CHAPTER 15
COMMON BACTERIAL INFECTIONS IN CHILDREN

Iftikhar Ahmad Jan
Kokila Lakhou

Introduction
Bacterial infections are the cause of significant mortality and morbidity in children. Infections of surgical importance may affect virtually any organ or tissue in the body. These may be community or hospital acquired. The major groups of community-acquired infections are skin and soft tissue infections, bone infections, and infections of specific organs. Hospital-acquired infections may further be classified as infections of surgical wounds, infections in wards, and infections in immunocompromised and critically ill patients. Bacterial infections are more common at extreme ages, and thus babies less than 2 months of age are highly susceptible to bacterial infection. Other conditions such as malnutrition, immune-deficiency states, and prolonged illnesses, make children more susceptible to acquire infections.

Skin and Soft Tissue Infections
Skin and soft tissue infections are better described according to the depth of tissue involved. Common bacterial infections in children are impetigo, scalded skin syndrome, folliculitis, furuncle or boil, carbuncle, erysipelas, necrotising fasciitis, clostridial myonecrosis (gas gangrene), nonclostridial myonecrosis, synergistic gangrenes, lymphadenitis, and abscesses. In addition, ear and throat infections and infections of specific systems (e.g., urinary tract infections, respiratory tract infections) may be important in the management of paediatric surgical patients.

Impetigo
Impetigo is an infection of the superficial layers of the skin. It is caused by a minor breach in skin continuity and is common in babies with poor hygiene, in crowded living conditions, and living in warm and humid areas. Impetigo is a disease of babies and children, and may constitute 4–6% of all bacterial infections in the paediatric population. Underlying conditions, such as eczema, insect bites, small cuts, or abrasions, may initiate the process.

Impetigo may be described as bullous and nonbullous, according to the presentation. Nonbullous impetigo constitutes more than 70% of impetigo infections. It presents as a thick, honey-colored crust on the face or limbs. It may be mildly painful, but other constitutional symptoms are not present. Healing is spontaneous and usually does not lead to scarring, it is mostly caused by Staphylococcus aureus or Streptococcus pyogenes. Bullous impetigo is less common and is often seen in babies and young children due to their soft skin. It is mostly caused by Staphylococcus aureus and presents as blister-like lesions filled with fluid pus. Both varieties of impetigo are diagnosed by the classic appearance, and investigations are not necessary. A swab from the lesion may cause growth of the causative organism. Nasal swabs from patients and mothers may help, however, in identifying the source in patients with repeated appearances of new lesions.

Topical antibiotics, such as fuscidic acid, mupirocin, or polymyxin-B, may help in early healing of the lesions. Most impetigos heal without any sequel, but such complications as toxic shock syndrome may be seen in immune-compromised cases. Poststreptococcal glomerulonephritis and rheumatic fever are threats that warrant early and adequate treatment of skin lesions in children.

Folliculitis
Folliculitis is an infection of the hair follicle. It usually presents as a small tender nodule of the hair follicle. It is commonly caused by Staphylococcus aureus. Two types of lesions are seen. In superficial folliculitis, multiple hair follicles are involved and cause small pustules at the opening of the adjacent hair follicles. The deeper form of folliculitis affects a single hair follicle and causes local swelling and tenderness. These are usually seen on the scalp in children. There is local tenderness but no fever or other constitutional symptoms. It is usually caused by local trauma, sweating, friction, and local lesions such as eczema. Hot tub folliculitis is caused by Pseudomonas aeruginosa and occurs in hot tubs and pools with improper cleaning and disinfection. Folliculitis is a self-limiting condition in most cases but may progress to form furuncle, which is a severe infection of the hair follicle and appendages. The treatment of folliculitis is by cleaning and topical antibiotics. In immunocompromised patients and diabetics, systemic antibiotics should be started to avoid any complications.

