partner treatment. Testing and treatment for sexually transmitted infections such as gonorrhea, chlamydia, HIV, and syphilis are recommended, and repeat testing for gonorrhea and chlamydia is advised at three months or at the next clinical visit to detect reinfection [5]. The role of testing for Mycoplasma genitalium remains uncertain within routine care for TOA. Contraceptive management requires individualized planning. Removal of an intrauterine device in the setting of acute TOA is debated; device removal may be considered when the IUD is believed to be the nidus of infection or when clinical response is inadequate, but routine removal is not universally required if the patient improves on antibiotics [5]. Special populations warrant tailored approaches. Pregnant patients with TOA present unique challenges because imaging choices and antibiotic selection must balance maternal benefit and fetal safety. Multidisciplinary care with obstetrics, gynecology, radiology, and infectious disease specialists is essential in these cases. Immunocompromised patients may exhibit atypical presentations and require broader diagnostic scrutiny and earlier consideration of invasive intervention. Postmenopausal patients merit expedited evaluation for possible malignancy and more aggressive surgical management when indicated [7].

Finally, systems-based measures enhance outcomes. Early recognition through clinician education, prompt access to diagnostic imaging and interventional radiology, availability of experienced minimally invasive surgeons, and robust pathways for

STI screening and partner notification reduce delays in care and lower the likelihood of severe sequelae. Documentation of the rationale for chosen management, clear discharge instructions concerning symptom monitoring and follow-up, and arrangements for reproductive counseling form essential components of comprehensive treatment planning. Integrating these clinical, procedural, and preventive strategies optimizes the balance between effective infection control and preservation of reproductive function in women with tubo-ovarian abscess.

Differential Diagnosis

The presentation of a tubo-ovarian abscess (TOA) often overlaps with a broad range of intraabdominal and pelvic conditions. Pain, fever, leukocytosis, and an adnexal mass are common to many disorders. Clinical differentiation rests on careful integration of history, focused examination, laboratory data, and targeted imaging. The diagnostic challenge lies in separating TOA from other acute and subacute processes that demand distinct interventions, some of which require immediate surgical management while others respond to conservative care. A structured approach reduces diagnostic error and shortens time to definitive treatment. Renal colic from a ureteral stone may mimic TOA when pain localizes to the lower quadrant and radiates toward the groin. Stone pain typically has abrupt onset, severe intensity, and colicky character. Hematuria and absence of pelvic inflammatory signs such as mucopurulent cervical discharge or cervical motion tenderness favour a urologic source. Non-contrast CT of the abdomen and pelvis provides definitive diagnosis of ureteral calculi; ultrasonography and focused urinalysis offer adjunctive data. Imaging that documents a urinary calculus in the setting of a noninflammatory pelvic exam argues against TOA.

Appendicitis classically presents with periumbilical pain that migrates to the right lower quadrant, associated anorexia and vomiting. When the appendix lies in a pelvic location, the inflammatory signs may project to the adnexal region and produce urinary symptoms or diarrhea, creating a close clinical mimic of TOA. Localized peritoneal signs, rebound tenderness confined to the right lower quadrant, and a pattern of pain migration support appendicitis. CT with contrast reliably imaging distinguishes appendicitis and identifies an appendiceal abscess or phlegmon that can extend to the adnexa. When pelvic anatomy is distorted by prior surgery or adhesions, diagnostic laparoscopy often provides the definitive differentiation. Acute cholecystitis and biliary disease may be included in the differential when right upper quadrant pain is prominent. Characteristic features such as a positive Murphy sign, elevations in liver enzymes or bilirubin, and gallbladder wall thickening on right upper quadrant ultrasound support biliary pathology rather than a pelvic abscess. A careful abdominal examination and focused hepatobiliary imaging typically exclude TOA in these cases. Hernias, including inguinal and obturator types, can present with groin pain and a palpable mass and may be mistaken for adnexal enlargement. Incarcerated hernias often demonstrate localized tenderness, visible or palpable groin swelling, and potential signs of bowel obstruction. Clinical inspection and CT imaging clarify the diagnosis and prevent inappropriate pelvic procedures.

