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Abscess

Types, Causes and Treatment

Edited by Selim Sözen

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Meet the editor

Dr. Selim Sözen is an expert in general surgery who received his medical degree from Ondokuz Mayıs University, Turkey, in 1998. From 1999 to 2004, he was an assistant doctor at Ankara Atatürk Education and Research Hospital, Turkey. From 2004 to 2013, he worked as a specialist at different government hospitals in Turkey. He joined the Department of General Surgery, Medicine Faculty, Namık Kemal University, Turkey, as an associate professor in 2013. He completed liver transplantation surgery at İnönü University, Turkey, in 2014–2015. Since 2016, Dr. Sözen has run his surgery clinic in İstanbul, Turkey. He is a member of the Turkish Surgical Association and a review board member for several journals. He has published several journal articles and presented sixty-four poster papers at scientific congresses. His research interests include general, gastrointestinal, emergency, and trauma surgery, bacterial translocation, liver disease, and hernia surgery.

Contents

Preface

This book is a collection of chapters on the types, causes, and treatments of abscesses. In addition to sharing their knowledge, the authors provide their personal clinical experiences, making this book a useful resource for scientists and physicians practicing in the field of abscess treatments.

Chapter 1, "Introductory Chapter: Loop Drainage Technique for Management of Skin and Cutaneous Abscess" by Selim Sözen, discusses skin and cutaneous abscess treatments. The loop drainage technique is a minimally invasive treatment that allows for continuous drainage and eliminates the need for packing change. Chapter 2, "Skin Abscess" by Zekiye Kanat and Selim Sözen, focuses on skin abscesses. The exact incidence of skin abscesses is unknown. Some can become infected and even lead to death, and thus, all skin abscesses should be closely observed and treated with antibiotic therapy if necessary. Chapter 3, "Intraabdominal Abscesses" by Dr. Bashir M. Umar, focuses on intraabdominal abscesses, a serious challenge in surgical practice. The discovery of a wide variety of antibiotics, more aggressive surgical drainage techniques, intensive care management, and other factors have decreased mortality related to intraabdominal abscesses to less than 25% over the past century. Recently, more conservative and less invasive source control techniques have been developed. Chapter 4, "Ovarian Abscess within an Endometrioma: Risk Factors and Management" by Dr. Shashwati Sarkar Sen, discusses ovarian endometriosis, which increases the risk of development of an abscess within the ovary. Chapter 5, "Intracranial and Intraventricular Abscess – Neurosurgical Management" by Dr. Erin McCormack et al., reviews the most common infections of the ventricular system within the neuroaxis, including their source, spread, and clinical presentation. The chapter discusses the neurosurgical management of these patients, including the indications for surgical management, nonoperative management, when an external ventricular drain is indicated, alternative surgical options, and complications.

I thank the authors for their professional dedication and outstanding work in summarizing their clinical and research practice.

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Chapter 1

Introductory Chapter: Loop Drainage Technique for Management of Skin and Cutaneous Abscess

Selim Sözen

1. Introduction

Skin abscess occurs when purulent fluid collects in the dermis and deep tissues. They are painful, tender, and fluctuating red nodules. It is usually polymicrobial. Rarely, bacteremia may progress to septic arthritis and osteomyelitis.

Cutaneous abscess: It is a condition that often follows minor traumas to the skin. It is characterized by the accumulation of purulent material in the dermis and deep tissues. It often starts as an inflamed erythematous papule and turns into painful nodules that are sensitive to palpation and have increased temperature. It is often surrounded by a capsule to differentiate cellulite. It can also be quite large and multiloculated. A mature abscess has an area of thinned skin over which the purulent material accumulated underneath is visible, which can later drain. Although pain is very pronounced, fever does not often accompany uncomplicated abscesses. Fever, lymphatic involvement, rapidly spreading tissue edema, and redness indicate secondary cellulitis [1, 2]. Its etiology involves the flora bacteria of the body region where it occurs and is generally polymicrobial. In 25% of cases, *Staphylococcus aureus* may be the only causative agent.

Skin abscesses are recognized by their appearance. Ultrasound is used in the diagnosis of abscesses that do not fluctuate and are not fully mature in location; Computed tomography (CT) can be used to diagnose abscesses in areas such as deep subcutaneous, intramuscular, neck, and perineum. Primary incision and drainage, followed by systemic antibiotic therapy in complicated cases, are the cornerstones of treatment. In uncomplicated abscesses, the use of antibiotics after drainage is controversial [3]. In a patient with skin abscess, if the body temperature is >38°C or < 36°C, pulse >90/min, respiratory rate > 24/minute, and leukocyte count >12,000/mm^3 or < 4000/mm^3, antimicrobial treatment should be started [4]. It should be investigated whether there is a facilitating factor in recurrent abscesses and culture samples should be taken before treatment begins. In case of relapse, decolonization (e.g., chlorhexidine bath, nasal mupirocin, cleaning of personal belongings, etc.) is also recommended for 5 days.

2. Technique

Incision and drainage technique is primary treatment of cutaneous abscesses. A single incision should be made, long enough to allow full drainage, loculated

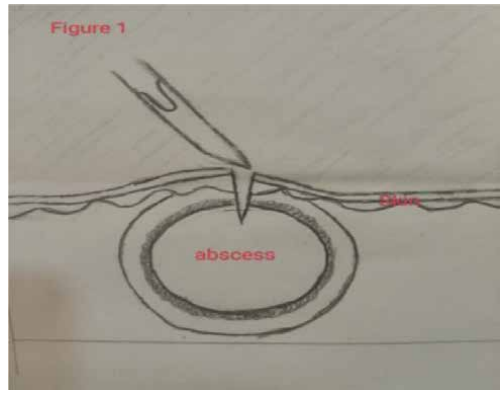

Figure 1.
Incision and drainage technique is initiated in the typical manner with lidocaine injections and incision into the apex of fluctuance. The majority of skin abscesses can be adequately drained through a small incision (average 1 cm).

areas should be disrupted with blunt equipment, and incisions should be made parallel to the natural skin folds to minimize scar formation. Cosmetic results can be optimized if the incision is made parallel to existing skin stretch lines (**Figure 1**) [5, 6]. A common mistake is not making the incision deep enough to allow complete drainage. Gram stain and culture should be performed on the sample taken. In typical cases, treatment can be started without taking a sample. In empirical treatment, it is recommended to start treatment with an agent effective against staphylococci. If there are signs of systemic inflammatory response syndrome (SIRS) or hypotension, unresponsiveness to initial treatment, or impaired host defense, effective treatment for methicillin-resistant *Staphylococcus aureus* (MRSA) should be initiated [4, 7].

Pain and poor cosmetic appearance after healing are significant disadvantages of incision and drainage technique [8–10].

In the loop drainage technique, the provider makes two small 4- to 5-mm incisions around the abscess. A hemostat is used to disrupt the loculations and the vascular loop is then passed and pulled through both incisions (**Figure 2A** and **B**). This technique is less painful than the incision and drainage method. Incisions are small, no packing is required [11, 12]. The loop drainage technique is cosmetically better [11]. Additionally, decrease in cellulite and antibiotic use was found [13]. Thus, health care costs decrease [11, 13]. The loop itself (Penrose vessiloop or even a sterile glove cuff) is removed when the cellulite has resolved, usually after 7–14 days [14].

Gottlieb et al. found that the loop drainage technique resulted in less treatment failure than the conventional incision and drainage technique [15]. However, they suggested more researches are needed [16]. Long et al. reported that the loop drainage approach had a lower risk of treatment failure compared to incision and drainage technique [17]. Hamreus reported that loop drainage provides lower cost, shorter hospital stay, and lower surgery failure rate [18]. According to the retrospective study by Ladd et al., no recurrence or serious morbidity was observed due to the operation. Loop drainage technique is a successful approach to the drainage and treatment of complex abscesses in children [19]. Lautz et al. recommend incision and loop drainage technique in pediatric patients because it is safe and successful in the treatment of subcutaneous abscesses [20].

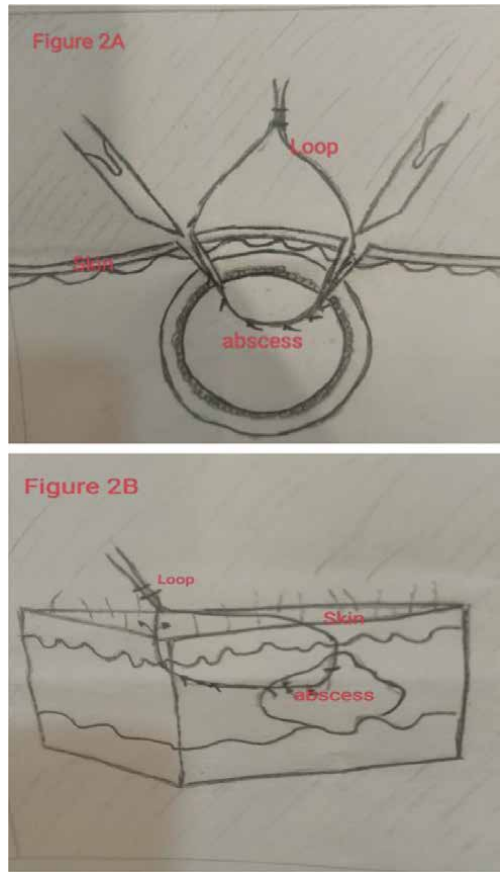

Figure 2.
(A) The provider makes a small incision around the abscess. While clearing loculations with a hemostat, the provider extends the hemostat to the other side of the abscess (opposite from the initial incision) and makes a second incision at that site; (B) Then, a sterile rubber tube—Or "loop"—Is grabbed by the hemostat, looped through the wound, and tied off.

3. Conclusion

In conclusion, loop drainage technique is better in the treatment of skin and soft tissue abscesses in terms of the overall failure rate (need for repeated incisions and drainage, need for use of intravenous antibiotics, and need for hospitalization or surgical intervention) in pediatric patients. However, results in adult patients are controversial [17]. The loop technique is a minimally invasive treatment of abscesses that allows for continuous drainage and eliminates the need for packing change.

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Chapter 2

Skin Abscess

Zekiye Kanat and Selim Sözen

Abstract

Our skin, which is the largest organ of our body, is one of the organs most prone to abscess formation. They are infections that develop as a result of microbial invasion and inflammation of the epidermis, dermis, and subcutaneous tissues. Among these infections, we often encounter folliculitis, furuncles, and carbuncles. We often see gram-positive microorganisms such as staphylococcus and streptococcus found on the skin as causative agents. Although the treatment of these infections varies depending on the patient's clinic, it is generally provided with topical or systemic antibiotics. Most of the time, the clinic goes smoothly, but if neglected, it can cause serious problems.

Keywords: epidermis, dermis, folliculitis, carbuncles, subcutaneous tissue, bacterial infection

1. Introduction

As the largest organ in the body, the skin's ability to protect against viruses, control body temperature, and detect touch is among its most crucial capabilities. The epidermis, dermis, and hypodermis are the three primary layers of skin. Numerous issues, including cancer, acne, wrinkles, and rashes, can affect the skin [1].

1.1 What is skin?

The largest organ of the body, the skin is composed of water, protein, fat, and minerals. It controls body temperature and shields your body from pathogens. Skin nerves enable us to experience emotions like heat and cold. The skin, along with hair, nails, sebaceous glands, and sweat glands, is a component of the entire system. The term "skin" refers to the body's outer layer [2, 3].

1.2 What are the layers of skin?

The skin consists of three layers of tissue [4]:

1. Epidermis (top layer)

2. Dermis (middle layer)

3. Hypodermis (lower or fatty layer) (**Figure 1**)

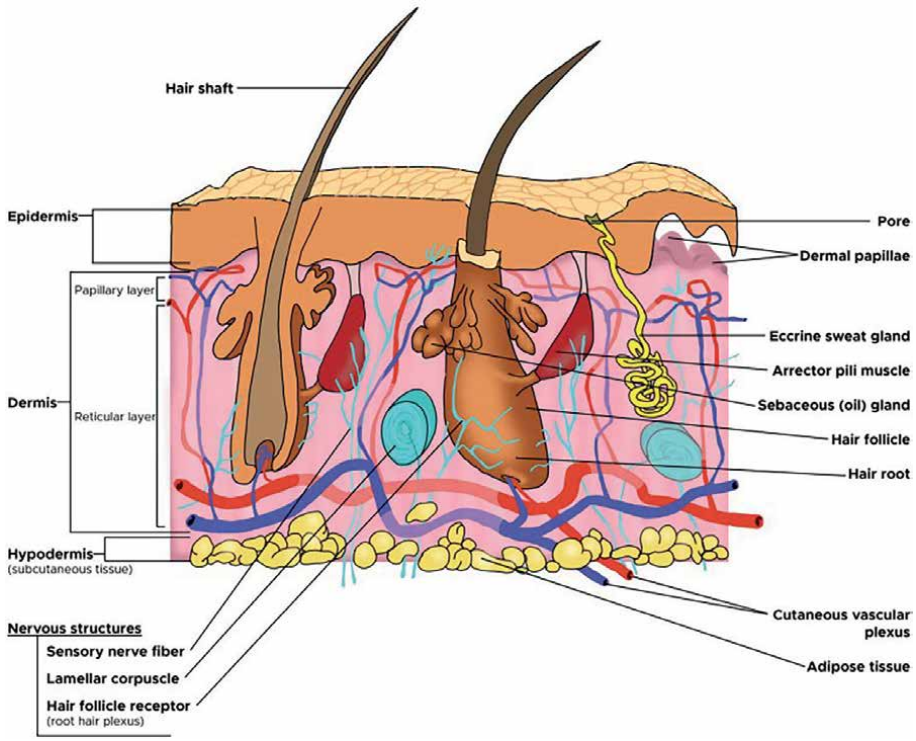

Figure 1.
Layers of skin [5].

1.3 What does the epidermis (top layer of skin) do?

Your epidermis is the top layer of your skin that you can see and touch. Keratin, a protein inside skin cells, makes up the skin cells and, together with other proteins, sticks together to form this layer [6–8].

Epidermis:

- Serves as a barrier of protection: The epidermis serves as a barrier to stop bacteria and germs from infecting your body and bloodstream. In addition, it shields from the sun, rain, and other elements.

- Creates new skin: The epidermis continually generates fresh skin cells. Your body eliminates about 25.000–40.000 old skin cells per day, which are replaced by these new ones. Every 30 days, your skin is renewed [6–8].

- Safeguards your body: The epidermis contains Langerhans cells, which are a component of the immune system. Infections and bacteria are fought off by them.

- Provides skin color: Melanin, the pigment that gives skin its color, is found in the epidermis. The color of your skin, hair, and eyes depends on how much melanin you have. The skin tone and rate of tanning are darker in those with more melanin production [6–8].