Furuncle or Boil
Furuncles are severe infections of the hair follicles, sweat glands, and surrounding tissues. They may occur anywhere in the hair-bearing areas of body, but they are most common on the face, neck, armpits, buttocks, and thighs. Staphylococcus aureus is the causative organism in most cases, but other organisms may be involved. Furuncles usually start as painful nodules and develop into large inflamed and tender areas with constitutional symptoms of fever, malaise, and anorexia. They are uncommon lesions in the paediatric population, but may be seen in patients with immune deficiency and diabetes mellitus. Multiple lesions may occur, especially after inadequate treatment. If untreated, the lesion may lead to spreading cellulitis and can be dangerous in areas such as the nose and face, where it may lead to thrombosis of the cavernous sinus with serious consequences.

Diagnosis is obvious by the classic appearance of the lesion, which initially presents as a tender lesion with a wide red indurated area and an obvious central punctum. Pus expressed through the head of the lesion gives relief of symptoms. Furuncles are especially dangerous in immunocompromised patents. Multiple crops of the lesions may occur and combine to form a carbuncle. Furuncles need treatment by oral antibiotics, cloxacillin, amoxicillin clavulanate, macrolides, or cephalosporins. Once the abscess has drained, the patient improves rapidly; however, antibiotics treatment should be continued until complete healing of the lesion to prevent new lesions.

Carbuncle
A carbuncle is a spreading infection in the subcutaneous tissue planes caused by multiple infected hair follicles. Carbuncles are rare in children but may be seen in hairy and immunocompromised patients; these
usually occur in the neck. It is characterised by extensive necrosis, with multiple abscesses that drain on the surface by multiple sinuses and ultimately unite to form a large area of tissue necrosis. *Staphylococcus aureus* is the causative organism in most cases. Treatment consists of drainage of abscesses and excision of the dead and necrotic tissue. Intravenous broad-spectrum antibiotics help in the control of infection. Extensive tissue destruction may require skin grafting in few cases.

**Cellulitis**

Cellulitis is a spreading infection in the subcutaneous tissue planes. It may occur after a small skin breach, especially in immunocompromised children. Insect bites, local trauma, vascular insufficiency, and diabetes are some of the predisposing factors in the causation of cellulitis. Cellulitis may also occur secondary to other soft tissue infections such as furuncles or carbuncles. Classic signs of inflammation include redness, pain, swelling, warmth, and loss of function. The skin overlying the affected area is shiny and red. The patient may develop fever with chills, malaise, and body aches. It is important to identify the underlying primary pathology, such as diabetes. *Staphylococci*, *streptococci*, and even gram-negative organisms may be responsible for the lesion. The goals of treatment are control of infection and prevention of complications. Treatment includes correction of contributing factors, broad-spectrum antibiotics, and analgesics. Hospitalisation may be required in extensive involvement. Facial cellulitis, also called erysipelas, is a serious condition and needs hospitalisation and intravenous antibiotics. Spreading cellulitis in the floor of the mouth may cause Ludwig’s angina, which may threaten the life of the patients due to laryngeal oedema and airway obstruction. Steroids may be required along with antibiotics, and in some cases a tracheotomy may be required to relieve airway obstruction.

**Pyomyositis Tropical**

Pyomyositis tropical is a fulminant infection of the muscles. It is caused by *Staphylococcus aureus*, but other organisms such as streptococcus and gram-negative organisms may be responsible. Multiple abscesses may be formed in different parts of the body. The child presents with high-grade fever and chills with abscess formation. Abscesses are formed in various muscles of the body. The infective process may affect other tissues of the body; meningitis, pericardial effusions, and endocarditis may be present. Empyema thorax is another serious complication of pyomyositis tropical. The disease is commonly seen in immunocompromised patients, especially those with human immunodeficiency virus (HIV) and those on chemotherapy. Blood cultures will grow the organism and also help in deciding appropriate antibiotics. Antibiotics of choice for pyomyositis tropical are cloxacillin, streptococci, and even gram-negative organisms are responsible for the lesion. The goals of treatment are control of infection and prevention of complications. Treatment includes correction of contributing factors, broad-spectrum antibiotics, and analgesics. Hospitalisation may be required in extensive involvement. Facial cellulitis, also called erysipelas, is a serious condition and needs hospitalisation and intravenous antibiotics. Spreading cellulitis in the floor of the mouth may cause Ludwig’s angina, which may threaten the life of the patients due to laryngeal oedema and airway obstruction. Steroids may be required along with antibiotics, and in some cases a tracheotomy may be required to relieve airway obstruction.