Bowel obstruction, diverticulitis, inflammatory bowel disease may produce lower abdominal pain, fever, and leukocytosis. Obstruction classically yields colicky pain, abdominal distension, vomiting, and failure to pass flatus or stool. Diverticulitis typically presents with left lower quadrant pain in older patients and shows segmental colonic wall thickening and pericolic fat stranding on CT. Inflammatory bowel disease often has a chronic course with diarrhea, weight loss, and endoscopic or histologic evidence of mucosal inflammation. These gastrointestinal causes are distinguished from TOA by their bowel-specific signs on imaging and by the absence of pelvic inflammatory markers such as mucopurulent cervical discharge. Other gynecologic conditions require close attention. Ovarian torsion produces sudden, severe unilateral pelvic pain with nausea and often an adnexal mass on ultrasound. Doppler flow studies may show reduced or absent blood flow to the ovary, a key feature that differentiates torsion from an infectious abscess. Ruptured ovarian cyst presents with acute pain and free pelvic fluid but usually lacks systemic infection signs such as prolonged fever or marked leukocytosis. Ectopic pregnancy must be excluded in any woman of reproductive age with pelvic pain; a positive and transvaginal ultrasound pregnancy test demonstrating an extrauterine gestation are diagnostic and necessitate urgent management that differs markedly from TOA care. Lower urinary tract infections and pyelonephritis can mimic pelvic infection when they cause fever, dysuria, or flank pain. Pyelonephritis produces costovertebral angle tenderness and bacteriuria on urinalysis, and it generally lacks pelvic tenderness on bimanual examination. Simple cystitis is usually limited to dysuria and urinary frequency with minimal systemic signs. Careful urinalysis and bladder ultrasound or renal imaging as indicated aid in distinguishing urinary sources from TOA [7].

Pelvic inflammatory disease without abscess formation sits within the same diagnostic spectrum as TOA but differs in severity and management in some cases. PID may present with lower-grade fever, cervical motion tenderness, and adnexal tenderness without a discrete complex mass on imaging. The presence of a complex septated adnexal collection, loculated cul-de-sac fluid, or purulent material on imaging supports a diagnosis of TOA rather than uncomplicated PID. Clinical and radiologic

correlation is essential because PID can progress to TOA, and early empirical therapy for PID is warranted when clinical criteria are met [7]. In all cases, laboratory data and imaging guide differentiation. A pregnancy test is mandatory and changes the differential significantly. Elevated inflammatory markers, leukocytosis, and positive nucleic acid amplification tests for sexually transmitted pathogens increase the likelihood of a pelvic infectious etiology. Transvaginal ultrasonography remains the initial imaging modality of choice for suspected TOA because it identifies complex adnexal masses, internal septations, and loculated fluid. Computed tomography affords broader evaluation when ultrasound is inconclusive or when non-gynecologic intraabdominal pathology is considered. Magnetic resonance imaging may be employed for problem solving in select cases. When diagnostic uncertainty persists or when imaging and clinical signs conflict, diagnostic laparoscopy offers direct visualization, the opportunity for therapeutic drainage, and tissue diagnosis. Diagnostic strategy should prioritize exclusion of life-threatening and time-sensitive conditions such as ectopic pregnancy, ovarian torsion, bowel perforation, and ruptured appendicitis. At the same time clinicians must recognize that TOA can coexist with or arise secondary to several of these disorders. Collaborative evaluation gynecology, radiology, general surgery, and urology as clinically indicated ensures that alternative diagnoses are considered and that the chosen intervention aligns with the underlying pathology. Early and accurate differentiation limits morbidity and preserves reproductive potential when possible [7].