1.4 What is the dermis (middle layer of the skin) for?

The dermis makes up 90% of the skin thickness. This middle layer of skin

- Contains collagen and elastin: Collagen is a protein that makes skin cells strong and durable. Elastin, another protein found in the dermis, keeps the skin supple. It also helps stretched skin to take its old shape.
- Grows hair: The roots of the hair follicles are connected to the dermis.
- Keeps you in touch: Nerves in the dermis let you know when something is too hot, itchy, or too soft to touch. These nerve receptors also help you feel pain.
- Makes oil: Sebaceous glands in the dermis help keep the skin soft and smooth. Oil also prevents your skin from absorbing too much water when you swim or get caught in a rainstorm.
- Produces sweat: Sweat glands in the dermis release sweat through the skin pores. Sweat helps regulate your body temperature.
- Produces blood: Blood vessels in the dermis supply nutrients to the epidermis, keeping the skin layers healthy [1–9].

1.5 What does the hypodermis (bottom layer of the skin) do?

The bottom layer of the skin, or hypodermis, is the fatty layer. Hypodermis:

- Cushions muscles and bones: The fat in the hypodermis protects muscles and bones from injury when you fall or have an accident.
- Connective tissue: This tissue connects layers of skin to muscles and bones.
- It helps nerves and blood vessels: Nerves and blood vessels in the dermis (middle layer) grow in the hypodermis. These nerves and blood vessels branch out to connect the hypodermis to the rest of the body.
- It regulates body temperature: The fat in the hypodermis prevents you from getting too cold or hot [1–9].

2. Skin abscess

Skin abscesses are caused by the accumulation of pus in the dermis or subcutaneous tissue and have a swollen, red, tender, fluctuating appearance, often accompanied by surrounding cellulitis [10]. The diagnosis of skin abscesses is based on physical examination, but rarely an ancillary technique such as ultrasonography may be used.

Healthy human skin has many physiologic barriers against microorganisms. The stratum corneum provides mechanical protection with its covering layer. Desquamation, which develops as a result of the skin's continuous self-renewal, also provides the removal of colonized bacteria.

Skin abscess formation is of two types: under the skin or above the skin. Although skin abscesses affect people of all ages, they are usually caused by bacterial infection. Bacterial infections of the skin are among the most common infections in the community. Roughly 20% of outpatients in dermatology outpatient clinics are diagnosed with bacterial skin infections. Skin abscess formation can occur anywhere on the body. The treatment of bacterial skin infections, defined as pyoderma, can be simple drainage or severe enough to require intensive care conditions, sometimes even resulting in death [11–13].

The most frequently isolated pathogenic microorganisms in bacterial skin infections are *Staphylococcus aureus* and *Streptococcus pyogenes*. Cutaneous and superficial abscesses are the most common skin diseases seen by physicians. In parallel with the increase in community-acquired MRSA (Methicillin-Resistant *S. aureus*) infections, the incidence of skin abscesses has also increased. MRSA infection is the most common cause of skin abscesses [11–13].

Many bacteria may be responsible for the formation of skin abscesses. When evaluated under main groups, it can be classified as infection of gram-positive bacteria, gram-negative bacteria, and other microorganisms. As a result, many diseases with different clinical presentations and courses are observed in the skin. However, the most common factors observed in abscesses are streptococci and staphylococci, which are gram-positive bacteria.

2.1 Classification

Different classifications can be made in its simplest form;

1. Light
2. Middle
3. Severe.

2.2 Risk factors

Risk factors can be skin-related or systemic diseases. In addition, other factors such as diet, clothing habits, and cleaning habits may also predispose to infections. The most common skin-related factors are, of course, conditions that disrupt the integrity of the skin such as incisions, burns, trauma, and insect bites. Again, in itchy skin diseases such as atopic dermatitis and contact dermatitis, the loss of the protective function of the skin due to erosions caused by scratching may prepare the ground. Skin diseases such as chickenpox and pemphigus, in which the integrity of the skin is lost, also pose a risk [14]. Other risk factors are given in the **Table 1**.

2.3 Abscess symptoms

- A hard or palpable soft swelling under the skin
- Pain and tenderness in the affected area
- Increased temperature and redness in the same area

• Trauma
• Steroid therapy
• Chemotherapy
• Chronic Diseases (DM, morbid obesity, malignancy, etc.)
• Immune Deficiencies (AIDS etc.)
• Sickle cell disease
• Peripheral vascular disorders, vascular insufficiency (lymphatic, venous)
• Inflammatory Bowel Diseases (Crohn's disease, Ulcerative colitis)
• Serious burns
• Alcoholism
• IV drug use
• Poor hygiene
• Some Chronic diseases
• Animal or human bites
• Fungal infections of the skin
• Organ Failures (liver/kidney failure)
• Surgical procedures that disrupt lymphatic drainage
• Fever above 38°C in infants

Table 1.
Risk factor [14].

- Visible accumulation of white or yellow infectious material under the skin
- Systemic fever
- Tremor

2.4 Types of skin abscess

There is a confusion in the definition of skin abscess. In many places in the literature, bacterial infections of the skin are also examined under this title. However, in this article, we will talk about lesions and conditions with abscess clinics. Among these, folliculitis, furuncle, and carbuncle are related to the skin and we will also mention pilonidal abscesses that can be considered as skin abscesses.

2.5 Differential diagnosis of skin abscess

- Chickenpox; Varicella
- Herpetic İnfections
- Ruptured Epidermoid cyst
- Hidradenitis Suppurativa
- Skin Tuberculosis

- Deep fungal infections
- Lymphadenitis
- Pilonidal Sinus Infection

3. Folliculitis

Folliculitis is an inflammation of the hair follicles. Anywhere there is hair on our body, it can grow there. It is a typical skin condition. Since it frequently does not require medical attention and might disappear on its own, it is challenging to determine the exact incidence. Usually, a simple skin inspection is sufficient for diagnosis. Sometimes it is possible to perform a culture and antibiogram.

An injury or irritant may be the cause (infection or non-infection). Bacterial or non-bacterial (viral, fungal, parasitic) infectious folliculitis can develop in either the superficial or deep region of the hair follicle. Normal causes of non-infectious folliculitis include follicular damage or blockage. They are painful sores that frequently contain pus. When the inflammation around the hair follicles deepens, a boil develops [15–17].

Most frequently, the bacterium *S. aureus* is the responsible party. Multiple groups of tiny, elevated, itchy, erythematous papules, often less than 5 mm in diameter, are the hallmarks of bacterial folliculitis. The onset is frequently acute, and pustules may be visible.

Depending on which area of the hair follicle is affected, folliculitis can either be superficial or profound. Neutrophils invading the area around the hair follicle is typically a sign of acute bacterial folliculitis. Neutrophils are restricted to the infundibulum in superficial folliculitis; in deep folliculitis, they invade the deeper portion of the follicle and the surrounding dermis. Later stages of the lesions display persistent granulomatous inflammation with enormous cells that contain fragments of hair and keratin. In general, infectious folliculitis is more susceptible to therapy than folliculitis brought on by non-infectious reasons, and superficial folliculitis is easier to treat than deep folliculitis.

It is important to distinguish between bacterial folliculitis and other infectious causes of facial folliculitis, such as viruses, fungi, and parasites, such as Demodex folliculorum, as well as bacteria like Candida and Pityrosporum. We must take into

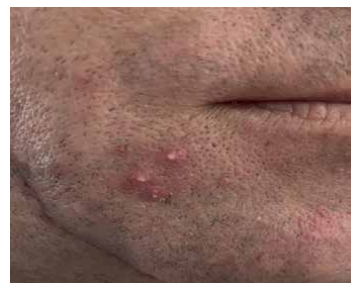

Figure 2.
Folliculit.

account the etiology, severity, and anatomical distribution when handling folliculitis. Warm normal saline compresses (one teaspoon table salt to two cups tap water) used topically, followed by bacitracin or erythromycin ointment and sterile absorbent gauze bandages, are effective treatments for many types of folliculitis. A 7–10 day regimen of oral erythromycin (250–500 mg/day for adults and 30–50 mg/kg/day in evenly divided groups) may be used for moderate S aureus infections [15–17] (**Figure 2**).

4. Furuncle—carbuncles

A furuncle is an infection of the hair follicles that spreads to the surrounding skin and underlying deep subcutaneous tissue. They often present as a painful, swollen lump covered in many sinus tracts or pustules. A contiguous group of two or more furuncles is known as a carbuncle. Regional lymphadenopathy is a possibility, and systemic symptoms are typically present. Although they can appear on any area of the body with hair, they are more frequently found in places with thick skin, such as the back of the neck, the back, and the thighs. There may be one or several carbuncles [18, 19].

Bacteria can enter the sebaceous gland duct and hair follicle, resulting in clinical symptoms ranging from a straightforward infection to a serious and life-threatening condition. Carbuncles are brought on by a bacterial infection of the hair follicle. *S. aureus* is the most frequent culprit and frequently includes methicillin-resistant *S. aureus*. Sometimes, particularly in recurring cases involving the anogenital region, anaerobic bacteria might be the source of carbuncles.

On typically healthy skin, *S. aureus* can be discovered, most frequently in intertriginous regions like the groin, axillae, buttocks, and neck. Additionally, the nostrils may also contain it. Scratching can spread *S. aureus* to several anatomical locations. Bacteria can infect the hair follicle when the skin's protective barrier is breached. The bacteria can grow after being inoculated and result in folliculitis, boils, and/or carbuncles.

4.1 Who is more likely to have a froncle/carbuncle?

- Elderly
- Obese
- People with diabetes
- Individuals with weak immune systems
- People living in poor hygienic conditions
- People living in hot and humid climates
- People with chronic skin diseases
- People with kidney and liver disease

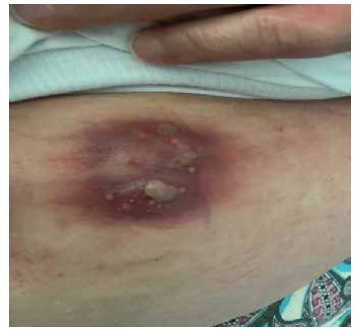

Figure 3.
Carbuncle.

They are mostly found in young to middle-aged adults and are rare in early childhood. Carbuncles are known to affect men more than women. Disruption of the skin barrier can be caused by eczema, diabetes, alcohol abuse disorder, malnutrition, immune deficiency, obesity, and poor hygiene [20] (**Figure 3**).

4.2 Diagnosis

A patient presenting with a carbuncle typically has a history of a tender nodule that grows slowly. According to the patient, the lesion initially appeared as a "pimple" or pustule that they attempted to pop, but over the course of a few days to weeks, it grew increasingly larger. It is well recognized that carbuncles can induce systemic symptoms, but this is not necessary for diagnosis. Regional lymphadenopathy, fever, tiredness, and malaise are examples of systemic symptoms.

Carbuncles are often diagnosed based on the results of a physical examination. It's crucial to collect a bacterial culture and sensitivity test from the purulent fluid contained within the carbuncle when one is suspected of having one. Before beginning antibiotic treatment, a bacterial swab should be done. To direct antibiotic therapy and rule out MRSA or any other gram-negative bacteria as the cause of the illness, bacterial cultures, and sensitivities are crucial [16–20].

4.3 Treatment

The patient should be provided with rest and monitored at short intervals. Typically, carbuncles necessitate both medicinal and surgical treatment. Typically, carbuncles are drained and incised while under local anesthetic. If the abscess is immature, on the other hand, a warm dressing is used to assist it in developing before an incision is made to drain the adult abscess. It is common to utilize antiseptics, antibiotic creams, oral broad-spectrum antibiotics, and analgesics.

Typically, oral antibiotics are started following incision and drainage. Dicloxacillin and cephalosporins are typical first-line oral antibiotics. Oral antibiotics such as clindamycin, tetracyclines, trimethoprim-sulfamethoxazole, linezolid, or glycopeptide may be taken if MRSA is suspected or cultivated. As an additional treatment, topical antibiotics like clindamycin or mupirocin may be applied.

Septicemia, cavernous sinus thrombophlebitis, and scarring are possible side effects of carbuncles. Patients must be given information on how to avoid developing carbuncles, including how to maintain excellent hygiene, lose weight, manage

their diabetes, eat well, and receive suitable treatment for any underlying illnesses or immunological deficiencies. Furthermore, by administering mupirocin to the interior nostrils twice daily for 12 to 30 days, staphylococcal decolonization of the nostrils can be accomplished [18–21].

5. Approach to recurrent conditions

- Local causes related to the site of infection should be reviewed [20];
- If there is the presence of foreign body, pilonidal cyst, hidradenitis suppurativa, etc., treatment for these conditions
- Abscess must be drained
- In recurrent abscesses, culture should be taken and a 5–10-day treatment should be organized with an antibiotic that is effective for the isolated pathogen.
- Personal hygiene and decontamination can be performed
- Intranasal treatment can be given

6. Conclusion

The exact incidence of skin abscesses is unknown. Because the patient may go from a minor illness to a situation that can lead to death. When the clinician detects an abscess on the skin, he/she should observe it closely and start antibiotic therapy if necessary. It should not be forgotten that a small, oversimplified abscess focus may cause trouble for the patient and the physician for months. At the same time, patients should also consult a physician.

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Chapter 3

Intraabdominal Abscesses

Bashir M. Umar

Abstract

Intraabdominal abscesses continue pose challenge to surgeons due to their rather ominous presentation. Most often, high index of suspicion is required to initiate a diagnostic workup. The discovery of a wide variety of antibiotics, more aggressive surgical drainage techniques, intensive care management, and other factors have decreased mortality below 25% over the past century. Recently, more conservative, less invasive source control techniques have recently been developed. These include laparoscopic, needle, and percutaneous image-guided drainage techniques. This chapter will discuss the epidemiology of intraabdominal abscesses including frequency of occurrence of the various types and age and sex distribution. Relevant anatomy of the peritoneal cavity will be given. Risk factors will be discussed. Classification will include intraperitoneal, solid organ abscesses and retroperitoneal abscesses. Pathology and pathophysiology of the various types of abscesses will be discussed followed by clinical presentation, morbidity and complications. Aetiology and bacteriology of the abscess types will be elaborated. A discussion will be provided on resuscitation, patient evaluation and preoperative workup. Management will be discussed as follows: Overview, percutaneous image-guided drainage vs. open drainage, (including indications, anaesthesia, access and technique of drainage), pearls and postoperative/postprocedural care and complications. Prognosis of each abscess type will be given.

Keywords: abscess, intra-abdominal, intraperitoneal, retroperitoneal, pyogenic, amoebic, splenic, image-guided drainage

1. Introduction

Intra-abdominal abscesses can result from a number of conditions, including perforated hollow viscera, de novo intraabdominal infections, and following operative abdominal procedures [1]. Abscesses are well-defined collections of infected, purulent material which may be walled off from the remainder of the peritoneal cavity by inflammatory adhesions, loops of intestine and their mesentery, the omentum, or other abdominal viscera. Abscesses can develop within the peritoneal cavity as well as in the retroperitoneum. It may also occur within the tissues of intraabdominal solid organs [2].

The peritoneal cavity, tissues of intaraabdominal solid organs and the retroperitoneal space are normally sterile. A disruption of the gastrointestinal tract, external inoculation, such as through a penetrating abdominal injury, or seeding of blood-borne pathogens can cause intraabdominal infections and abscess formation. Less

frequently, abscess may form from the urinary or gynaecological tract. The majority of abdominal infections are polymicrobial [3]. Prompt detection and treatment help to lower the high morbidity and mortality associated with this condition.