**Necrotising Fasciitis**

Necrotising fasciitis (NF) is a fulminant soft tissue infection causing extensive fascial, fat, and muscle necrosis. Due to extensive tissue destruction, it has been described as the “flesh-eating disease”. The condition is not common and is mostly seen in malnourished, immunocompromised, and debilitated patients. Two types are described: Type-I NF is a polymicrobial synergistic infection caused by anaerobes (bacteroides and peptostreptococci), facultative anaerobes (non–beta-haemolytic streptococci) and *Enterobacter species* (*Escherichia coli*, *Enterobacter*, *Klebsiella*, and *Proteus*). Type-II NF is caused by group A beta-haemolytic streptococcal (GABHS) infection, and is often described as streptococcal myonecrosis. The true incidence of NF is not known; however, the disease is commonly seen in children in the developing countries.

**Type-I Necrotising Fasciitis**

Various forms of type-I NF include Melaney’s progressive bacterial gangrene, Fournier’s gangrene, cancrum oris, and noma vulva. In children, severe systemic diseases, such as gastroenteritis, sepsis, gut perforations, and omphalitis, may predispose the patient to infective gangrene. Type-I NF involves the abdominal wall, perineum, groin, and postoperative wounds. Rarely, it may affect the oral cavity. In newborn babies, umbilical infection secondary to poor hygiene can cause rapidly spreading gangrene of the umbilicus. Initially, erythema and hyperaemia develop in the affected area and rapidly spread to local tissue necrosis and may spread to the abdominal and chest walls. Most babies have polymicrobial infections. Multiple organisms are usually recovered from the patients, and the number of isolates varies from two to six, averaging 3.5 isolates per specimen. Patients need aggressive supportive therapy along with a combination of antibiotics from gram-
positive, gram-negative, and anaerobic coverage. Usually, a combination of third-generation cephalosporin with metronidazole is used. Extensive tissue debridement is necessary in most children. Mortality remains high, and more than 50% of patients with Type-I NF may die secondary to overwhelming sepsis.

**Meleney's progressive bacterial gangrene** is a form of necrotising fasciitis caused by microaerophilic streptococci, aerobic staphylococci, bacteroides, and gram-negative organisms. It is usually seen as a complication after abdominal surgery, especially after bowel surgery, abdominal abscess drainage, mass abdominal closure under tension, and surgical drains. The symptoms appear one to two weeks after the initial procedure. The skin around the wound becomes red and tender, with a foul-smelling discharge. Wide areas of skin and tissue necrosis then occur, and the patient may become very sick and toxic. Aggressive resuscitation and use of a combination of broad-spectrum antibiotics are necessary. Wound debridement should be done as soon as the patient is stable, and the wounds left open for adequate drainage of infected material. With the removal of dead and necrotic tissue, the patient’s condition may improve. Mortality, however, remains high, especially in diabetics, immunocompromised patients, and malnourished children. See Chapter 21 for additional information.

**Fournier's gangrene** is a polymicrobial synergistic gangrene. This usually affects the perineal area (Figure 15.1). It starts as a small itching area, which forms a small ulcer, and then widespread tissue necrosis occurs. The condition is initially painful but pain subsides with the loss of skin and subcutaneous tissue.

Figure 15.1: Fournier's gangrene.

Cancrum oris and noma vulva are mucocutaneous gangrenes affecting the mouth and vulva. These occur in severely malnourished children with infectious diseases such as gastroenteritis, measles, and chicken pox. A polymicrobial fulminant infection occurs, causing extensive skin and soft tissue necrosis. The progression of the inflammatory process is rapid, and extensive tissue destruction may be noted in a few days. This may cause serious disfigurement of the face, and extensive surgical procedures may be required later to cover the facial defects. The patient should be managed by correction of malnutrition, control of infection with broad-spectrum antibiotics, and tissue reconstruction. Corrective surgery should be performed only when the patient has fully recovered, to avoid any recurrence.