Prognosis

The prognosis for tubo-ovarian abscess (TOA) is generally favorable when clinicians diagnose the condition early and apply appropriate, timely therapy. Most patients show measurable clinical improvement within 24 to 48 hours after initiation of recommended parenteral antibiotic regimens, and many can transition to oral therapy once clinical stability and downward trends in inflammatory markers are evident [5]. Contemporary series report that antibiotic therapy alone resolves TOA in roughly 70 percent of cases, reflecting improvements in broadspectrum antimicrobial protocols and supportive inpatient care [13]. These figures assume prompt presentation, absence of rupture, and close clinical monitoring that permits escalation to drainage or surgery when indicated. The likelihood of therapeutic success hinges on multiple factors that clinicians must assess at presentation. Abscess size influences response to medical therapy. Larger, multiloculated collections correlate with higher rates of antibiotic failure and a greater need for image-guided drainage or operative intervention. Clinical indicators of severe infection, such as sustained high fever, marked leukocytosis, rising C-reactive protein, hemodynamic instability, predict poorer early response and prompt more aggressive management [13][8]. Delay in diagnosis and initiation of effective antimicrobial therapy worsens outcomes. Rupture of an abscess converts a contained pelvic process into diffuse peritonitis and sepsis, increasing morbidity and the need for laparotomy and intensive supportive care. Comorbid conditions that impair host defenses, including diabetes and immunosuppression, also increase the risk of complicated courses and prolong recovery.

Long-term reproductive outcomes represent a distinct component of prognosis and require specific attention during counseling. TOA can cause irreversible tubal damage, pelvic adhesions, and ovarian compromise. Older series reported poor subsequent fertility after conservative management; one study found that only 7.5 percent of patients with TOA achieved pregnancy following their index illness, underscoring the potential for lasting reproductive harm [5][1]. Contemporary practice that integrates prompt drainage with fertility-sparing techniques achieves substantially improved pregnancy rates. Reported pregnancy rates after combined antibiotic therapy and laparoscopic drainage range between 32 percent and 63 percent, indicating that invasive but conservative intervention can restore reproductive potential for a substantial proportion of affected women [25]. These outcomes vary with baseline fertility status, extent of tubal damage at the time of intervention, patient age, and the presence of coexisting pelvic pathology. Clinicians must therefore discuss realistic expectations and consider early referral to reproductive medicine when patients desire future fertility.

Recurrence and chronic sequelae also affect prognosis. Some patients develop persistent pelvic pain or recurrent pelvic infections despite initial resolution. Chronic pelvic pain can diminish quality of life and necessitate long-term pain management, physiotherapy, or additional surgical procedures. The risk of ectopic pregnancy increases after TOA because tubal scarring impairs embryo transport. Providers should counsel patients about these risks and arrange appropriate follow-up, including early pregnancy evaluation in subsequent conceptions. Routine retesting for sexually transmitted infections and partner treatment reduces the risk of reinfection and may improve long-term reproductive outcomes [5]. Interventions influence prognosis in other predictable ways. Image-guided drainage offers a fertilitypreserving option that shortens hospital stay and reduces the need for major laparotomy in many cases. performed for appropriately selected When collections, drainage improves symptomatic recovery and reduces infectious burden while preserving adnexal structures. Conversely, when tissue necrosis, uncontrolled sepsis, or suspected malignancy necessitates resection, definitive surgery such as salpingo-oophorectomy or hysterectomy sacrifices fertility but may represent the only safe option to

control infection and preserve life [20][26]. Postmenopausal women commonly require more aggressive surgical management due to the higher probability of coexisting neoplasm.

Mortality associated with TOA has fallen markedly since the antibiotic era, and deaths are now rare when care occurs in a timely manner and in settings with access to imaging and interventional services. Nonetheless, delayed presentation, abscess rupture, or inadequate resources for drainage and surgical care can still lead to life-threatening sepsis. Early hospital admission, gynecologic consultation, and multidisciplinary coordination improve survival and limit complications. Follow-up practices affect long-term prognosis. Serial clinical assessment and laboratory monitoring during the acute phase detect early treatment failure and reduce the interval to drainage or surgery when needed. Imaging reassessment helps confirm resolution or identify persistent collections that require intervention. Counseling about abstinence until treatment completion, STI testing and partner notification, and fertility implications helps patients engage in protective behaviors and seek timely care for future pregnancies. When reproductive goals exist, timely referral to fertility specialists and consideration of assisted reproductive technologies may mitigate the impact of tubal damage. In summary, TOA carries a good short-term prognosis in the modern era when clinicians provide prompt antibiotic therapy and escalate to drainage or surgery for nonresponders. Long-term reproductive outcomes vary widely. Early recognition, appropriate procedural selection, and coordinated follow-up improve the likelihood of fertility preservation and reduce chronic morbidity. Clinicians should integrate clinical severity, abscess morphology, patient age, and fertility desires when formulating management plans and prognostic counseling [5][13][1][25].

