Comprehensive Journal of Science
Volume (10), Issue (37), (NOV. 2025)
SICST2025, www.sicst.ly

مجلة العلوم الشاملة
عدد خاص بالمؤتمر الدولي الثالث للعلوم والتقنية
المجلد (10)، العدد (37)، (نوفمبر2025)
ردمد: 3014-6266
ISSN: 3014-6266

Prevalence of Bacterial Meningitis in Ajdabyia City

High Medical Institute of Ajdabyia

Sara Saad Elgeroshi

1.INTRODUCTION

Meningitis is an inflammation of brain membranes and spinal cord. These membranes are collectively known as meninges and these membranes provide protection against external hazards and various microorganisms .This bacterial infection has high mortality rate if untreated because in this infection host defense mechanism of meninges to fight against bacteria becomes week or no more. H influenzae is the main important causative organism, especially in those who are not vaccinated and ranging in age of two months to two years . Meningitis is a serious health problem caused by a variety of pathogenic organisms that vary in age and spread. The infection could spread through the bloodstream or through the ears, nasopharynx, cranial injury, or congenital meningeal malformation . Due to acute meningeal inflammation, bacterial meningitis is associated with considerable morbidity and mortality. More than one million individuals are infected each year around the world, and it is more prevalent in developing nations such as Africa's meningitis belt. This belt runs from Senegal to Ethiopia, and it is here that the illness load is highest. [1]

1.1 AIM OF THE STUDY

1. To isolate and identify bacteria responsible for meningitis .
2. To determine the incidence and etiology of meningitis in children in Ajdabiya city

2. REVIEW OF LITERATURE

Bacterial meningitis is one of the most severe infectious diseases, causing neurologic sequelae and accounting for an estimated 171,000 deaths worldwide yearly. Although most disease occurs in infants, the societal impact is also important because of the continued high incidence in healthy older children and adolescents. Despite many new antibacterial agents, bacterial meningitis fatality rates remain high, with reported rates between 20% and 30%. Furthermore, permanent sequelae, such as epilepsy, mental retardation, or sensorineural deafness are observed in 10%–20% of those who survive

2.1 Bacteriology

2.1.1 Neonatal Meningitis

GBS is a beta-hemolytic, Gram-positive, encapsulated bacterium that commonly colonizes the gastrointestinal and genital tracts . GBS is a commensal organism of the normal vaginal and intestinal microbiome of healthy adults . This bacterium can lead to severe disease in neonates and, occasionally, in postpartum women and individuals with an impaired immune system or underlying medical condition . It is estimated that GBS is present in the vagina, rectal sites, or both of up to 40% of healthy pregnant women, with maternal colonization as the leading risk factor for neonatal exposure and infection . [(1)]

2.1.2 E. Coli: Pre-mature newborns are also at a higher risk of getting bacterial meningitis by E. coli. E. coli is a major cause of neonatal meningitis, being the second most common bacterial cause after Group B Streptococcus. This infection is particularly common in preterm infants and is associated with high mortality and neurological complications, such as ventriculitis and brain abscesses.

2.1.3 Haemophilus influenzae type b (Hib) is a type of bacterium that can cause serious, sometimes fatal, infections like meningitis, pneumonia, and bloodstream infections, especially in children under five, but when the vaccination program of Hib was introduced, there was an extreme reduction in the cases of this meningitis. This now remains limited to the US and is observed in patients who are not vaccinated. However, the Hib in developing countries is still there because most of the people didn't

have access to the vaccine causing a rise in the numbers of the patient suffering from this disease. Only 42% of children globally by 2007 had the facility of Hib immunization program. [2]

2.1.4 Neisseria Meningitidis

Six serogroups of Neisseria meningitides are there that can cause severe and life-threatening meningitis. Neisseria meningitides can cause either an epidemic form of infection or endemic infection. There were around about 170,000 deaths due to this strain in 2010. In industrialized countries, the fatality rate is approximately 5-10%. These strains cause disease at the incidence of 1–3/100,000, which are endemic. Major cases of this disease were caused by Serogroup A and C in China and in the Middle East. Meningitis belt', which is present in Africa, can cause major sporadic epidemics because of the serogroup A that occurs after every 6–12 years, with attack rates of 5000/100,000 inhabitants. [2]