**Type-II Necrotising Fasciitis**

Children with varicella zoster infection are at a high risk of developing GABHS-associated necrotising fasciitis. Children younger than 10 years of age who have chicken pox have a 58-fold risk of developing invasive GABHS infections. The infections usually appear 4–6 days after the onset of a rash in children with chicken pox and may cause widespread tissue necrosis. Mortality is high, and aggressive management is needed to control infection and save the life of the patient. Mortality in children after NF may be from 10% to 20%. The clinical presentation in GABHS-associated necrotising fasciitis may start as the flu-like symptoms of fever with chills, malaise, and pain, but the patient soon develops signs of toxicity, tachypnoea, local tenderness, and erythema. Severe pain in the affected area with constitutional symptoms is an important indicator of a rapidly developing fulminant infection. Local signs, such as redness, induration, skin color changes, and pouring of pus, are indicative of serious tissue infection.

**Nosocomial Infections**

Nosocomial infections are those caused by a hospital stay but not related to the patient’s original condition. Infections are considered nosocomial if they appear after 48 hours in hospital or within 30 days after discharge from hospital. Nosocomial is a Greek word and comes from *nosos*, which means “disease”, and *komeo*, which means “care”. Nosocomial infections are now considered one of the major causes of morbidity and mortality in patients with prolonged hospitalisation. In developed countries, the incidence of nosocomial infections ranges from 7% to 14%; it is higher in developing countries with limited resources. The most common sites of nosocomial infections are the urinary tract, surgical wounds, cannula sites, and respiratory tract. There are at least five modes of transmission of infections in admitted patients, including direct and indirect contact, droplet infections, as well as airborne, vehicle-borne, and vector-borne infections.

Direct contact between patients (hand shaking, sitting together, or sharing beds) may transfer bacteria from one patient to another. In these cases, the patient harboring the bacteria acts as a source and the one infected is the target. Indirect infection occurs by the use of various intermediate substances, such as infected instruments, gloves, syringes, dressings, and so forth. Droplet infections occur by cross infection when coughing, sneezing, and talking. Vectors (e.g., flies, mosquitoes, and rodents) may also cause transmission of infection in the admitted patients. Vehicle-borne infections are secondary to food products, water, and ward equipment. Hospital staff who are harbouring the infection are another important source of infection. The incidence of nosocomial infections rises with the duration of the stay in hospital.

Various factors, such as premature birth, advanced age, immunodeficiencies, indwelling catheters, prolonged antibiotic therapy, and repeated blood product transfusions, predispose patients to nosocomial infections. The most important factor, however, is the lack of cleanliness in the wards. Prevention is the mainstay in warding off nosocomial infections. Prevention may be achieved by avoiding direct contact among patients and isolating patients with any active infection. Open wounds and contaminated utensils are the main cause of cross infections in the hospitals. All patients with open wounds should be treated with utmost care; any cross contamination may be avoided by using disposable lines, gloves, and gowns. In case of an outbreak of ward infections, the wards should be closed immediately and properly fumigated, and all trolleys, beds, and utensils should be fumigated. Any source of infection in the ward staff should be identified by nasal swabs and armpit cultures, and staff with positive cultures should be treated before returning to work. Staphylococci are the commonest organisms that may stay for a long time in the naris of carriers. In affected patients, local fuscidin crème should be used until the cultures are negative.

It has now been proven without a doubt that the incidence of nosocomial infections can be minimised significantly by hand washing before coming in contact with the patients. Therefore, hand washing must be ensured in all wards, and hand-washing areas should be present in accessible locations in the wards. Chlorhexadine lotion may help in the prevention of infection if the water supply is scarce. In developing countries, the use of disposable gloves may not be possible for all patients, but must be used for patients with open and infected wounds, for the safety of the other patients as well as the treating personnel.