In the past, complicated intra-abdominal infections, such as peritonitis or intra-abdominal abscesses, were linked to high fatality rates of 90% or more. The discovery of a wide variety of antibiotics with various modes of action, more aggressive surgical drainage techniques, intensive care management, and other factors have decreased mortality below 25% over the past century [4]. More conservative, less invasive source control techniques have recently been developed. These comprise laparoscopic, needle, and percutaneous image-guided drainage methods.

2. Epidemiology

The incidence of intraabdominal absesses was reported to be 7% for of all patients managed for intraabdominal infections by an international study involving 309 hospitals across the world in 2016. The reported organ failure and septic shock as the commonest causes of death [5]. It is estimated that about 70% are postsurgical and that 6% of patients undergoing colorectal surgery may develop a postoperative abscess [3]. Barie *et al*. reported an incidence of intra-abdominal infection of 5.75% among 465 critically ill surgical patients managed for hollow viscus perforation and/or peritonitis from 1991 to 2002 with a mortality rate of 22.6% [6].

Hepatic abscesses constitute about 13% of all intra-abdominal abscesses. Due to its bigger size and better blood supply, the right lobe of the liver is where most of hepatic abscesses occur. According to a population-based research, hepatic abscesses have a yearly incidence of 3.6 cases per 100,000 people in North America with a male-to-female ratio of about 1.5 to 1. According to the study hepatic abscesses have no racial, or regional disparities in occurrence. Pyogenic liver abscess is commonly associated with cirrhosis, chronic renal failure, and a history of cancer [7]. Over the years, the average age of patients with pyogenic liver abscess has risen. With a reported mean age of 47 to 65 years, it is currently a condition that primarily affects middle-aged and older people. Risk factors for case fatality in older patients include age and an APACHE II (Acute Physiology and Chronic Health Evaluation II) score below 15 on admission. Pyogenic liver abscesses frequently develop in children who have impaired immune function, sickle cell anaemia, congenital hepatic fibrosis, polycystic liver disease. Hepatic abscesses may complicate liver transplantation [8].

Splenic abscess is an uncommon infection. The incidence of splenic abscess in autopsy series is reported to be 0.05–0.7%. Splenic abscess mostly follows haematogenous spread. It also results from endocarditis or seeding from some contiguous sites of infection. Other risk groups include immunosuppressed patients, hemoglobinopathies, and diabetes mellitus [9]. It is mostly solitary but can be multiple. Splenic abscess may coexist with extrasplenic abscesses including liver abscesses [10].

3. Surgical anatomy

The peritoneum, which is composed of a single sheet of simple squamous epithelium of mesodermal origin, is situated deep to the extraperitoneal fascia and fat. The peritoneal cavity is sealed in men, but it is exposed to the outside in females through the ostia of the fallopian tubes. The peritoneum has parietal and visceral

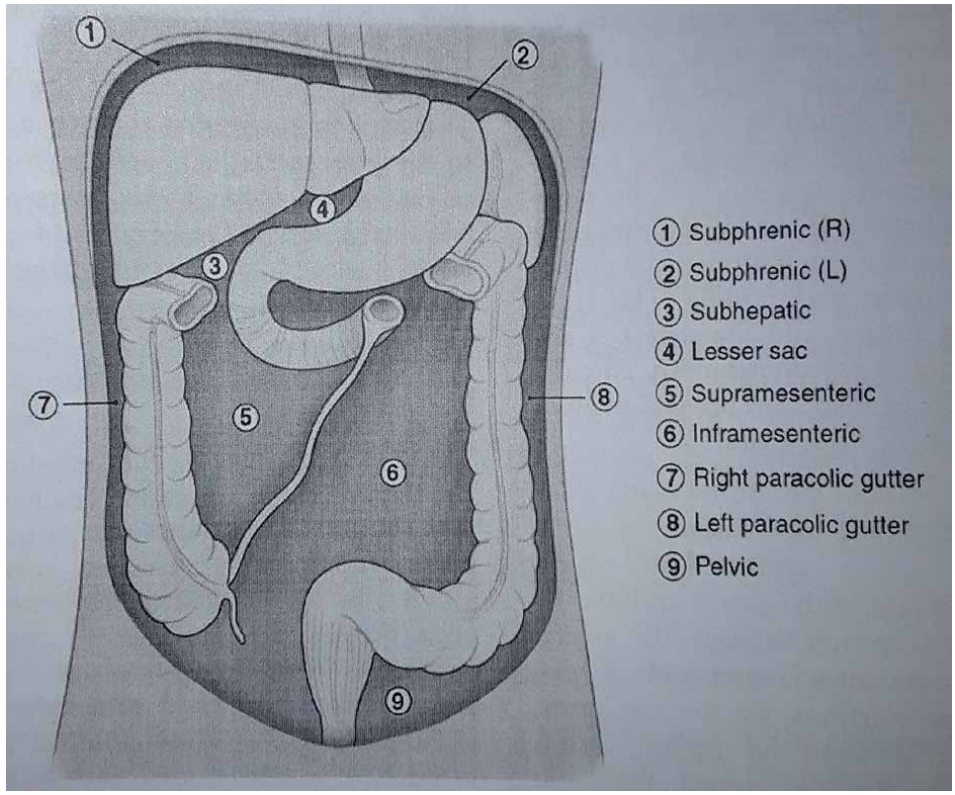

Figure 1.
Peritoneal cavity and its potential spaces [11].

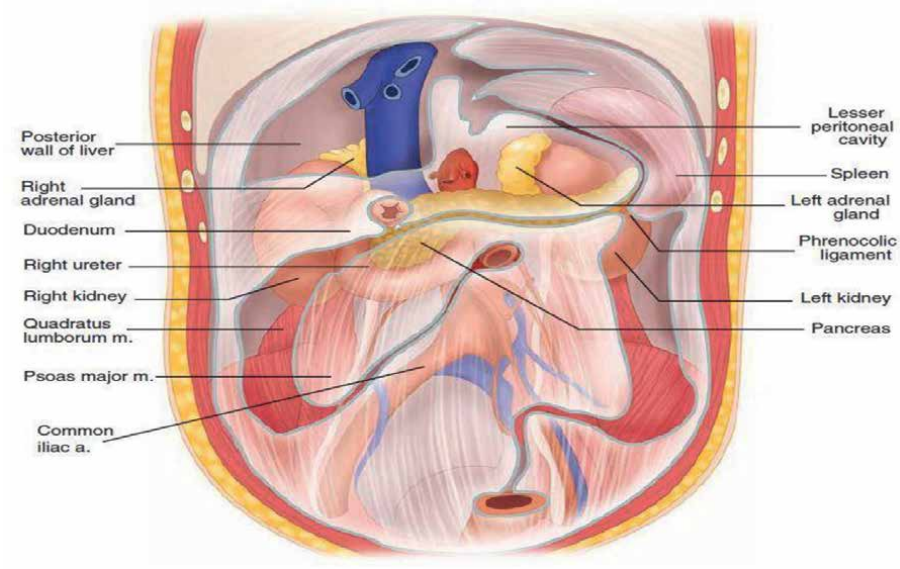

Figure 2.
Anatomy of the retroperitoneum [14].

components. The parietal peritoneum is the mesothelial layer that lines the inferior surface of the diaphragm, the anterior, lateral, and posterior abdominal wall surfaces, as well as the pelvis. The stomach, jejunum, ileum, transverse colon, liver, spleen, and the anterior portion of the retroperitoneal organs (duodenum, left and right colon, pancreas, kidneys, and adrenal glands) are mostly covered by the visceral peritoneum. Ligaments and mesentries partition the peritoneal cavity into nine potential spaces—right and left subphrenic, subhepatic, supramesenteric and inframesenteric, right and left paracolic gutters, pelvis, and lesser space (see **Figure 1**). These spaces are important sites of abscess collection following various disease processes [12, 13].

The retroperitoneal space is located between the peritoneum and posterior parietal wall and runs from the diaphragm to the pelvic floor. Located in this region are the iliac and lumbar fossae which are continuous with one another. The diaphragm, spinal column, quadratus lumborum, and psoas major muscles respectively form the superior and posterior borders of the retroperitoneal region while levator ani muscles form the inferior border. The adrenal glands, kidneys, ascending and descending colons, duodenum, and lymph nodes are located in this region. The ureter, renal vessels, gonadal vessels, inferior vena cava, and aorta also pass through it. See **Figure 2** [13, 15].

4. Classification

Intra-abdominal abscesses are classified into (i) intraperitoneal, (ii) retroperitoneal, and (iii) visceral. Examples are listed in **Table 1** [16]. Retroperitoneal abscesses are categorised as either primary or secondary depending on whether hematogenous dissemination caused the infection or if it was caused by an infection in a nearby contiguous structure [13].

5. Intraperitoneal abscess

5.1 Aetiology and microbiology

Following perforation of a hollow viscus, as in ruptured appendix, gastric peptic perforation, typhoid ilial perforation, or perforated colonic or gastric cancer, peritonitis and/or intraaabdominal abscess may occur depending upon degree of

Intraperitoneal	Retroperitoneal	Visceral
Subphrenic	Pancreatic	Hepatic
Subhepatic	Post-traumatic	Splenic
Right or left lower quadrant	Perinephric	
Paracolic		
Interloop		
Lesser sac		
Pelvic		

Table 1.
Classification of intra-abdominal abscesses.

contamination and containment ability of the peritoneum and other host factors. Other conditions that can cause intra-abdominal abscess formation include appendicitis, diverticulitis, Crohn disease, pancreatitis, and pelvic inflammatory disease. Post-operative complications such as anastomotic leak may also result in abcess formation. Subhepatic abscess may result from thoracic pathologies such empyema and lung abscess. Abscesses may form following penetrating abdominal injuries, notably those to the colon, liver, pancreas, and spleen [16]. The risk of developing intra-abdominal abscesses is increased by diabetes mellitus, malnutrition, HIV/AIDS, anaemia, and liver disease.

The primary source determines the microbiological spectrum of an intra-abdominal abscess. In intra-abdominal abscesses, various intestinal flora are typically prevalent, indicating the prevalence of concomitant illnesses originating from this anatomic location. Accordingly, coliform bacteria, particularly *Escherichia coli* and *Bacteroides fragilis,* are the main agents. *Klebsiella* spp., *Proteus* spp. and *Enterobacter* spp. are important coliforms in abscess formation. Others are streptococci, enterococci, and anaerobic bacteria. A particularly invasive anaerobic pathogen in abdominal infections and abscesses is *B. fragilis* which is an obligate anaerobic gram-negative bacillus. In the evolution of abdominal sepsis and an intra-abdominal abscess, coliform bacteria in a mixed form are mostly responsible for the early sepsis, whereas anaerobes are responsible for the late sequelae with abscess formation [6, 16].

Following penetrating abdominal injury, skin flora as well as bacteria on surfaces of sharp objects may be responsible for an intra-abdominal abscess. In females with pelvic inflammatory disease, *Neisseria gonorrhoeae* and *Chlamydia* spp. are the most often found pathogens [17]. Recent studies shows link between SARS-CoV-2 (Covid-19) infection and gastrointestinal complications such as bowel perforation and intraabdominal abscesses [18, 19].

5.2 Pathology and pathophysiology

Generally, local peritoneal infections usually resolve spontaneously or under appropriate antibiotic regime. However, once tissue destruction occurs, decomposition is inevitable because the micro-organisms in the dead tissue are no longer accessible to systemic antimicrobial agents.

Intra-abdominal abscesses are confined by an inflammatory barrier. The omentum, inflammatory adhesions, or neighbouring viscera may all be a part of this barrier. Bacteria from the GI tract, both aerobic and anaerobic, are typically present in the abscesses. Polymorphonuclear cells are induced by bacteria in the peritoneal cavity, particularly those coming from the large bowel. The viscera and omentum have a tendency to localise the infection location, resulting in a phlegmon. Anaerobe development is aided by the resultant hypoxia, which also reduces granulocyte bactericidal activity. Leucocyte phagocytic activity breaks down cellular and bacterial waste, generating a hypertonic environment that causes the abscess to expand in reaction to osmotic pressures. Abscess may spread to other actual or potential intraperitoneal spaces (see **Figure 1**). The thoracic cavity may become infected by subdiaphragmatic abscesses, leading to empyema, lung abscess, or pneumonia. A lower abdominal abscess may spread to the thigh or perirectal space. Most patients with intra-abdominal abscesses exhibit a moderate systemic reaction. Others experience a significant septic reaction, volume depletion, and a catabolic state. They may also have decreased urine output and tachycardia. Sequential organ failure strongly suggests intra-abdominal sepsis [2, 17, 20].

5.3 Clinical presentation and patient evaluation

One of the main challenges in the management of complicated intra-abdominal infections is early recognition. The associated high mortality can be reduced by early diagnosis, adequate source control and intervensions to restore anatomy and physiological function [5]. The presentation of intra-abdominal abscesses may be highly variable. Persistent abdominal pain, focal tenderness, spiking temperatures particularly the swinging variety, persistent tachycardia, prolonged ileus, leukocytosis, or intermittent polymicrobial bacteremia in patients with predisposing primary intra-abdominal disease or in those who have undergone abdominal surgery should raise the suspicion of an intrabadominal abscess.

In patients with deep seated abscess many of these features may not be present. Nonlocalizing debilitating disease, modest liver dysfunction, chronic gastrointestinal (GI) dysfunction, or persistent fever may be the only early warning signs. There are some indicators that could point to an intraabdominal abscess. Epigastric pain or discomfort and a painful, immovable epigastric enlargement may be felt in patients with abscesses in the smaller sac. Pain in the right upper abdomen, unexplained tarchypnoea and tarchycardia are common in subphrenic abscess. There may also be hiccups. Diaphragmatic irritation may cause referred in the right shoulder. Right lung examination may demonstrate pleural effusion or basal crepitations [17].

Pelvic abscess may develop after generalised peritonitis has been treated; it may also occur in conjunction with gangrenous appendicitis, ileal or duodenal rupture. In females, pelvic abscess can complicate septic abortion or pelvic inflammatory disease. Pelvic abscess localises in the Pouch of Douglas in females and in between the bladder and the rectum in males. Patients with pelvic abscesses may have lower abdominal pain, fluctuating temperatures, malaise, and anorexia. They may have diarrhoea, tenesmus from irritation of the rectum, or frequent and urgent urination from bladder irritation. Lower abdominal pain may be associated with a tender suprapubic mass, and a tender, boggy mass on the anterior wall of the rectum on digitalrectal examination. Pelvic abscess may about the iliopsoas muscle and present atypically with hip pains and limping due to referred pain [21].

Plain abdominal radiographs may show an air pocket under the diaphragm, a localised ileus, extraluminal gas, air-fluid levels, soft tissue masses that are mottled, the absence of psoas outlines, or visceral displacement. The chest radiograph may reveal pleural effusion, raised hemidiaphragm, basal infiltrates, or atelectasis in subphrenic or even subhepatic abscesses. An ultrasonography may identify the abscess location, size, and number. It is readily accessible and cheap with accuracy rate of more than 90%. It can be performed on patients in intensive care units who are immobile and critically sick.