Complications

Tubo-ovarian abscess (TOA) can cause a spectrum of short term and long term complications that affect morbidity and reproductive outcomes. Acute complications include progression to systemic infection and sepsis when the infected adnexal mass breaches local containment. Rupture of an abscess leads to peritonitis and can precipitate septic shock, necessitating emergent operative intervention and intensive supportive care. Bacteremia may follow rupture or persistent deep pelvic infection and increases the risk of distant septic emboli and multi organ dysfunction. These events account for most of the immediate life threatening outcomes of TOA and require rapid recognition and escalation of therapy to drainage, surgical washout, and broad spectrum antimicrobial coverage [25][5]. Local pelvic consequences of TOA produce chronic morbidity through constructive and destructive processes. Persistent inflammation and suppuration generate dense peritoneal adhesions and fibrosis. Adhesion formation distorts pelvic anatomy and alters the spatial relationship between the fallopian tube, ovary, and uterus. This distortion contributes directly to tubal occlusion and impaired gamete transport. The resultant anatomic damage manifests clinically as infertility and as an increased risk of ectopic pregnancy when implantation occurs outside the uterine cavity. Recurrent episodes of pelvic inflammatory disease after an initial TOA compound this risk and worsen the probability of permanent tubal injury [25][5]. Chronic pelvic pain is a frequent long term outcome. Pain arises from adhesions, persistent low grade inflammation, and altered pelvic biomechanics. The pain may be intermittent or constant and can substantially impair quality of life and function. Management of chronic pain often requires multidisciplinary care that includes analgesic strategies, physiotherapy, and sometimes adhesiolysis when symptoms correlate with surgically remediable pathology.

Complications related to treatment warrant careful attention. Invasive interventions and operative management carry procedural risks. Surgical exploration, drainage, or resection can result in bowel injury, particularly when dense adhesions and distorted anatomy increase the likelihood of enteric involvement. Intraoperative hemorrhage is a recognized hazard and may require transfusion or conversion to a more extensive operative procedure. Postprocedural bacteremia and septic shock remain possible despite appropriate perioperative antibiotics, especially when intervention is delayed or the infection is extensive [28]. Allergic reactions to antimicrobial agents present an additional treatment related complication and may necessitate urgent substitution of antibiotics and supportive care. Fertility preserving decisions may produce tradeoffs. Conservative, drainage oriented approaches reduce the need for adnexal resection but may leave residual damaged tubal tissue that continues to impair fertility. Conversely, definitive surgical removal of infected adnexa eliminates the source of sepsis but permanently reduces reproductive potential. Counseling and shared decision making are essential to balance infection control against future fertility goals. Prevention and mitigation of complications rely on early diagnosis, appropriate empiric broad spectrum antibiotics, timely imaging to evaluate abscess size and complexity, and prompt escalation to drainage or surgery when indicated. Serial clinical assessment and laboratory monitoring identify early treatment failure. When invasive therapy is indicated, interdisciplinary coordination with interventional radiology and experienced gynecologic surgeons reduces procedural complications. Finally, testing and treatment of sexual partners and follow up STI screening decrease the risk of reinfection and further pelvic damage [25][5][28].

Enhancing Healthcare Team Outcomes

Effective management of tubo-ovarian abscess (TOA) relies heavily on an interprofessional healthcare team approach due to the condition's complex presentation, which often mimics other intraabdominal and pelvic pathologies such as appendicitis, ureteral stones, cystitis, or obturator hernia. The variable clinical manifestations of TOA, including abdominal pain, fever, and adnexal mass, can delay diagnosis if not promptly recognized, increasing the risk of severe morbidity and potential mortality. Coordinated action among physicians, advanced practitioners, nurses, pharmacists, radiologists, and interventional specialists is therefore critical to optimize patient-centered care, enhance safety, and improve clinical outcomes. Triage and early recognition often begin with the nursing staff in emergency departments or primary care settings. Nurses are usually the first point of contact and must rapidly identify signs suggestive of TOA. This includes assessing fever, pelvic or lower abdominal pain, cervical motion tenderness, and systemic signs of infection. Immediate notification of physicians or advanced practitioners ensures that timely evaluation, laboratory testing, and imaging can be initiated. Early involvement of a gynecologist is critical for confirming the diagnosis and providing recommendations regarding medical or surgical management, particularly in cases of large or complex abscesses.