**Abscesses**

An abscess is a collection of pus in a cavity surrounded by a pyogenic membrane. Pus is composed of necrotic leucocytes, tissue cells, and bacteria. Abscess formation occurs secondary to bacterial invasion of tissue. The bacteria multiply rapidly in the tissue and initiate an inflammatory
response, which includes increased blood flow in the tissue and attraction of inflammatory cells, especially leukocytes. The capillary becomes porous, and plasma proteins, especially fibrinogen, is released into the tissue spaces. Fibrinogen forms a fibrin plug to restrain the invading organism. The neutrophils start phagocytosis of bacteria, release proteolytic enzymes, and ultimately undergo necrosis. The abscess cavity is isolated from the surrounding tissue by the formation of pyogenic membrane. The proteolytic enzymes digest the dead tissue and give liquid consistency to pus. With the progression of the inflammatory process, granulation tissue is formed around the abscess cavity. The granulation tissue helps to prevent the spread of bacteria and inflammatory processes into the surrounding tissue, but it also prevents adequate concentration of antibiotic penetration into the tissue, making antibiotics less effective. Bacteria have a limit to proliferation, and after the maximum number of bacteria per unit volume has been achieved, further proliferation is stopped. Bacteria become less active and thus less vulnerable to antibiotics. The production of various enzymes, such as beta-lactamase, also cause a breakdown of the antibiotics. As the inflammatory process progresses, macrophages appear in the inflammatory zone and start the process of demolition. These macrophages then replace the neutrophils within the pyogenic membrane and ultimately start healing by secondary intention to leave a residual scar.

**Locations**

Abscesses may form anywhere in the body (Figures 15.2 and 15.3). The main sources of abscess formation are open or penetrating wounds, local extension from an adjacent focus of infection, haematogenous spread, or infections via lymph vessels and lymph nodes. The infecting organism varies according to the site and source of infection. Most abscesses on the skin and subcutaneous tissue are caused by *Staphylococcus aureus*. Causative organisms in deep abdominal abscesses depend upon the source of infection. In colon and appendix perforations, the abscess is usually polymicrobial and may cause fulminant infection or serious necrotising fasciitis. This form of infection is not common in the paediatric population; however, malnourished, immunocompromised children and those on chemotherapy are at a high risk of such infections.

**Diagnosis**

The diagnosis of a surface abscess is obvious by its classic appearance, local pain, redness, tenderness, and central yellow punctum, from which it may drain if not treated adequately. An internal abscess may be difficult to diagnose due to overlapping symptoms with the preceding illness. The key diagnostic features are persistent fluctuant pyrexia, chills, and lack of appetite.

**Treatment**

Treatment of abscesses anywhere in the body is drainage. This holds true for most abscesses except for small lesions. If not drained in time an abscess tends to find its own path in the area of least resistance. It may therefore drain itself externally onto the surface, or internally as seen in subcutaneous tissue abscesses, or internally into other hollow organs such as the gut or urinary bladder. Spontaneous drainage of an abscess may have serious consequences, and every effort shall be made to control infection and drain the abscess surgically. This may be achieved for surface abscesses by making an incision, or for internal abscesses, by percutaneous drainage by ultrasound or computed tomography (CT) guidance. In some cases, an internal abscess will need exploration if it is not in an accessible area for percutaneous drainage. The surface abscess cavity should not be closed after drainage, as pus continues to form for days and weeks, and even after adequate drainage this may cause a recurrence in closed wounds. Repeated drainage of an internal abscess may be required. All abscesses must be sent for culture and sensitivity to ensure proper antibiotic coverage after drainage.

The role of antibiotics in an abscess is to control spread of infection. Once the abscess has been drained, the patient usually recovers rapidly, but antibiotics should be continued to prevent the spread of infection, especially in debilitated and immunocompromised patients.