The limitations of ultrasonography include of presence surgical dressings, open wounds, intestinal gas, intervening viscera, significant obesity, and stomas. It is also heavily operator-dependent. Computed tomography (CT) gives better details of abdominal abscesses than abdominal sonography. It has greater than 95% diagnostic accuracy and is not limited by the presence of ileus, stomas drains or dressings. The appearance of an air bubble within a fluid collection or a low-attenuation extraluminal mass is diagnostic of an intra-abdominal collection. CT scan can document inflammatory edema in the adjacent fat (obliteration of fat plane) and hyperemia in the abscess wall (enhancement). Until about postoperative day 7, when absorption of non-suppurative fluids (such as hematoma, seroma, or intraoperative irrigation fluid) should have been complete, CT is not advised for use in the immediate post-operative

period for the purpose of identifying abscesses. Water-based oral contrast and intravenous (IV) contrast are used for good anatomic resolution. Extravasation of oral contrast indicates a fistula or an anastomotic leak. IV contrast may enhance the abscess by concentrating within the abscess wall. The use of oral and IV contrast may be limited by ileus, allergy to contrast material, and renal insufficiency. The limitations of CT include nonportability, a potential lack of patient participation, and a relative difficulty in accurately detecting intraloop abscesses. In the majority of hospitals in developing nations, it is also not easily accessible. Gallium-67 or indium-111-tagged leukocytes may be used to pinpoint the location of the inflammation. Such radio-isope scans take a lot of time, and they frequently provide false-positive results due to nonpyogenic inflammation, intestinal leukocyte buildup, surgical drains, and incisions. CT and MRI has largely replaced the use of radio-isope scan in the diagnosis of intarabdominal abscess except for viscereal abscesses such as liver abscesses [17].

5.4 Management

The following principles guide the management of intra-abdominal abscesses.

1. Resuscitation

2. Anti-microbial therapy

3. Source control

5.4.1 Resuscitation and supportive measures

While confirmation of diagnosis is pursued using appropriate imaging and laboratory tools, patient assessment and optimization is also carried out. The first step of resuscitation and optimization is determined by the patient's specific problems. It is appropriate to focus on the ABCs (airway, breathing, and circulation) while tailoring the intervention to each patient's divergence from normal physiology. Anaemia, dehydration, dyselectrolytemia, malnutrition and sepsis are all recognised and treated. Systemic diseases including diabetes mellitus, chronic kidney, and liver disease, are all assessed and optimised. When considering the patient's presentation, the differential diagnosis of an intraabdominal abscess are sought after. Inflammatory bowel disease, prolonged ileus, and unexplained postoperative fever are among the differentials [2, 17, 18, 20].

5.4.2 Antimicrobial therapy

Appropriate bactericidal wide spectrum antibiotic are commenced while the causative bacterial agent(s) and their sensitivity pattern are identified. Antimicrobial management is typically standardised, and numerous regimens—either as monotherapy or combination therapy—have demonstrated varying effecacies. In the context of each intraabdominal abscess clinical scenario, empiric antimicrobial therapy should be comprehensive and should address all possible infectious agents. When there is evidence of candida involvement or when the patient has risk factors for invasive candidiasis such as immunodeficiency state or prolonged antibacterial therapy, antimicrobial coverage of *Candida* spp. is advised. Antimicrobial treatment should typically last 5–7 days. After a week, if sepsis still exists, a diagnostic workup and, if necessary, a surgical re-intervention should be considered [2, 5].

5.4.3 Source control/abscess drainage

The term *source control* refers to any physical actions performed to control an infection focus, such as the removal of necrotic tissue, surgical repair, resection, and/or exteriorization of the anatomical defect that is the source of peritoneal contamination [2]. Source control for intra-abdominal abscesses can be accomplished by either image-guided or open surgical drainage.

5.4.4 Abscess drainage (indications, methods/imaging, preparation and procedure)

Percutaneous image-guided drainage is a minimally invasive method of abscess source control which has been proven to be an effective alternative to surgery with comparable success rates and lower morbidity rates. It is cheaper with fewer complications, and avoids general anaesthesia.

It is performed for abscesses that are in readily accessible locations, unifocal and unilocular and without having to pass through hollow organs. Some multifocal and multiloculated abscesses can also be drained by this method but this may be more technically challenging. There is also the risk of incomplete drainage.

Drainage is accomplished by the use of image guidance such as computed tomography or ultrasonography. Where necessary, a local anaesthetic agent is injected into the skin. Sedation may be necessary for some uncooprative patients and in children. Skin preparation is done with clorhexidine, iodine solution or other suitable antiseptic agents. Trocar as well as Seldinger techniques are frequently employed for catheter placement. The choice of method is based on the surgeon's preferences, experience, and clinical situation [22]. After the abscess has been located, the initial diagnostic aspiration is submitted for Gram stain and microbiological culture. The drainage catheter should be as small as possible to ensure safety while still being large enough to prevent the tube from being easily blocked (usually 8–12 Fr). Following entry into the abscess cavity, the catheter is connected to a closed drainage system. Suction or irrigation of these catheters does not seem to be beneficial, whilst flushing with saline once a day is suggested to maintain patency (**Figure 3**) [2, 5].

5.4.5 Pearls and pitfalls

Heart rate, respiratory rate, blood pressure, and oxygen saturation should be actively monitored during percutaneous drainage of intraabdominal abscess because of the possibility of haemorrhage, and oversedation [22]. An abscess cavity normally decompresses following proper catheter insertion. To ensure tube patency, the catheter should be irrigated once daily. Within 48 hours of catheter placement, patients are expected to have an improvement in symptoms. With cessation of drainage, repeat imaging is done to check for any residual contents before extubation.

If abscess drainage increases or continues over time, an anatomic defect, a need for change in antibiotic therapy or background impaired immunity should be suspected. Bacteremia, sepsis, enterocutaneous fistula, vascular injury, and intrapleural catheterization are uncommon but possible complications of image-guided catheter insertion into the abdomen.

Smaller appendiceal abscesses in less ill patients may be successfully treated with antibiotics alone. If the abscess is of large volume, drainage can be achieved by percutaneous image-guided approach; interval appendectomy is performed after 6 weeks. If fever and leukocytosis persist after an initial drainage, an urgent appendectomy is

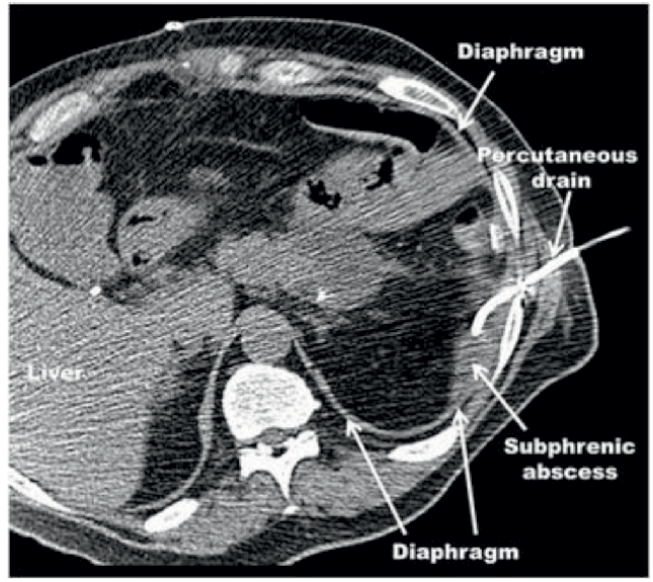

Figure 3.
CT-guided drainage of a subhepatic abscess [23].

performed during same hospital admission. Pelvic abscesses less than 2 cm in diameter can be treated conservatively; transvaginal or transrectal drainage with endoscopic ultrasonographic guidance is used to treat larger abscesses. A lower abdominal percutaneous approach is occasionally employed [13].

5.4.6 Surgical drainage

Surgical drainage of an intraabdominal abscess may be achieved by either a minimally invasive method (laparoscopic) or by an open technique (laparotomy). Indications for surgical drainage include (1) abscesses located deep to hollow organs not amenable to image-guided drainage, (2) multiple large volume abscesses, (3) a known intra-abdominal source requiring surgical intervention or (4) an abscess of unidentified aetiology.

Subhepatic abcess not completely drained by a percutaneous method, or if deep-seated with intervening gallbladder, bowel loops, or adhesions may require an open drainage. This may be performed extraperitoneally through the bed of the 12th rib for posterior abscess (Morrison's pouch) or through a right subcostal incision if it is located anteriorly. If abdominal exploration is also required, transperitoneal drainage may be performed [20].

Laparoscopy is a minimally invasive method that allows for exploration of the abdominal cavity without the need for a large incision. Abscesses can be drained safely while also examining the whole peritoneal cavity. Anatomic defects can also be addressed by this method. Both laparotomy and laparoscopy have carry the risk of visceral injuries especially in the presence of bowel obstruction, adhesions, and loss of anatomic integrity from bowel edema. Careful examination is therefore necessary. Laparotomy also causes more metabolic response and prolonged hospital stay.

The prognosis of patients with an abdominal abscess before the era of advanced imaging was very high. Today, with the availability of ultrasonography and CT, the

diagnosis is made much earlier, and, in many cases, drainage with the aid of these imaging modalities has helped lower morbidity and mortality. Generally, risk factors that increase mortality and morbidity in patients with intraabdominal abscess include advanced age, multi-organ failure, multiple recent surgeries, complex abscess and delay in diagnosis [3].

6. Retroperitoneal abscesses

6.1 Aetiology and microbiology

The origin of retroperitoneal infections and abscesses is commonly the organs and tissues present within or abutting the retroperitoneal space. Retroperitoneal abscesses are caused by a variety of conditions including complicated pancreatitis, retrocecal appendicitis, contained duodenal ulcer perforation, iatrogenic perforation during esophagogastroduodenoscopy (EGD) or endoscopic retrograde cholangiopancreatography (ERCP), and renal infections [12]. The bacteriological profile of retroperitoneal abscesses is related to the cause. Gram-negative rods like *Proteus mirabilis* and *Escheritia coli* are frequently involved in kidney infections and are frequently monomicrobial in nature. *E. coli, Enterobacter* spp., enterococci, and anaerobic species like Bacteroides are commonly involved if the source is GI tract. Retroperitoneal abscess may follow penetrating trauma to the central areas of the back. It may also be caused by spinal tuberculosis (TB) in immunocompromised patients and in patients in TB endemic areas of developing countries [12, 13]. Lumbar acupuncture was reported to cause retroperitoneal abscess [24].

6.2 Pathology and pathophysiology

Retroperitoneal abscess may be single or multiple, of small volume or of large quantity. Due to the substantial space and non-discrete boundaries of the retroperitoneum, abscessess can accumulate in large quantity prior to diagnosis. It may expand toward the peritoneal cavity or may track through areas of weakness on the posterior abdominal wall and appear on the subcutaneous space, eventually rupturing and draining through the skin. Patients usually have comorbidities such as diabetes mellitus, renal failure and malignancy. These contribute to increased morbidity of the disease [12, 13].

6.3 Clinical presentation and evaluation

Retroperitoneal abscesses are characterised by an insidious, poorly localised illness marked by diagnostic delay, inadequate drainage, and significant morbidity [25]. Lower abdominal or flank pain, which affects 60–75% of patients, fever and chills, which affect 30–90% of patients, malaise, and weight loss are the most typical symptoms of retroperitoneal abscess. Psoas abscess may cause referred pain in the hip, groin, or knee. Features of the primary infectious focus may be elicited in the history. Symptoms typically last for more than 1 week. In severe cases there may be features of sepsis. Chronic conditions like renal calculi, diabetes mellitus, HIV infection, or cancer are frequently identified in patients with retroperitoneal abscesses. CT confirms the diagnosis, identifies the location of the abscess, its size and relationship to neighbouring structures. The lesion appears as a hypodense retroperitoneal mass

with surrounding inflammation. As many as one-third of these lesions contain gas. CT helps identify possible source of the infection offers details on the potential best drainage route [13].

6.4 Treatment

The principles of treatment of retroperitoneal abscesess include (i) patient optimization, (ii) use of broad spectrum antibiotics, (iii) drainage of the collection, and (iv) identification and treatment of the underlying retroperitoneal infectious source. All contributing co-morbid conditions are also addressed (see Section 5.5.1 above). Image-guided percutaneous drainage (**Figure 4**) is strongly preferred although operative drainage may occasionally be required for adequate drainage of complex or multiple collections. The prognosis in retroperitoneal abscess is commonly affected by delay in presentation and diagnosis. In rare instances of retroperitoneal necrotizing fasciitis, the mortality rate can be as high as 25% [2, 13].

7. Visceral abscesses

7.1 Aetiology and microbiology

Pyogenic liver abscesses are the most common types of liver abscesses. They are caused by intestinal infections like acute appendicitis and diverticulitis, which spread to the liver through the portal circulation. Pyogenic liver abscesses can also develop as a direct result of infections like diverticulitis or Crohn's disease spreading into the

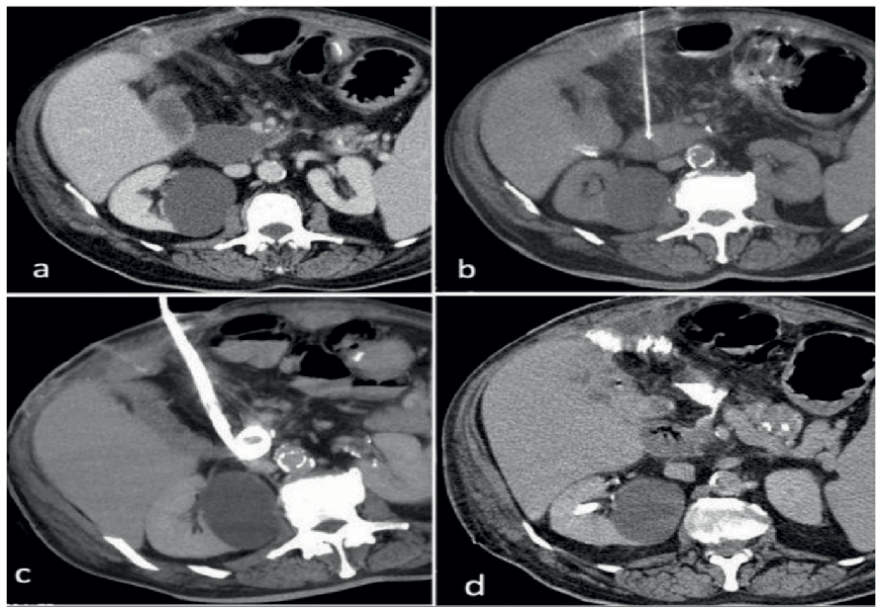

Figure 4.
Drainage of a retroperitoneal fluid after pancreatic surgery (a). Percutaneous puncture with a Chiba needle (b), a 12Fr pigtail catheter placed (c) MIP reconstruction using the Seldinger technique. Repeat CT after 2 weeks with complete resolution (d) [26].

liver, impaired biliary drainage, subacute bacterial endocarditis, infected indwelling catheters, dental procedures, or other conditions. About 40% of these abscesses are monomicrobial, 40% are polymicrobial, while 20% culture-negative. *Escherichia coli,* (present in two-thirds of cases), *Streptococcus faecalis*, Klebsiella, and *Proteus vulgaris* are the most common bacterial agents. *Bacteroides fragilis* and other anaerobic organisms are cultured as well. Risk factors for pyogenic liver abscesses include diabetes mellitus, cirrhosis, pancreatitis, inflammatory bowel disease, pyelonephritis. Pyogenic liver abscess is seen in 17–36% of patients with lymphoma, leukaemia, and solid organ cancers [27]. Intestinal and extraintestinal amebiasis such as *amoebic liver abscess* are caused by the ubiquitous protozoan *Entamoeba histolytica. Entamoeba dispar* occassionally causes amoebic liver abscess. *Splenic abscesses* may result from extension from infections of a contiguous structure such as the colon, kidney, or pancreas. Most common organisms are Gram-positive cocci (*Staphylococcus, Streptococcus,* or *Enterococcus* spp.) and Gram-negative enteric bacteria. In immunocompromised individuals, splenic abscess may be caused by *Mycobacterium tuberculosis, Mycobacterium avium, Actinomyces* spp. and *Candida* spp [9, 28].