Radiologists and interventional radiologists play an essential role in both diagnosis and minimally invasive treatment. Transvaginal or transabdominal ultrasonography serves as the first-line imaging modality to identify complex adnexal masses, fluid collections, and loculated abscesses. In cases where inconclusive, ultrasonography is computed tomography (CT) or magnetic resonance imaging (MRI) can provide additional anatomic detail and pathologies. differentiate TOA from other Interventional radiologists contribute by performing image-guided drainage, offering a fertility-preserving therapeutic alternative to open surgery, particularly for abscesses greater than 3 cm in diameter or those unresponsive to antibiotics. Pharmacists and infectious disease specialists ensure optimal antimicrobial therapy. They evaluate culture results, adjust antibiotic regimens based on sensitivity profiles, and counsel both patients and clinical teams on adherence, duration of therapy, and potential drug interactions. Maintaining adherence to prescribed antibiotic therapy is vital for abscess resolution, prevention of recurrence, and reduction of long-term complications such as infertility, chronic pelvic pain, and recurrent pelvic inflammatory disease. Nurses further enhance outcomes by providing patient education regarding risk factor modification. Patients should receive counseling on safe sexual practices, including consistent condom use, limiting the number of sexual partners, and routine STI screening, which

can reduce the incidence of conditions that predispose to TOA. Education also extends to postoperative and post-treatment care, emphasizing medication adherence, recognition of recurrent symptoms, and follow-up appointments to monitor recovery.

Communication within the healthcare team is fundamental for coordinated, high-quality care. Regular interdisciplinary discussions, clear handoffs, and structured updates ensure that all team members are aware of clinical status, treatment plans, and potential complications. Effective communication reduces delays in escalation of care, particularly in urgent scenarios such as abscess rupture, which requires immediate surgical intervention to prevent sepsis and death. In summary, patient outcomes in TOA are significantly improved when care is delivered through a structured, interprofessional approach. Timely recognition by nursing staff, accurate imaging by radiologists, appropriate antimicrobial therapy guided by pharmacists and infectious disease specialists, and surgical or drainage interventions coordinated with gynecologists collectively minimize morbidity. Continuous education, preventive counseling, and ongoing communication within the team reinforce patientcentered care and support both short-term recovery and long-term reproductive health. By leveraging the strengths of each discipline, the healthcare team ensures that TOA management is both effective and comprehensive.

Conclusion:

The management of tubo-ovarian abscess (TOA) requires a sophisticated, patient-centered, and multidisciplinary approach to mitigate its significant acute and long-term risks. While broad-spectrum intravenous antibiotics remain the cornerstone of initial treatment, a substantial proportion of patients, particularly those with larger or complex abscesses, will require escalation of care. The integration of image-guided drainage techniques has revolutionized management, offering a minimally invasive and fertility-sparing alternative to traditional surgery for many cases. However, surgical intervention remains imperative in scenarios of abscess rupture, clinical deterioration, or when malignancy is suspected. The prognosis for TOA has dramatically improved with modern protocols, but its potential to cause permanent sequelae, including tubal factor infertility, chronic pelvic pain, and ectopic pregnancy, underscores the necessity of early recognition and aggressive treatment. Effective outcomes hinge on seamless collaboration across family medicine, emergency medicine, gynecology, radiology, and nursing. From the initial suspicion in an outpatient setting to complex inpatient management and long-term follow-up, this interprofessional coordination is vital for accurate diagnosis, timely intervention, patient education, and prevention of recurrence. Ultimately, a comprehensive strategy that addresses both the acute infection and the patient's future reproductive goals is essential for optimal care.

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