**Intraabdominal Abscess**

Intraabdominal abscesses are commonly seen as a sequel of abdominal surgery; however, gut conditions, such as Crohn’s disease, ulcerative colitis, and abdominal malignancies, may also cause a primary intraabdominal abscess. Abdominal abscesses are also seen after gut perforations secondary to trauma, penetrating injuries, and infective processes such as necrotising enterocolitis, enteric fever, and appendicitis. Immunocompromised patients are at a high risk of developing abdominal abscesses. In these patients, due to their altered immunological response, gut flora may grow rapidly and bacterial translocation may cause intraloop abscesses. The clinical features of intraabdominal abscesses depend upon the causative factors. In postoperative cases and bowel resection, the patient may initially show good recovery, but between 5 and 10 day postoperative, the patient develops intermittent fever, local tenderness, abdominal distention, and a palpable lump in the abdomen. In pelvic abscesses, rectal examination will reveal a tender bulge anteriorly. Supphrenic abscesses may also cause respiratory symptoms and breathing difficulty. Abscesses have the history of incidence secondary to penetrating injury or abdominal surgery, with or without gut perforation. Patients with inflammatory bowel disease (e.g., ulcerative colitis and Crohn’s disease) will experience a deterioration in their general condition. Plain abdominal x-ray will show dilated gut loops due to the ileus and elevation of the hemidiaphragm in supphrenic collections. Ultrasonography will show the size and dimension of collection, but an abdominal CT scan may be required to evaluate the exact anatomy of the abscess cavity and its extension. Most localised intraabdominal abscesses may now be treated with ultrasound- or CT-guided aspiration along with broad-spectrum antibiotics. Open external drainage may also be required in patients who fail to improve after percutaneous aspiration or those forming repeated abscesses.

**Cold Abscess**

Cold abscess is the name given to a specialised form of abscess caused by *Mycobacterium tuberculosis*. Unlike acute pyogenic abscess, cold abscess has an insidious onset. It usually affects the neck area but may form abscess in areas such as the spine and psoas muscle. The patient
usually has clinical features of tuberculosis, with anorexia, weight loss, low-grade fever and night sweats, but these may be absent in some cases. In the neck, the cold abscess usually affects the jugulo digastrica lymph nodes, which are initially enlarged. Over a period of months the lymph nodes become fluctuant and show signs of a localised abscess, but acute signs are classically absent. The abscess may burst if left untreated, and a persistent sinus then develops. Usually, infected patients have multiple sinuses that not only cause significant disfigurement but also are a source of infection for other people. Paraspinal cold abscesses are usually due to caries of the spine. The lumbar area is mostly affected. A classic example is psosas abscess, with visible fluctuant swelling in the inguinal region. The diagnosis is often easy due to the classic presentation; however, confirmation is by high erythrocytes sedimentation rate, positive tuberculin test and isolation of gram-positive rod from the pus staining. Fine-needle aspiration cytology will help in confirming the diagnosis by the identification of cseating granuloma and rarely acid-fast bacilli. Lymph node biopsy is confirmatory and is usually taken during drainage of a large cold abscess.

Treatment of tuberculosis abscess depends upon the causative organism. Most cases are due to *Mycobacterium tuberculosis*, and a four-drug therapy is usually curative. Many patients, after good initial response, unfortunately stop the treatment, and these patients are at high risk of developing resistant strains of *Mycobacterium tuberculosis*. Treatment is then by culture and sensitivity of the pus, and treatment may have to be given for a long time. Infections caused by atypical mycobacteria are now on the rise due to the bovine strain of mycobacterium. Treatment is by extensive local excision and use of ethronomycin.

**Post-BCG Tuberculosis Lymphadenitis**

BCG is a tuberculosis vaccine. Post-BCG abscess is the name given to a specialised form of cold abscess seen in babies who have been vaccinated for tuberculosis. There is usually a history of delayed healing of the BCG vaccination site for several weeks or months. This is followed by an enlargement of the regional lymph nodes and then suppuration and abscess formation. Axillary lymph nodes on the affected side are usually involved, but other nodes (e.g., preauricular nodes) have also been affected in some patients. The baby is usually symptom free except for the fluctuant swelling in the axilla. Final-needle aspiration cytology will show green-yellow pus with acid-fast bacilli on microscopy, and cseating granulomas may be visible on histology in intact nodes. Treatment is by aspiration of the nodes followed by single- or two-drug antituberculoly therapy. The response is quick, and 3-6 months of treatment is sufficient for a permanent cure. In large abscesses, drainage or local excision of the affected nodes may be necessary.