2.2 The disease

typically begins abruptly with symptoms such as headache, neck stiffness (nuchal rigidity), and fever. However, the classic signs of meningitis confusion, headache, fever, and neck stiffness are observed in only about half of infected individuals. The onset is characterized by a sudden episode of severe headache coupled with some meningeal symptoms, including nuchal rigidity and pyrexia. While the hallmark symptoms of meningitis remain confusion, headache, fever, and nuchal rigidity, they are not universally present in all cases. In advanced infections, ecchymotic skin lesions may appear and lead to necrosis. These lesions can vary in presentation and may manifest as dysplastic nevi, transient abnormalities, or may be completely absent in individuals suffering from meningococcal sepsis. This condition can result in extensive tissue death, contributing to coagulopathy, low blood pressure, and Waterhouse–Frederiksen syndrome. Additionally, thrombosis may lead to gangrene in the extremities, while cardiovascular collapse further increases the risk of mortality. [3]

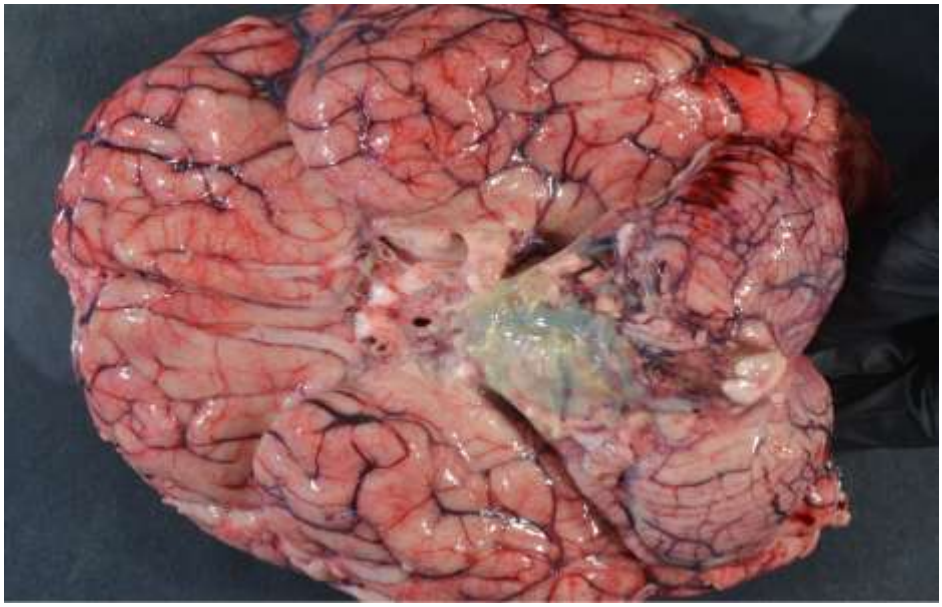


Figure . 1 Brain With Meningitis

2.3 Clinical Features Some can show minute symptoms, and others can have no symptoms at all. Some infants might experience pyrexia, exhaustion, poor feeding, hypothermia, irritability, diarrhoea, seizures, vomiting, respiratory distress, or bulging fontanelles. Some infants may also develop nuchal rigidity at a certain stage. Clinical symptoms in young children are photophobia, fever, nausea, headaches, vomiting, lethargy, irritability & confusion. When the physical examination was done, other indications of bacterial meningitis were discovered, which includes, Kernig's signs and Brudzinski's neurological focal findings. 75% of children may experience irritation in the meninges. The assemblage of bradycardia increased blood pressure, and Cushing's triad is a late sign of increased intracranial pressure. [(4)]

2.4 The major risk factors that are

Age is a significant factor linked to the prevalence of meningitis. This condition is more frequently observed in neonates, children, and adults above the age of 60. Among neonates specifically premature or full-term babies within the first three months of life Group B streptococci, particularly the subtype III strain, have been identified as the leading cause of meningitis. Various studies have highlighted this age-group susceptibility, with meningococcal infections being notably prominent under certain conditions.

2.5 Health Related Risk Factors: Malnutrition In underdeveloped countries, malnutrition is a frequent issue affecting many, especially children. Malnourished children are at a higher risk of infections and increased mortality. Their weakened immune systems struggle to fend off pathogens effectively, making them more vulnerable to conditions such as meningitis.

2.6 Respiratory Tract/Viral Infections

Numerous studies have been conducted on the risk factors associated with meningococcal meningitis, highlighting respiratory tract infections and viral infections, especially influenza, as significant contributors.