7.2 Pathology and pathophysiology

The liver is frequently exposed to loads of bacterial from the GI tract via the portal venous system. The liver's reticuloendothelial cells clear this bacterial load without sequelae. However, when an inoculum of bacteria exceeds the ability of the liver to clear it, bacterial tissue invasion, neutrophil infiltration, and abscess formation occurs. Pyogenic liver abscesses are more frequently found in the right lobe of the liver due to its higher vascularity and oxygenation. Left lobe involvement occurs in only 20%. Bilobar involvement is uncommon. Approximately half of hepatic abscesses are solitary. The sizes of hepatic abscesses vary from less than 1 mm to 3 or 4 cm in diameter. If multiple, they may coalesce to give a honeycomb appearance. *Amebic Abscesses* are caused by *Entamoeba histolytica.* The parasite exists as cysts in a vegetative state which are acquired faeco-orally, passes through the stomach and small bowel and transform into trophozoites in the colon where it invades the colonic mucosa, forming typical flask-shaped ulcers. They enter the portal venous system and are carried to the liver. The superior-anterior aspect of the right lobe of the liver, close to the diaphragm, is where amebic abscesses are most frequently found. They have a necrotic central area that contains a thick, reddish-brown pus-like substance [27, 29].

Splenic abscesses are more common in tropical regions and are frequently seen in sickle cell patients in whom thrombosis and infarction of splenic vessels commonly occurs. Splenic abscess is seen in the following conditions: (a) hematogenous spread; (b) contiguous infection; (c) hemoglobinopathy; (d) immunosuppression and (e) trauma. *Streptococci* Spp. and *Escherichia coli* are the most prevalent organisms in most series. *Mycobacterium tuberculosis* and *Salmonella typhii* are occasionally isolated [29].

7.3 Clinical presentation and evaluation

Patients with *pyogenic liver abscess* presents with right upper quadrant pain and fever. Fever, jaundice, and right upper quadrant pain with tender hepatomegaly are the typical description of a hepatic abscess; however, only 10% of cases have this classic presentation. A thorough history and physical examination are therefore necessary in assessing these patients. Endogenous endophthalmitis, a rare complication unique to Klebsiella hepatic abscesses, affects about 3% of patients. Diabetics are more

susceptible to this ocular complication. The best means of preserving visual function is early diagnosis and treatment [29].

Hepatic amoebiasis should be considered if right upper quadrant pain, fever, hepatomegaly, and a hepatic cystic lesion are present in a patient who resides in an endemic area or patient who migrated from amoebiasis endemic area. Diaphragmatic involvement may cause cough or dyspnea. Occasionally, rupture of the abscess into the peritoneal cavity may cause peritonitis. Rupture into the pleural space or pericardium has been reported but this represents an uncommon occurrence. Although the hepatic lesions follow an initial bowel infection, the presence of diarrhoea at the time of diagnosis of amoebic liver abscess is unusual Amoebic liver abscess can manifest with fever and pain in the right upper quadrant, weight loss. Shoulder pain or right-sided pleural pain are symptoms of diaphragmatic involvement. Between 10% and 35% of patients will experience gastrointestinal symptoms like nausea, vomiting, abdominal pain, abdominal swelling, diarrhoea, and constipation. Tender hepatomegaly or localised subcostal tenderness is typical. Elevated transaminase levels and jaundice are unusual, whereas leukocytosis is common. A slightly elevated alkaline phosphase level is the most typical biochemical abnormality. Fluorescent antibody tests for *E. histolytica* are typically positive and about 90% of patients continue to test positive for some time even after a clinical cure. Due to the high sensitivity of this serologic test, amebiasis is unlikely if the test is negative. However patients with *Entmoeba dispar* infection will have negative serologies. Periodic acid-Schiff stain of biopsies of the edge of the ulcers or the wall of an abscess may reveal the typical trophozoites. Amebic abscesses are usually seen on CT as well-defined hypodense round lesions with enhancement of the wall, somewhat ragged in appearance with a peripheral zone of edoema, the central cavity with septations as well as fluid levels [8, 27, 29].

The majority of patients with splenic abscess experience symptoms for 2–3 weeks before presentation. In about 33% of patients, symptoms include fever, left upper quadrant pain, leukocytosis, and splenomegaly. Both an ultrasound and a CT scan a have about 95% sensitivity and specificity in confirming the diagnosis [27, 30].

7.4 Treatment

Various treatment modalities have been developed for the management of *pyogenic liver abscesses*. The clinical situation determines which modality is most appropriate. They include the following.

- Unimodal treatment with broad-spectrum *antibiotics* alone
- Single or repeated *needle aspiration* and *antibiotic* therapy
- Image-guided percutaneous *catheter drainage* and *antibiotic* therapy
- *Laparoscopic drainage* and *antibiotic* therapy
- *Laparotomy* (open drainage) and *antibiotic* therapy

Fluoroquinolones (or aminoglycosides) and clindamycin (or metronidazole) are among first line agents used in empiric treatment of pyogenic liver abscess. Single-agent therapy with ticarcillin-clavulanate, imipenem-cilastatin, or piperacillin-tazobactam is also acceptable. Carbapenems are recommended when extended spectrum

β-lactamase–producing strains are isolated or strongly suspected. Traditionally, 4 to 6 weeks antibiotic therapy is recommended however, a 2-week antibiotic regimen may suffice [8, 30].

Most patients with small pyogenic liver abscesses respond well to antibiotic therapy alone. Percutaneous image-guided drainage may be required if abscess volume is large or in patients with small abscesses who do not respond to initial antibiotic therapy. It should be noted however that pyogenic abscesses can be very viscous and catheter drainage may be ineffective. Needle aspiration (which is less invasive, less expensive, and avoids the complications associated with catheter placement and care) if frequently ineffective and carries greater recurrence rates with patients requiring additional intervention. Surgical drainage either via the laparoscopic or open approach may be necessary if initial treatment fails. Occasionally, an anatomic surgical resection can be performed in patients with recalcitrant or deep abscesses. Laparoscopic or open drainage is considered in patients with (1) multiple large volume abscesses, (2) a known intra-abdominal source requiring surgery, (3) an abscess of unidentified aetiology, (4) ascites, and (5) abscesses requiring transpleural passage. In the past, management of right-sided abscess is achieved by removing the 12th rib followed by extraperitoneal drainage. This avoids contaminating the peritoneal cavity.

Transperitoneal laparoscopic or open drainage has the following advantages: (1) treating the inciting pathology in the remainder of the abdomen/pelvis; (2) gaining access and exposing the entire liver for evaluation and treatment; and (3) access to the biliary tree for cholangiography and bile duct exploration if indicated [27, 30].

Pearl: It is important to keep in mind that in patients with presumed pyogenic abscess, a necrotic hepatic malignancy may be mistaken for a hepatic abscess. Therefore, *early* diagnosis and progression to surgical resection should be considered for patients who do not respond to initial broad spectrum antibiotic therapy.

Amebic liver abscess. Amebic liver abscess is primarily treated non-operatively. Metronidazole has excellent efficacy, low cost, and ability to treat both extraintestinal and intestinal amebiasis. The recommended dosage is 750 mg three times a day for 10 days. If defervescence does not occur after 72 hours or if the patient is critically sick, chloroquine may be added. Usually, amebic liver abscess responds within 48 to 72 hours of amebicidal therapy.

Image-guided drainage of amoebic liver is abscess indicated if there is bacterial suprainfection, a probable pyogenic liver abscess, and a large, left-sided abscess (segments 2 and 3) which poses danger of rupture into the pericardium. In addition to amebicides, pleuracentesis or pericardiocentesis, as appropriate, may be used to treat abscess rupture into the pleura or pericardium. Laparotomy is indicated for burst amebic abscesses into the peritoneum [27, 30].

Splenic abscess. Broad-spectrum antibiotics are started as soon as a splenic abscess is suspected, and depending on the results of the culture, the antibiotic regimen should be modified and continued for a further 14 days. The preferred treatment is splenectomy, although individuals who cannot tolerate the procedure can be offered percutaneous image-guided drainage as alternative. Patients with unilocular abscess can successfully be treated with percutaneous drainage [30, 31]. Lee *et al.* reported gender, age, number of abscess cavities, immunodeficiency state, underlying comorbid diseases and type of microorganism as important prognostic factors in splenic abscess [9]. Chang *et al.* found high APACHE II scores and Gram negative bacillus abscess as additional prognostic factors. Mortality rate for splenic abscess range from 15 to 20% in previously healthy patients who have single unilocular lesions; it may as high as 80% for multiple abscesses in immunocompromised patients [10, 28].

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Chapter 4

Ovarian Abscess within an Endometrioma: Risk Factors and Management

Shashwati Sarkar Sen

Abstract

Ovarian endometriosis increases the risk of development of an abscess within the ovary. Tubo-ovarian abscess within an endometrioma occurs when the infected tube serves as a portal of infection and the endometriotic blood serves as a culture medium. Pelvic Inflammatory Disease, Intra uterine contraceptive device (IUCD), ultrasound guided oocyte retrieval in IVF-ET and endometrioma aspiration are possible sources of infection. Primary ovarian abscess without involvement of the fallopian tube, is a rare complication of an underlying endometrioma and may be due to iatrogenic introduction of pathogen during transvaginal surgical procedures. De novo primary ovarian abscess within an endometrioma in the absence of a risk factor is even rarer. Weakened endometriotic cyst wall and hematogenous spread of infection are possible explanations for bacterial implantation. Infected ovarian endometrioma is a surgical emergency and preserving the fertility in a nullipara is a challenge which can be overcome by timely intervention.

Keywords: endometrioma, ovarian abscess, infection, infertility, ART, ovum retrieval, laparoscopy

1. Introduction

Endometriosis is the development of estrogen-dependent endometrium-like tissue outside the uterine cavity. It primarily involves the pelvis but may be found at extra-pelvic sites too. In the pelvis, the lesions are predominant on the peritoneum, ovaries and rectovaginal septum. Extra pelvic implants can be seen on the bowel, scar of previous surgical incisions and at distant locations like lung parenchyma, cerebellum etc. About 10% of the reproductive aged women are affected by this condition worldwide.

The ectopic endometrial tissue being hormonally active, will undergo shedding during the menstrual phase forming a hematoma. Recurrent bleeding induces a chronic inflammatory reaction with formation of adhesions and adnexal masses. Endometriotic lesions undergo a stepwise phenotypic progression. The earliest lesion is the red vesicular subtype and bleeding stimulates the development of fibrin-mediated adhesions. In the final stage, cicatrization leads to the formation of black

powder-burn lesions. Recurrent inflammatory reaction may result in a peritoneal defect referred to as an Allen-Masters window.

Endometriosis is a debilitating condition with a range of painful symptoms that include dysmenorrhea, dyspareunia, dyschezia and dysuria. The most common presenting complaints are persistent pelvic pain and/or infertility. Often, the severity of the condition does not correlate with the severity of the patient's symptoms leading to a delay in seeking help. Patients may even ignore the condition due to confounding indicators and the condition may only be revealed during infertility evaluation. A mean latency of 6 to 7 years from onset of symptoms to conclusive diagnosis of endometriosis is reported in most studies [1].

1.1 The ASRM classification system

The ASRM (American Society for Reproductive Medicine) classifies endometriosis into four grades using a point system corresponding to the number of lesions and depth of infiltration.

Stage 1: Minimal
Stage 11: Mild
Stage 111: Moderate
Stage 1v: Severe

This grading system most often fails to correlate with the degree of pain or the probability of a successful outcome of infertility treatment in the patient.

To overcome these limitations, The Endometriosis Foundation of America (Endofound) proposed a more descriptive classification system based on the anatomical location and pathophysiology of the lesions.

1.2 The Endofound classification system

i. Category 1: Peritoneal Endometriosis

ii. Category 11: Ovarian Endometrioma

iii. Category 111: Deep Infiltrating Endometriosis I (DIE I)

iv. Category 1v: Deep Infiltrating Endometriosis II (DIE II)

2. Ovarian endometrioma

Ovarian Endometrioma occurs when endometrial stromal and glandular tissue appear in ovaries giving rise to cystic lesions. It is seen in 2–10% of reproductive age women. The incidence almost 50% in women seeking assisted reproductive treatment (ART).

The ectopic endometrial tissue is responsive to proliferative effect of estrogen and undergoes cyclical shedding during menstruation. Bleeding within a closed space leads to formation of ovarian cysts enclosed by an endometrial epithelium. The collected blood is thick and brown resembling chocolate sauce and hence, endometriomas are also referred to as "chocolate cysts".

3. Etiopathogenesis of endometrioma

The etiopathogenesis of endometriomas remains debatable and several hypotheses have been suggested.

1. Retrograde menstruation through patent fallopian tubes with implantation of endometriotic tissue on ovaries.

2. Invagination of the ovarian cortex and subsequent collection of menstrual debris from bleeding of active endometriotic implants invading the cortex [2].

3. Failed resorption of the entrapped blood within a corpus luteum due to the presence of endometriotic lesions and adhesions on the ovarian cortex [3].

4. Colonization of functional ovarian cysts by endometriotic cells.

5. Coelomic metaplasia of the invaginated ovarian mesothelium forming mesothelial inclusions in the ovarian cortex [4].

4. How do endometriomas reduce fertility?

Ovarian endometrioma is a more severe form of the disease corresponding to ASRM Stage 111 or Stage 1v. About 17–44% of women diagnosed with endometriosis will have an endometrioma. It has a deleterious effect on the fertility of a woman in the following ways:

1. Decreased Ovarian Reserve: It causes destruction of the ovarian cortical tissue with loss of antral follicle quality and quantity.