**Liver Abscess**

Liver abscess in children is not uncommon in developing countries. Two main forms are seen: pyogenic liver abscess and amoebic liver abscess. In adults, liver abscesses are usually seen as an extension of infections from other viscera, such as appendicitis, ulcerative colitis, hepatobiliary calculi, enteric fever and penetrating injuries. In children, liver abscesses usually occur from hematogenous spread. Many of these children also have underlying immune deficiencies; chronic granulomatous disease in childhood has shown a strong association with pyogenic liver abscesses. Liver abscesses are also seen in children who are on chemotherapy or on immunosuppression for transplant surgery. Rarely, liver abscesses may occur after hepatobiliary surgery, such as biliary atresia and choledochal cyst.

The classic presentation of pyogenic liver abscess is high-grade fever with chills, abdominal pain, tender hepatomegaly, and jaundice. A high leukocyte count is suggestive, but in some patients the leukocyte count may not be very high. Nearly half of the patients will have positive blood cultures. Liver function tests are often marginally deranged. Diagnosis is confirmed by ultrasonography, and a CT scan will help in differentiating this from other cystic lesions. Serological tests for amoebae may be performed to exclude amoebic liver abscess. The treatment of liver abscess is drainage of the abscess, which may be performed either percutaneously under ultrasound or CT guidance. Where facilities for such treatment are not available, open drainage may be required. Complications of percutaneous drainage may be seen in nearly 4% of patients.

*Staphylococcus aureus* is the commonest organism isolated from liver abscesses. Other organisms, such as pseudomonas, *E. coli*, streptococci, and even bacteroides and *Candida*, may be isolated from pus cultures. Multiple organisms are seen in a significant number of patients. Patients with pyogenic liver abscess should be treated with broad-spectrum antibiotics with coverage for gram-positive, gram-negative, and anaerobic organisms. These may later be adjusted according to the sensitivity report, but they usually need long-term treatment. The abscess cavity gradually regresses, and thus ultrasonography may show activity for a long time, but that does not mean the patient has active infections.

**Amoebic Liver Abscess**

Amoebic liver abscess is caused by *Entamoeba histolytica*, which causes colitis with diarrhoea and colicky abdominal pain. The stools are stained with mucus and blood, but some patients may be asymptomatic and act as chronic carriers. Transmission is through the faeco-oral route. The protozoa find their way through the gut mucosa into the portal circulation and into the liver, where they multiply rapidly and cause abscess formation. Due to the necrosis of the liver substance, the pus has an anchovy-chocolate-like appearance. The right lobe is commonly affected, but the disease may involve any lobe of the liver. If the abscess is not treated in time, it may have a tendency to burst into the pleural and peritoneal cavities. The consequences may be disastrous, and the patient may present with severe respiratory symptoms or may go into shock. Children with amoebic liver abscesses look unwell. Due to the prolonged infective process, the patients are anorexic and lose weight, and they have abdominal pain, tender hepatomegaly, and fever Leukocytosis is present, and stool examination may show cysts of *Entamoeba histolytica*. Ultrasonography and CT scan will suggest the diagnosis of liver abscess. Diagnosis of amoebic liver abscess can be confirmed by serological tests such as an indirect haemagglutination test or growth of organisms from pus. Once a diagnosis is made, treatment with either oral or intravenous metronidazole may be started. Many amoebic liver abscesses will resolve with medical treatment only; however, it takes a long time and may need external drainage. The treatment of choice for amoebic liver abscess is ultrasound- or CT-guided aspiration of the abscess along with metronidazole therapy. Some patients may need open drainage of the liver abscess if these measures fail.

**Central Venous Line Infection**

In the paediatric population, central venous access may be required in a variety of situations. With the use of small-calibre lines, such as percutaneously inserted central (PIC) venous lines, the incidence of line sepsis has decreased significantly; however, incidences of line infections in short- and long-term lines may still occur. The signs of line sepsis may not be easy to identify in patients with other sources of infection. Redness over the skin, fluctuating or persistent pyrexia, and generalised sepsis may be indicative of line sepsis. Definitive diagnosis is made by the culture of similar organisms from the peripheral blood, entry site of the line, and blood from the line. Any temporary line should immediately be removed, along with treatment with broad-spectrum antibiotics. In patients who have had long-term lines inserted (e.g., Hickman, Broviac, or portcath lines), salvage of the line may be attempted by giving high-dose broad-spectrum antibiotics through the line. If, however, the symptoms persist, then removal of the line should not be delayed. Serious consequences secondary to line sepsis include bacteria endocarditis, multiple abscesses, and meningitis. If a line is broken or damaged with significant sepsis, it may be replaced over a guide wire under antibiotic cover.
Table 15.1: Evidence-based research.