2.7 Routes of Infection

Meningitis is a severe infection that develops when bacteria enter the bloodstream, often originating from the ears, sinuses, or throat. Once in the blood, these bacteria can cross the blood-brain barrier, leading to inflammation of the meninges. Meningococcal transmission typically happens through respiratory droplets. [5]

2.8 Pathogenesis

Meningitis typically arises after microorganisms colonizing mucosal surfaces gain access to the bloodstream. In neonates, pathogens are predominantly acquired during birth through contact with and aspiration of maternal intestinal and genital tract secretions, although this is not the sole mode of transmission. Extended stays in nurseries can also expose neonates to various nosocomial pathogens. For infants and children, meningitis commonly occurs when encapsulated bacteria colonizing the nasopharynx are disseminated via the bloodstream. Viral upper respiratory tract infections frequently precede bloodstream invasion, eventually allowing pathogens to breach the blood-brain barrier at vulnerable sites such as the choroid plexus or cerebral capillaries before infiltrating the subarachnoid space. Direct extension from localized infections in areas such as the paranasal sinuses or middle earthrough structures like the mastoid can also result in meningitis. Head trauma involving skull fractures or cerebrospinal fluid (CSF) leakage (rhinorrhoea) may predispose individuals to meningitis, often caused by Streptococcus pneumoniae. Pathogens can enter the CSF through congenital dural abnormalities, including dermal sinus tracts or meningomyeloceles, as well as neurosurgical interventions such as CSF shunts. Penetrating injuries or infections extending from adjacent parameningeal sites also serve as potential pathways for bacterial inoculation. [6]

2.9 Management

The administration of antibiotics is essential. If there is a delay in the administration, e.g., 3 to 6 hours, the chances of mortality will increase. The selection of antibiotics depends upon the type of bacteria identified. If the diagnosis is delayed, treatment with ceftriaxone should be considered. Patients should receive ampicillin who are immunocompromised or older than 50. Patients with bacterial meningitis need to be covered for aerobic gram-negative organisms and Staphylococcus aureus. Patients should receive ceftazidime or cefepime and vancomycin. For HSV coverage Acyclovir should be administered. Decadron may increase survival if it is given for S. pneumoniae infections. Patients having bacterial meningitis should be monitored carefully and should be kept in observation until they have taken 24 hours of antibiotics. People who were in close contact with the patient should also be monitored and treated accordingly. Cephalosporin, including Ciprofloxacin and ceftriaxone can be used. Close contacts mean those people who were at a distance of 3 feet from the patient for more than 8 hours during a week before and after taking antibiotics for 24 hours. [6]

2.10 Prevention

Prevention of meningococcal and pneumococcal disease is based on chemoprophylaxis and vaccination . The advancement of vaccine design in enhancing immunogenicity has been shown to be important in preventing meningitis caused by Nmeningitidis and Spneumoniae. Protein-conjugated capsular polysaccharide vaccines have almost completely eliminated meningitis caused by vaccine serotypes. Meningococcal Capsular Vaccines. Meningococcal polysac-charide vaccines reduce the incidence of infection among mili-tary recruits, reduce the progress of epidemics of serogroup A disease, and protect susceptible complement-factor-deficient individual. Capsule polysaccharide vaccines are available for the pathogenic meningococcal serogroups A, C, Y, and W-135. These vaccines are safe with mild local adverse events and have good efficacy (>85 %) in older children and adults. However, due to lack of a T-helper response, the vaccines are poorly immunogenic below 2 years of age, fail to induce immunological memory, and provide protection for only 3–5 years. Polysaccharide vaccines are used by travelers visiting countries with a high incidence of meningococcal disease . [7]

2.11 Vaccination

It has been seen that meningitis caused by N-meningitides and S. pneumoniae can be prevented through advancement in vaccine design that enhances immunogenicity. Studies show that polysaccharide vaccine against Meningococcal disease decreases the frequency of infection in military recruits. Vaccines such as capsule polysaccharides are accessible only for the 4 meningococcal serogroups in children and adults; such vaccines have been found to be effective with efficacy more than 85% and mild adverse events. However, this vaccine is not effective in children under 2 years of age because of lack of T-helper response. Polysaccharide vaccine can provide immunity up to 3–5 years. There is an advancement of vaccine development against meningitis disease characterized by the development of meningococcal polysaccharide and protein conjugates that has been introduced in several parts of the world, such as USA, Canada, UK, and several other parts of Europe. These vaccines are found to be safe and cause immunogenicity in children and induce immunological memory. Recently, second-generation conjugated vaccines have been launched such as PCV10 and PCV13. Vaccine development against serogroup B is still challenge because the capsule of serogroup B has structure similar to polysialic structures. Developed outer membrane vesicle (OMV) vaccines have efficacy to almost 50–80 % but it is not efficient in children and these vaccines work against specific strain.[8]