2. Formation of adhesions: Repeated bleeding induces a chronic inflammatory reaction in the peri-adnexal region with formation of tubo-ovarian masses and adhesions.

3. Altered tubo-ovarian anatomical relationship: The ovaries may be pulled medially behind the uterus due to fibrosis which then appear as "kissing ovaries".

4. Tubal blockage: The fallopian tubes may be buried inside adhesions with loss of fimbriae. Scarring of the tubes can lead to loss of patency.

5. Etiology of formation of ovarian abscess in endometrioma

Infected endometriotic cysts are rare and very few cases have been reported. This may be due to the following reasons:

- Endometrioma capsule is generally thick, and this may impede the entry of bacteria from the infected fallopian tube.

- Under-reporting of infected endometriomas due to lack of clear pathologic guidelines for diagnosis.

Infected endometrioma was recognized as a serious gynecologic entity for the first time in 1981 when Cecilia, et al. conducted a retrospective analysis of 11 patients with pathologically confirmed infected endometriotic cysts. None of them had any history of long standing Pelvic inflammatory Disease (PID). 10 of the 11 patients had salpingitis suggesting that the fallopian tube was a probable portal of infection. The source of infection could not be established in 1 patient who had ovarian abscess with healthy tubes [5].

Different explanations have been given for the development of an abscess within an endometrioma:

1. Altered menstrual blood collected within an endometrioma acts as a potential culture medium for pathogenic microorganisms. Altered blood within the cyst provides an anaerobic environment favorable for the growth of bacteria.

2. There is an altered immune environment within the endometrioma due to a decreased natural killer (NK) cell cytotoxicity in the peritoneal cavity and peripheral blood of women with endometrioma leading to a compromised local immune system. Resistance to lysis by natural killer (NK) cells and a weakened macrophage function contribute to persistence of ectopic endometrial tissue.

3. Endometriotic cyst walls are more friable than normal ovarian epithelium.

4. Hematogenous and lymphatic spread of infection due to urinary tract infection, appendicitis, diverticulitis, tonsillitis and tuberculosis.

Associated pelvic inflammatory disease (PID), hydrosalpinx, use of intrauterine contraceptive device (IUCD), bowel infection, transvaginal invasive procedures increase the risk of developing a tubo-ovarian abscess within an endometrioma.

6. Types of ovarian abscess

Ovarian abscess can be:

1. Primary Ovarian Abscess – It is defined as inflammation originating in the ovarian tissue. The fallopian tubes are not involved.

It may result from disruption of the ovarian capsule, as may occur during ovulation or some surgical intervention, which gives bacteria access to the ovarian stroma.
This is a rare complication of an underlying endometrioma. It develops as an isolated ovarian lesion without simultaneous tubal infection. It may be of 2 types-

a. With an associated risk factor

This can be a complication of invasive procedures like:

i. Transvaginal Oocyte Retrieval as part of In-vitro fertilization (IVF)

ii. Transvaginal or percutaneous needle aspiration of an Endometrioma.

iii. Surgical intervention like cesarean section, vaginal hysterectomy, use of intrauterine device

Pathogenic microorganisms are possibly introduced into the ovarian stroma from the vagina during such procedures.

b. Without any associated risk factor

De novo primary ovarian abscess within an endometrioma is even more rare and very few cases have been reported. The first case of spontaneous abscess in an endometrioma was reported by Gary H et al. in 1991. The possible source of infection is suggested to be hematogenous or lymphatic spread from the urinary tract or gastrointestinal tract [6].

2. Secondary Ovarian Abscess – It arises in extraovarian locations with simultaneous fallopian tube infection.

Secondary Ovarian abscess in more common than primary where the infected fallopian tube serves as the portal of infection. It is associated with tubo-ovarian abscess, salpingitis, pelvic inflammatory disease (PID) or complications of gastrointestinal infections like diverticulitis, appendicitis, Crohn's disease.

Endometrioma is a risk factor for PID as the cyst provides a favorable site for bacterial proliferation. A distorted anatomy of the ovaries and fallopian tubes, impaired immune system, an increased inflammatory cytokines production and alterations in the vaginal microbiome further increase the risk of PID. A diagnosis of ovarian abscess should always raise the suspicion of an underlying endometrioma.

7. Route of infection

1. Ascending infection via the tubes: The cervical opening has a mucus plug which acts as a barrier and prevents the entry of vaginal microorganisms into the uterus. A breach in this barrier leads to ascending infection via the fallopian tubes into the pelvic cavity.

2. Hematogenous and lymphatic spread

3. Iatrogenic –due to invasive surgical procedures

4. Unknown

8. Risk factors for ovarian abscess

- ASRM Stage 111 and 1v Endometriosis
- Nulliparity
- History of pelvic surgery
- Bacteremia from skin wounds

- Dental treatment
- Congenital genitourinary anomalies
- Compromised immune system.

9. Pathogenesis

Ovarian abscesses are often polymicrobial with a predominance of anaerobic bacteria. The most commonly isolated microorganisms are Streptococcus type B, *Escherichia coli*, *Gardenella vaginalis*, Enterococcus sp., *Candida albicans*, *Brucella*, *Morganella morganii*, *Enterobius vermicularis*, *Streptococcus milleri*, *Peptococcus* and *Peptostreptococcus*. Ascending infection from the vaginal canal of normal commensals like Bacteroides has been suggested after the bacteria was isolated from the pus culture of an infected endometrioma [7].

10. Endometrioma and art

ASRM Stage 111 and 1v are considered risk factors for the development of tubo-ovarian abscess, particularly in nulliparous women [8]. Surgical management of endometriosis is controversial in a woman with subfertility as it may further reduce the ovarian reserve in an already compromised ovary. In-vitro fertilization and embryo transfer (IVF-ET) is considered the first line of management in such patients.

Oocyte retrieval (OR) is a standard IVF procedure following ovarian stimulation in assisted reproductive technology (ART) where the follicles are aspirated with the help of a needle introduced under transvaginal sonographic guidance. Risk of post-puncture pelvic infection is less than 1% but this can dramatically increase in the presence of endometriomas [9]. Endometriosis itself is a risk factor for recurrent pelvic inflammatory disease (PID) [10].

Presence of endometriomas may limit the accessibility of follicles which are behind the cyst. It is advisable to aspirate only the easily accessible follicles without puncturing the endometrioma. Yet, more often, it may be necessary to puncture the cyst to approach the follicles and this increases the risk of development of ovarian abscess in an endometrioma.

11. Clinical presentation

Classically, an ovarian abscess presents as an adnexal mass with acute abdominal-pelvic pain, fever, raised white blood cell count and/or vaginal discharge. An intrauterine or ectopic pregnancy should be ruled out with a urine pregnancy test. Rupture of the abscess may result in life-threatening sepsis with increased morbidity and mortality.

12. Diagnosis

1. Ultrasound: A transvaginal ultrasound provides a relatively easy and accurate diagnosis of endometrioma. Ovarian abscess appears as a complex multilocular

mass with internal echoes consistent with inflammatory debris. Thickening of the fallopian tube wall with presence of incomplete septa within, suggests tubal involvement. Fluid will be present in the Pouch of Douglas.

2. Magnetic resonance imaging (MRI) and Computed Tomography (CT) Scans: MRI and CT imaging help in better characterization of the pelvic masses. MRI is comparable to transvaginal ultrasound in diagnosing Ovarian Endometrioma. CT is not preferred due to its high cost and risk of exposure to ionizing radiation.

3. Laparoscopy: Laparoscopy is still considered the gold standard for diagnosis and facilitates the drainage of the abscess.

13. Management of endometrioma and ovarian abscess

It is pertinent to map the size and location of the endometriomas during infertility evaluation with the help of imaging techniques. Laparoscopy helps to determine the magnitude of the disease and its impact on fertility [11]. Surgical excision is a double edged sword and must be discussed in detail with the couple prior to IVF-ET. The decision depends on the age of the patient, her ovarian reserve, severity of the disease and any history of previous pelvic surgery.

Removal of small endometriotic cysts is not recommended. Surgery can have a poor impact on a woman's fertility by reducing her ovarian reserve. The risk of ovarian failure after bilateral ovarian endometrioma removal is reported to be 2.4% [12]. Risk of recurrence must also be considered. However, surgery is indicated if the endometriomas are large and there is a likelihood of inadvertent puncture with the needle during ovum retrieval as part of IVF-ET.

Laparoscopic surgery is preferred, and three approaches have been proposed in literature:

1. Surgical excision (Cystectomy).

2. Drainage and coagulation of the cyst wall.

3. Drainage and CO2 laser vaporization of the cyst wall.

The route of infection during oocyte retrieval starts in the vagina and hence, antibiotic prophylaxis should always be considered in the presence of an endometrioma irrespective of their size.

Development of an ovarian abscess in an endometrioma is a gynecological and surgical emergency and the woman should be immediately hospitalized for further care. Conservative treatment is generally ineffective in tubo-ovarian abscesses larger than 5 cm in diameter [13]. Failure of conservative treatment and suspected rupture are indications for urgent laparoscopic cystectomy.

Leakage of pus can cause peritonitis, extensive pelvic adhesions and sepsis. Early diagnosis and timely treatment of ovarian abscess is the key to prevent further complications like infertility, ectopic pregnancy and chronic pelvic pain.

After taking care of the hemodynamics, an emergency operative laparoscopy is performed to drain the abscess. Cyst drainage followed by stripping of the cyst wall is the procedure of choice to preserve the ovarian tissue in nulliparous women. Laparoscopic

surgery is less invasive as compared to laparotomy with less blood loss, less intraoperative and post-operative complications and a shorter hospital stay. A laparotomy should be considered if the patient is hemodynamically unstable, has extensive pelvic adhesions, generalized peritonitis or if facility for laparoscopy is not available. Care should be taken to preserve the uterus and at least one ovary and one fallopian tube if the patient is keen for future fertility. Irrigate the cavity well and initiate the patient on broad-spectrum antibiotics. Consider intra-operative aerobic and anaerobic cultures. Intra-operative tubal patency test should be avoided when an ovarian abscess is encountered in an endometrioma because of the risk of a flare-up of infection.

14. Histopathology

The pathologic criteria for an infected endometrioma are the presence of both endometrial glands and stroma within an ovarian cyst more than 4 cm in diameter along with frank pus within the cyst on gross examination or micro abscess formation within the cyst wall on histological examination. The cyst wall is lined by a fibrinopurulent exudate with underlying mixed inflammatory cells, including plasma cells.

15. Precautions

1. Every effort should be made to avoid puncturing an endometriotic cyst, if possible.

2. If there is a need to puncture an endometrioma during oocyte retrieval, flush the cavity of the cyst carefully. Consider prophylactic i.v. antibiotics.

16. Conclusions

An underlying endometrioma must be suspected in a young nulliparous woman presenting with an ovarian abscess. A history of transvaginal invasive procedures like oocyte retrieval for IVF and endometrioma aspiration increase the likelihood of

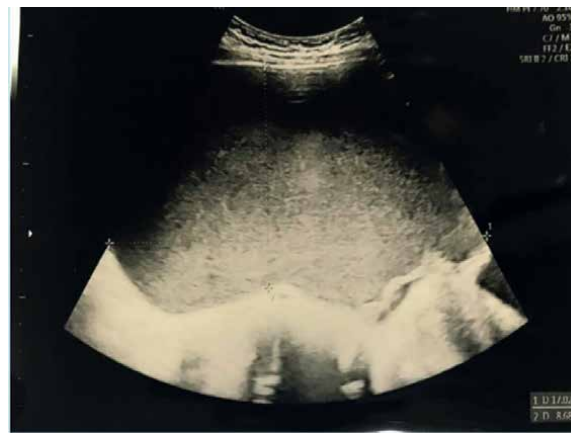

Figure 1.
Ultrasound picture of adnexal cyst with echogenic fluid.

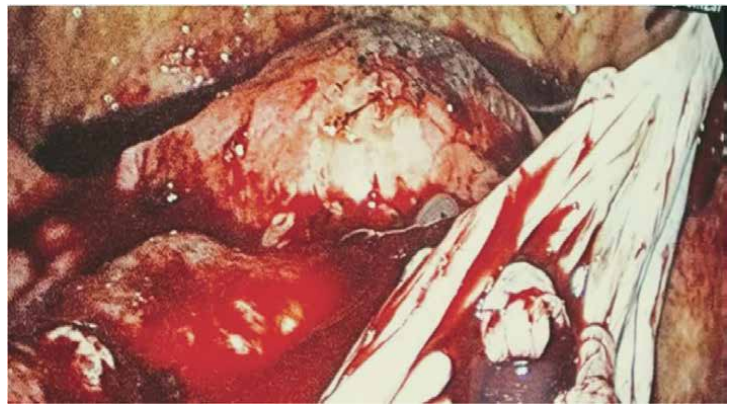

Figure 2.
Ovarian cyst wall after abscess drainage with endometriotic deposits on uterine fundus and pelvic wall.

development of an abscess in an endometrioma. Ultrasound is an easy and accurate diagnostic modality. Laparoscopic abscess drainage followed by ovarian cystectomy is the procedure of choice. An ovarian abscess is a surgical emergency and a timely intervention can save the future fertility of the patient (**Figures 1** and **2**).

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Chapter 5

Intracranial and Intraventricular Abscess – Neurosurgical Management

Marissa Tucci, Erin McCormack, Arthur Wang and Johnny Delashaw

Abstract

We will review the most common infections of the ventricular system within the neuroaxis including source, spread and clinical presentation. We will discuss the neurosurgical management of these patients including the indications for surgical management, nonoperative management, when an external ventricular drain is indicated, alternative surgical options and complications. We will review the treatment of the most common infections regarding antibiotic regimens, including when intrathecal therapy is required and how that is administered. Lastly, we will highlight the intracranial abscess, the lethal complication of rupture into the ventricular system, this pathophysiology and management of this devastating disease.

Keywords: intracranial, intraventricular, abscess, neurosurgery, infection

1. Introduction

A brain abscess (BA) is a focal infection of brain parenchyma most frequently caused by bacteria in an individual with underlying risk factors [1, 2]. Occurring at an estimated incidence of 0.33–1.3 per 100,000 per year [3–5], BAs occur more frequently in men than women, with a median age of 30 to 40 years [2, 6]. Symptoms and presentation vary drastically based on size, location, and number of foci. Lesions are typically supratentorial, with less than 10% occurring below the tentorium cerebelli [7].

The presence of an intracranial abscess can be life threatening, especially if intraventricular rupture occurs, often leading to massive cerebral edema, herniation syndrome, and death [8]. Mortality rate for uncomplicated BA is cited 0–20% [9], but intraventricular rupture of brain abscess (IVROBA) can be rapidly fatal due to malignant cerebral edema and herniation. IVROBA can occur in anywhere from 0.3–35% [10] of BA and carries a mortality rate greater than 80% [8, 11]. As neurosurgical technology has advanced, the treatment of intracranial and intraventricular abscess has evolved. With new treatment modalities for intracranial infection, in addition to

ever-changing bacteria with new patterns of antibiotic resistance, it is crucial to continually adapt and refine our approach to managing this life-threatening condition.