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<th>Title</th>
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<th>Intervention</th>
<th>Comparison/ control (quality of evidence)</th>
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<td>Comparison of healing of incised abscess wounds with honey and EUSOL dressing</td>
<td>Healing of wounds post abscess drainage</td>
<td>A prospective clinical randomized study. LOCATION: The Isolation Children’s Ward of the Wesley Guild Hospital, Ilesa, an affiliate of the Obafemi Awolowo University, Ile-Ife, Nigeria. SUBJECTS: 32 Nigerian children with 43 pyomyositis abscesses. INTERVENTIONS: All subjects had fresh surgical incisions and drainage of the abscesses and a 21-day course of ampicillin plus cloxacillin (Amoxiclox) and gentamicin; the wounds were left to close spontaneously with twice-daily wound dressing with packing of the abscess cavity with either crude undiluted honey or Edinburgh University solution of lime (EUSOL). OUTCOME MEASURES: The clinical conditions of the wound sites were documented on days 1, 3, 7, and 21 as either clean or dirty, dry or wet, granulation tissue present or absent, and epithelialisation present or absent. The length of hospital stay was also measured. RESULTS: Honey-treated wounds demonstrated quicker healing, and the length of hospital stay was also measured. RESULTS: Honey-treated wounds demonstrated quicker healing, and the length of hospital stay was shorter in patients with honey-treated wounds than those treated with EUSOL (t = 2.45, p = 0.019).</td>
<td>Honey is a superior wound dressing agent compared to EUSOL. Honey is recommended for the dressing of infected wounds, even more so in tropical countries, where it is most readily available.</td>
<td>J Altern Complement Med 2005; 11(3):511–513.</td>
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Table 15.2: Evidence-based research.

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<td>Perianal abscess and fistula in children in Zaria</td>
<td>Perianal abscess and fistula in-ano (FIA) are not uncommon in children, but reports from tropical Africa are uncommon. In a period of 17 years, 17 children aged 12 years and younger were treated for these conditions in Zaria, Nigeria. There were 14 boys and 3 girls, aged 4 months to 12 years (median age, 3 years). Eight had PAA (median age, 3 years), 5 ischiorectal abscess (median age, 5 years) and 4 FIA (median age, 10 months). FIA followed pull through for anorectal malformation in two patients, and in one it was preceded by PAA. FIA was associated with chronic fissure-in-ano in one patient and uncontrolled diabetes mellitus in one. One 16-month-old girl with an ischiorectal abscess developed severe perineal necrotising fascitis and separation and retraction of the anorectum. Escherichia coli was cultured in two patients with abscesses, and Staphylococcus aureus in another two. Culture was sterile in seven patients with abscesses. Treatment was by adequate incision and drainage for abscesses. Fishtulectomy was the treatment for FIA, but in one patient a diversion colostomy was performed in addition, as the fistula was a high one. The child who developed necrotising fascitis had debridement and a diversion colostomy. FIA recurred in one patient, necessitating repeat fistulectomy. Although the number of patients is small, perianal sepsis appears to be less common in the Tropical African environment compared to developed countries. Some differences are highlighted.</td>
<td>Niger Postgrad Med J 2003; 10(2):107–109.</td>
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Key Summary Points

1. Bacterial infections are the cause of significant mortality and morbidity in children.
2. Infections of surgical importance may affect virtually any organ or tissue in the body.
3. Infections may be community or hospital acquired.
4. Bacterial infections are more common at extreme of ages, and thus babies younger than 2 months of age are highly susceptible to acquire bacterial infection.
5. Other conditions such as malnutrition, immune-deficiency states, and prolonged illnesses make the children more susceptible to acquire infections.

Suggested Reading