2. Abscess formation and pathogenesis

An intracranial abscess initially begins as an area of cerebritis, secondary to iatrogenic introduction of an infection, hematogenous spread from an infection elsewhere in the body, or from an immunocompromised state, making the host more susceptible to infection [12]. Synergistic effects have been noted between aerobic and anaerobic bacteria, especially once the blood brain barrier has been violated [9]. The early cerebritis stage lasts approximately 3 days, followed by progressive or late cerebritis which over the course of an additional 4 to 5 days becomes a capsule (**Figure 1**) [12]. The early encapsulation phase of the bacteria takes place over days 10 through 13 of the initial infection, ending in the late capsulation phase which persists after 2 weeks of untreated infection [12]. If the abscess abuts the ventricular system (**Figure 1**), the capsule is thinnest where it contacts the ventricle [12], making this area not only the most likely source of spread of the infection, but also the most lethal, as intraventricular rupture sets off an inflammatory cascade that can lead to increased intracranial pressure and, eventually, to cerebral herniation syndrome and death as previously mentioned [14]. Within an encapsulated abscess, several histologic patterns are expected, most importantly a necrotic core of infection, followed by a layer of inflammatory cells, including fibroblasts, neutrophils, and macrophages (**Figure 2**) [12]. The remaining capsule is a thick layer of collagen surrounded by neovascularization, gliosis, and cerebral edema, which results in mass effect on neighboring structures, midline shift, and elevated intracranial pressure [12]. Bacterial pathogens are by far the most common etiology of BA, and it is of paramount importance to identify the source of the bacteria as this will guide diagnostic work up and eventual therapeutic

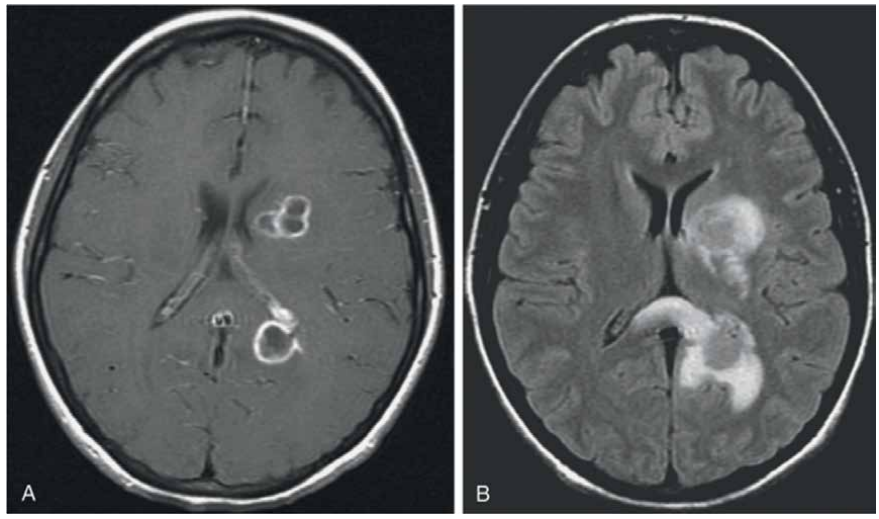

Figure 1.
Axial T1 – MRI post contrast (A) and axial fluid-attentuated inversion recovery (FLAIR) imaging (B) of a patient with multifocal intracerebral abscess. Reprinted from: [13].

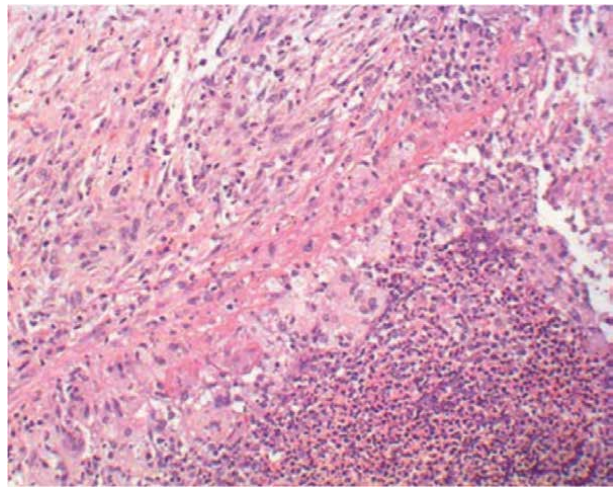

Figure 2.
Hematoxylin-eosin stain of fungal abscess—Reprinted from: [1].

management [9]. In a meta-analysis of 123 studies (n = 9699 patients) on BA, 68% of patients had a positive culture, 77% of which were monomicrobial. The most cultured pathogens were *Staphylococcus aureus* (13%), *Streptococcus viridans* (13%), *Proteus* species (7%), and *Bacteroides* species (6%) [6]. *Streptococcus intermedius* is a particularly prevalent species of *S. viridans* in BA [15] and oral *streptococci* of the *S. milleri* group has also shown dominance across the literature [15]. With regard to post operative infections, *S. aureus* has been shown in additional literature reviews to be the most common, with *S. intermedius* the most frequently isolated bacteria to form a BA amongst all comers [16]. Fungal pathogens account for only 1% of all BAs [6], and occur almost exclusively in the immunocompromised or those who have undergone prior transplant [17]. While the bacteria listed above are the most frequently isolated, it is likely that the abscesses are polymicrobial and that early initiation antibiotics and lack of standardized sample processing and culture technique affect results [9].

Hematologic spread of infection is quoted to represent between 30 and 40 percent of intracranial abscesses, with sources from the mouth, lungs, and heart being the most common [12]. Infective endocarditis, commonly caused by *S. aureus* or oral *Streptococci*, can present as cryptogenic abscess without major symptoms or bilateral disease given the dissemination from the heart. Congenital malformations of the cardiopulmonary circulation, such as pulmonary arteriovenous malformations [18] and extracardiac shunts [19], can predispose patients to BA [12]. Otogenic BA arise in the setting of chronic middle ear inflammation [20] and typically affect the temporal lobe (55%) and cerebellum (28%), with *Proteus mirabilis* isolated in up to 79% of cases [21]. Odontogenic infections, arising from poor dental hygiene, have a predilection for the frontal and temporal lobes given anatomical proximity; however bacterial spread is also hypothesized to be carried by the middle meningeal artery [16]. Interestingly, odontogenic pathogens are becoming a more common cause of BA, up to 32% in one study [3]. Dental plaque can contain more than 350 different bacterial subtypes and gingival infection and/or infection of the upper molars has been shown to place patients at higher risk for brain abscess formation [16].

3. Patient presentation and characteristics

Classic, although rare, presentation includes a nonspecific triad of headache, fever, and neurological deficits; the presence of all three is only seen in about 20% of cases [6, 22]. In the nation-wide cohort study by Bodilsen et al., most individuals studied had symptom onset 3 days prior to admission, 40% were immunocompromised and 82% had an unspecified neurologic deficit [23]. When subcategorized, motor or sensory loss was more common than cranial nerve palsy, and approximately 25% of patients presented with concern for seizure activity [23]. Compared to the Danish cohort, the literature reports a longer duration of symptoms prior to admission, 8 days on average, possibly due to access and cost of healthcare [5, 12, 22]. This emphasizes the need for increased awareness on the part of patients and providers, especially those who are at high risk for BA formation. On average each patient waited nearly 5 hours prior to first imaging and 4 days for their first neurosurgical procedure, with most undergoing two neurosurgical interventions on average [22]. With regard to imaging modality of choice a prospective study of 115 patients, of which the majority had a brain abscess, demonstrated that magnetic resonance imaging (MRI) with diffusion –weighted imaging (DWI), had a positive predictive value of 98% and a negative predictive value of 92%, emphasizing its utility in differentiating between abscess and metastatic disease [1]. While MRI is preferred, a post-contrast computed tomography (CT) can be used expeditiously to elucidate if an intracranial abscess may be present (**Figure 3**) [9]. CT or MRI imaging typically

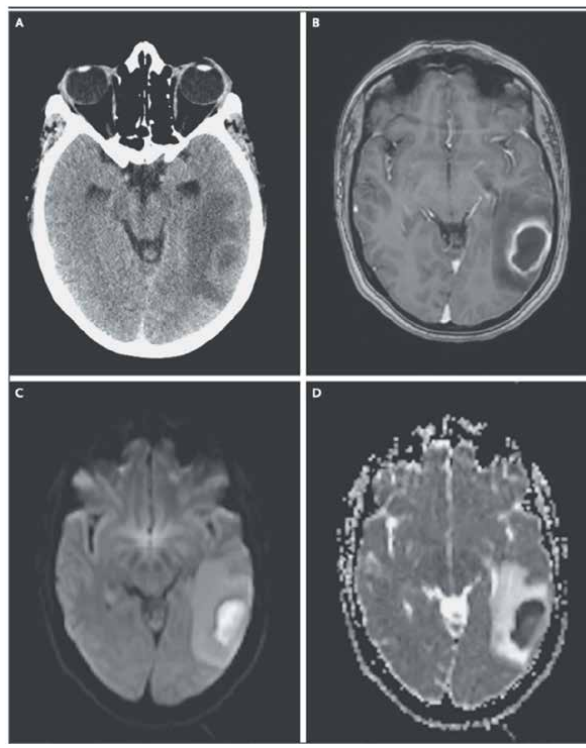

Figure 3.
Post contrast CT imaging (A), T1- weight MRI (B), DWI (C) and apparent-diffusion-coefficient (D) imaging of intracranial abscess—Reprinted from: [1].

demonstrates a ring-enhancing lesion, with MRI revealing a bright lesion with surrounding hypodense rim with surrounding edema as mentioned [24]. Other nonspecific findings on presentation include nausea/vomiting, altered consciousness, seizures, cerebellar involvement, gait instability, and meningeal signs [2, 6]. The nonspecific presenting symptoms might explain why only about 17% of patients with BA have central nervous system (CNS) infection listed as their admission diagnosis and requires increased vigilance on the part of the provider to keep a BA in their differential when an individual presents with any source of infection [1, 22]. As mentioned above, immunocompromised state, post-solid organ or hematologic transplant, and/or use of immunosuppressant medications, congenital cyanotic heart disease and arteriovenous malformations are all predisposing conditions to BA [1, 22]. More commonly, odontogenic or otologic infections, endocarditis, head trauma or history of neurosurgery, cancer, diabetes mellitus and alcohol abuse can also lead to BA [22].

4. Medical management

The mainstay of medical management is early initiation of empiric antibiotics, with prompt transition to targeted therapy upon resulting cultures; however, 6 weeks of polymicrobial coverage is recommended in non-endocarditis cases as more often than not BA are truly polymicrobial (**Figure 4**) [9]. Expedited treatment may reduce mortality from BA from 40–10% [16]. While blood and, if safely able, CSF cultures, are obtained from patients presenting with concern for BA, a causative bacterium is not always isolated, with sterile cultures cited at approximately 25% [24]. If the solitary abscess is less than 1 centimeter (cm) in size, empiric broad spectrum antibiotics can be continued for 6–8 weeks without neurosurgical aspiration, followed by a prolonged course of oral antibiotics [16]. In clinically stable patients, antibiotic administration may be postponed until definitive neurosurgical management, provided such intervention occurs promptly (within 24 hours) and blood cultures are drawn [22].

Empiric antibiotic regimens typically consist of vancomycin, a third-generation cephalosporin, and metronidazole which can later be narrowed according to cultures; however, this remains controversial [16]. In patients with acquired immunodeficiency syndrome (AIDS), an empiric regimen of sulfadiazine, pyrimethamine, and leucovorin targets *Toxoplasma gondii*. If a fungal pathogen is suspected, liposomal amphotericin B and flucytosine may be utilized [24]. Antibiotics are administered intravenously for 6 to 8weeks [16]. Complete surgical removal of abscess and capsule can reduce required duration [24]. Early transition to oral antibiotics may decrease risks associated with prolonged hospitalization [25], but overall efficacy has not been proven. Importantly, conversion to an oral regimen, depending on the causative bacteria, may lead to increased drug toxicity, antibiotic resistance, and diarrheal infection with *Clostridium difficile*, further complicating patient recovery [22]. Importantly, side effects and toxicity from intravenous medications also pose a risk which must be weighed against the benefits of prolonged treatment and narrowed as soon as medically appropriate [9].

Adjunctive treatments have been studied, however are not yet included in standard of care management. Corticosteroids have a well-established role in the treatment of bacterial meningitis, with a demonstrated reduction in mortality [26]. Initial animal studies suggested impaired antibiotic penetration [27] and delayed

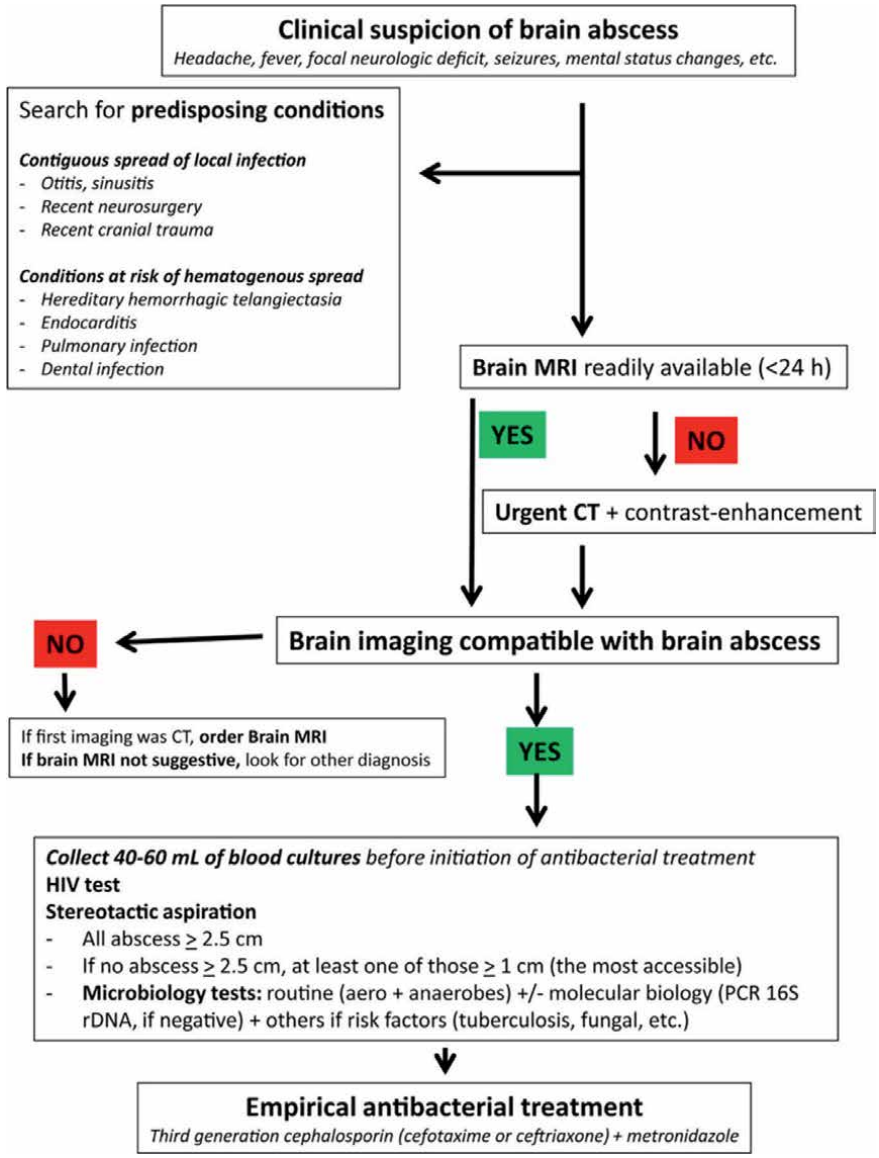

Figure 4.
Schematic flow diagram for diagnostic work up and therapeutic management of brain abscess. Reprinted from: [12].

capsule formation [28]. A more recent systematic review and meta-analysis found a non-statistically significant mortality benefit [29]. Despite a paucity of robust data supporting their use, corticosteroids are recommended in cases of life-threatening edema with mass effect [9]; however, use of high dose steroids may alter the radiographic findings, namely surrounding ring enhancement/edema, leading to false sense of improvement [30]. If utilized in these scenarios, duration should be minimized to mitigate the sequelae of immunosuppression [30]. Hyperbaric oxygen therapy has shown promise in small trials [31], potentially via improved antibiotic bactericidal activity [32].

5. Surgical management

Stereotactic aspiration is the treatment of choice for abscesses greater than 1 cm in diameter, regardless of location given significant advancement in technology and stereotaxis; open surgical management is first considered for lesions larger than 2.5 cm however other factors should be considered for open surgery including causative bacterium and superficial, easily accessible location [1]. Exceptions to stereotaxic aspiration include imaging without evidence of central cavitation, periventricular lesions, lesions with significant mass effect/impending herniation and lesions in patients with human immunodeficiency virus (HIV) with probable toxoplasmosis, who can be treated with antimicrobials following antibody results [1]. Resection may be considered for superficial abscesses in non-eloquent tissue, especially in cases of suspected tuberculosis, fungal, or branching bacteria; ultrasonography has grown in popularity for superficial lesions if stereotaxis is not available [1]. A systematic review and meta-analysis of 124 cases found that in superficial non-eloquent locations, resection was associated with several positive characteristics including decreased postop residual abscess, decreased re-operation rate, shorter duration of both post-op antibiotics and length of hospital stay, and higher rate of improved neurologic status at 1 month [33]. However, mortality and neurologic status at 3 months did not differ between those treated with aspiration versus resection [34].

At the index procedure, following maximum aspiration of the abscess, leaving a drain within the abscess cavity may promote decreased reoperation rates and allow for intracavitary antibiotic delivery, neither of which are currently included in standard management [1]. As mentioned, intracavitary antibiotic delivery has been used for refractory fungal abscesses but the literature has not proven this treatment to be effective across this pathology [24]. Indications for excision after initial stereotaxic drainage include failure to improve in 7 days, altered mental status, symptomatic elevated intracranial pressure, progressively expanding abscess, especially if migrating towards the ventricle, or if there is failure of abscess shrinkage within 2 weeks' time [24]. Several studies have explored the feasibility of bedside twist drill aspiration, specifically in peripherally located abscesses sized less than 2.5 cm [35]. A series of 103 patients treated with bedside twist drill aspiration found a mortality rate of 4.8% [36], comparable to the reported mortality rate of typical aspiration.

6. Complications

Acute complications, including intraventricular rupture and herniation, are discussed below. Aside from acute new neurologic deficit, decision making regarding interval imaging can vary. In their review in *The New England Journal of Medicine*, Brouwer et al. suggest interval cranial imaging following 1–2 weeks of initial management if clinical improvement is not apparent with biweekly imaging until improvement is seen, up to 3 months after initiation of therapy [1]. For individuals with either an identifiable or unidentifiable bacteria, septic emboli may cause remote infarcts in various organs, including the brain, with areas of ischemia prone to subsequent abscess formation, complicating treatment and recovery [24].

BA are not without morbidity, as permanent cranial nerve palsies, vision loss, hemiparesis, learning/cognitive deficits, and hydrocephalus are amongst the life-changing side effects of BA diagnosis and treatment [30]. Most importantly, the after-effects of BA formation regarding neuropsychological and psychological testing are

not well researched [1]. Excision of BA can cause an increased risk of epilepsy (41 vs. 20%), which subsequently increases mortality; however routine empiric use of anti-epileptic medications on presentation is still under investigation [23]. Interestingly, BA has also been associated with increased risk of cancer in the first 10 years after diagnosis (HRR 2.09 [95% CI 1.79–2.45]) [37].

7. Prognosis

A nationwide, population-based cohort study in Denmark from 2007 to 2020 (n = 485 cases) reported a 6% mortality rate at discharge to 12% at 6 months [22]. Prognostic factors impacting overall mortality from BA include IVROBA, abscess size larger than 3 cm, and age older than 65 years of age, with oral flora having a favorable impact on mortality in this study possibly secondary to susceptibility to oral antibiotic regimens [22]. Above all else, prevention of IVROBA is most critical; however, immunocompromised state, hematologic spread, and age above 65 have been associated with mortality specifically within 6 months following hospital discharge and/or poorer prognosis [22, 34]. Factors associated with poor outcome in surgically managed patients include diabetes, ventricular rupture, and supratentorial abscesses located in eloquent brain [38]. In the population of patients managed nonoperatively, neck stiffness or meningismus, septic shock and lower admission Glasgow Coma Score (GCS) were associated with poorer prognosis [39]. For the immunocompromised s/p hematologic or solid organ transplant, fungal BA hold a near 100% mortality rate [24]. Without IVROBA, since the increased utilization of CT/MRI, the mortality from BA has decreased from 40 to 60% to approximately 10%, although morbidity remains high [24]. Favorable prognostic factors across the literature have included GCS of 12 or greater as well as absence of criteria for septic shock [39].

8. Intraventricular abscess

Primary intraventricular abscess typically forms via slow growth from an area of cerebritis or ventriculitis [8]. A longitudinal single-center study on BA between 1986 and 2005 in Taiwan identified 179 patients with bacterial brain abscess, 62 of whom had IVORBA (45 on initial presentation, 17 later in hospital course). Pathogens implicated in IVORBA included *Synema viridans* (n = 10), mixed infection (10), and *Klebsiella* (6), although 13 cases had negative cultures [40]. Regardless of etiology, patients present with severe HA, meningeal irritation, and rapid deterioration usually within 10 days of the first presentation of meningeal signs [41]. emphasizing the importance of early diagnosis and treatment. Importantly, up to 34.6% of patients with BA can have concomitant meningitis, clouding the clinical picture and potentially complicating diagnosis [40]. Radiographic clues for IVROBA found on CT include intraventricular debris layering within the occipital poles and/or temporal horns, as well as ependymal enhancement within the ventricle (**Figure 5**) [41, 42].

Several factors can increase the risk of intraventricular rupture. For example, when compared to unilocular abscess, the risk of rupture is 4.2 times higher in multiloculated BA [10]. Strikingly, every 1 millimeter (mm) reduction in distance to ventricle has been associated with increased risk of rupture by 10% [40]. The proposed mechanism for preferential rupture into the ventricles rather than subarachnoid space is differential blood supply, resulting in reduced thickness of the abscess

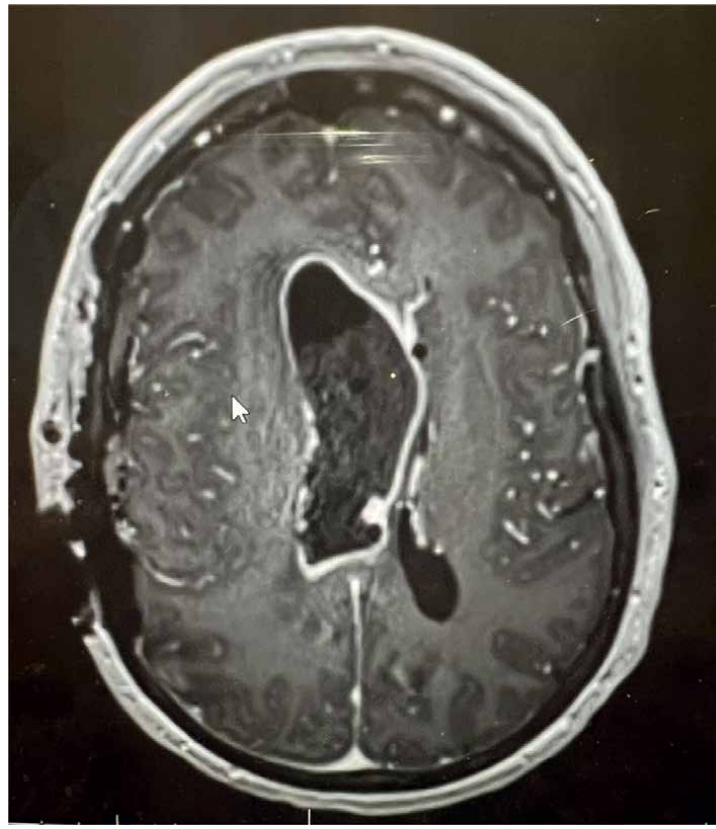

Figure 5.
Axial T1-W post contrast image of a polymicrobial intraventricular abscess. Reprinted from collection of Dr. Arthur Wang, Tulane Department of Neurological Surgery, New Orleans, LA, USA.

wall along the ventricles as previously mentioned [8]. Abscesses originating from hematogenous spread and those located in the temporal lobe have been shown to rupture the fastest [10, 39]. As such, aggressive CT-guided aspiration of deep abscesses, especially located in the parieto-temporal region improves outcome [14]. The danger of IVROBA is not limited to the days following the event, as it has been associated with 3.48 increase in relative risk for mortality 6 months after hospital discharge [22]. Identifying patients at risk for rupture is critical to optimize medical management and prioritize surgical intervention for BA with high-risk features, such as proximity to the ventricles.

9. Treatment of IVROBA

Additional treatment considerations are required when BA is complicated by intraventricular rupture. Pending up on the clinical status of the patient following abscess rupture, the role of external ventricular drain (EVD) placement and intrathecal antibiotics remains controversial [10, 43]. More often than not patients with IVROBA decline rapidly to a comatose state, partially due to the rise in intracranial pressure [43]. Mortality secondary to IVROBA has been estimated between 84 and 100% and has not declined as the mortality of unruptured brain abscesses has

declined [8, 43]. Some authors suggest that emergent placement of an EVD, followed by ventricular irrigation and intrathecal antibiotic therapy plays a role in overall survival however given the high mortality, this treatment strategy remains underexplored [8, 39]. In reviewing case studies of IVROBA survivors present within the neurosurgical literature, Omar et al. describes varying methods of antibiotic delivery including intravenous, intrathecal via EVD or lumbar drain, and into the abscess cavity directly as well as varying methods of drainage from needle aspiration to open decompressive craniectomy [10]. Antibiotic courses ranged from 31 to 180 days intravenously and up to 42 days via intrathecal administration, which some authors feel decreases the likelihood of ventricular septations [10]. The authors summarize that 67% of surviving patients required EVD placement with 29% eventually requiring ventriculoperitoneal shunt placement [10]. Of note the length of EVD drainage ranged amongst institutions as did the prevalence of systemic alone versus systemic and intrathecal antibiotics; 43% of survivors receiving both systemic and intraventricular therapies [10]. Zeidman et al. describes a case of survival from IVROBA with combined medical and surgical management including immediate craniotomy with abscess and ventricular irrigation and debridement followed by an extensive course of intravenous and intrathecal antibiotics with routine exchange of the ventriculostomy catheters [43]. This case taken with the systematic literature review by Omar et al. highlights the need for further research into the standard of care for the neurosurgical emergency that is IVROBA.

Intrathecal antibiotics are more often utilized for ventriculitis and/or meningitis as they reliably result in sterilization of the cerebrospinal fluid and have demonstrated safety both via ventricular catheter and lumbar drain [14]. Takeshita et al. published a protocol for both intrathecal and intravenous antibiotic therapy that reduced mortality rate from IVROBA in their study to 38.7% [14]. The authors describe aspiration for abscesses larger than 2 cm with placement of an EVD if the patient demonstrated hydrocephalus and or ventriculitis, with VPS placement delayed for 8 weeks [14]. Of clinical importance, the authors paid attention to, often subtle, meningeal signs and noted that IVROBA was associated with progressive headache, decline in neurologic status, and increased meningeal signs [14].

Aside from neurologic decline, cerebral herniation syndromes and malignant cerebral edema, additional complications from IVROBA include septic arteritis, noncommunicating hydrocephalus with possible trapped ventricles and ventricular septations or loculations that prevent physiologic circulation of cerebrospinal fluid [11]. While early aggressive ventricular irrigation at the index surgery may help prevent this complication [11, 44], patients may require further procedures such as ventriculoperitoneal shunt placement or endoscopic exploration to break up the septations [11]. Although patients with meningitis and ventriculitis who were treated with intraventricular or intrathecal antibiotics had lower relapse rates, the standard of therapy administration is not well supported by the literature [10]. A retrospective cohort study (n = 105) demonstrated an 88.4% CSF sterilization rate in ICU patients with meningitis or ventriculitis who were treated with intraventricular antibiotics [45]. Remes et al. studied IVT via external ventricular drain (EVD) or lumbar drain (LD) in post-neurosurgical patients with meningitis and ventriculitis with similar efficacy. Most notably, the study reported a mean time to CSF sterilization of 2.2 and 2.6 days in EVD and LD groups, respectively [30].

With the evolution of neurosurgical technology and increased sophistication of our intracranial pressure monitors, continuous ventricular irrigation has been introduced for the management of intraventricular hemorrhage, ventriculitis, amongst

other pathologies. For example, Hess et al. reports a case of IVROBA treated successfully with intraventricular vancomycin via a bilateral IRRAflow® catheter [46]. Prospective studies are needed to establish the optimal patient population, as well as the efficacy and safety of this route of administration.

10. Conclusion

IVROBA remains at the top of the list of neurosurgical emergencies given its high mortality rate however the persistent difficulty in diagnosing BA requires increased awareness on the part of all providers to consider BA in the differential for all patients, especially those with an infection. As neurosurgical technology has advanced, the mortality rate for BA has decreased, with a role for both medical and surgical management. Preventing IVROBA requires ongoing research and dedication to reduce the mortality, and hopefully at the same time morbidity, of BA for all patients.

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An abscess is a collection of pus in any part of the body. There are two different types of abscesses: skin abscesses and internal abscesses. Skin abscesses (cutaneous abscesses) develop under the skin. They are common and typically easy to treat. Internal abscesses develop inside an organ or in the spaces between organs and can be more difficult to identify than skin abscesses. The symptoms can be vague and there may be no obvious external signs of a problem. This book provides a comprehensive review of the different types of abscesses along with their causes and treatments.

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