2.12 Diagnosis of Pleocytosis and Meningococcal

Meningitis Meningococcal meningitis can be identified using pleocytosis and Gram staining, with or without cerebrospinal fluid (CSF) culture, as well as blood or skin lesion analysis. Gram staining combined with any type of culture remains a primary method for diagnosis. Early diagnosis of meningococcemia is challenging in the absence of rashes or meningeal symptoms. In severe cases of meningococcemia, patients often exhibit general signs of sepsis-like leg aches . However, meningococcal and pneumococcal infections do not typically induce these symptoms. Parents and relatives should monitor feverish children or adults for rashes, and healthcare providers must take concerns about rapid or abrupt deterioration seriously. Laboratory Diagnosis of Meningitis Accurate identification of all types of meningitis necessitates the evaluation of CSF. Specimens such as skin biopsies, blood samples, CSF aspirates, and nasopharyngeal swabs are used for detection when clinically indicated.

Since pneumococci and meningococci are sensitive to desiccation and extreme temperatures, rapid culturing after sample collection is crucial. Preliminary diagnosis

often utilizes Gram staining and acridine orange staining. For hazy CSF samples, Gram- and acridine orange-stained smears are prepared directly from the CSF or after centrifugation if the fluid appears clear. Gram-negative diplococci and polymorphonuclear cells are observable in smears when the bacterial count in CSF exceeds 105/milliliter. However, approximately 25% of samples fail to stain positive when bacterial density is below 103/milliliters. Between 60–90% of surveyed CSF samples that stain positive also yield positive culture results. Further identification methods include Gram stain smears combined with culture from petechial skin lesions related to the disease, which detect meningococci in 62% of patients . Latex agglutination and conglutination tests can identify meningococcal capsular polysaccharides directly in CSF . These techniques, which are highly sensitive, also enable the direct identification of meningococci in blood or CSF using nucleic acid amplification tests or polymerase chain reaction (PCR) techniques . [9]

These advanced approaches are particularly useful for confirming diagnoses in patients who have already received antibiotic treatment. Gram-positive stains were observed in 90% of children diagnosed with pneumococcal meningitis. Within this group, nearly 80% of patients had meningococcal meningitis, which is predominantly seen in children. Around half of the patients were identified with Gram-negative bacillary meningitis, while approximately one-third were affected by Listeria meningitis . The proportion of favorable staining increases significantly when cerebrospinal fluid is processed through Cytospin centrifugation . Differential diagnosis for various types of meningitis is often supported by analyzing cerebrospinal fluid cell counts and differentials, as well as glucose and protein concentrations. [10]

3.Methods

This study aimed to assess diagnostic methods and treatment approaches for children between 1 month and 5 years of age who were discharged with a diagnosis of bacterial meningitis. The condition was defined based on the attending physician's clinical assessment at discharge, which included laboratory-confirmed cases and probable ones. Probable cases were characterized by compatible clinical signs and symptoms, alongside cerebrospinal fluid (CSF) findings such as turbid appearance and/or leukocytosis (>100 cells/mm^3). Laboratory confirmation relied on identifying bacteria either directly through culture from CSF or blood, or indirectly via antigen detection from CSF. Data extracted from clinical records included demographic details (age, gender), the timeline of illness (onset of first symptoms, hospitalization, and discharge dates), diagnostic procedures (lumbar puncture at admission, CSF analysis, Gram stain, cultures, latex agglutination tests), and treatment specifics. Patients reported with different disease showed clinical signs (such as vomiting, neck rigidity, bulging fontanelle, reduced consciousness, sudden onset of fever, headache, convulsions and irritability) of meningitis. At the hospital, suspected cases of acute bacterial meningitis were diagnosis by a pediatrician using specific criteria signs of meningitis, such as fever (axillary temperature over 38°C), vomiting (more than three episodes within 24 hours), headache, or indications of meningeal irritation (bulging fontanelle, Kernig or Brudzinski signs, or neck stiffness) Treatment details covered both pre-admission antibiotic use and in-hospital interventions, with emphasis on timing—the interval between the suspected meningitis diagnosis and administration of the initial in-hospital antibiotic dose as well as the agents prescribed. Information on dexamethasone therapy was recorded regarding its administration, timing, and duration. Additional areas of assessment included imaging techniques such as cranial computed tomography (CT), magnetic resonance imaging (MRI), and transfontanellar ultrasound; auditory evaluations; complications encountered during hospitalization; neurological sequelae; and overall condition upon discharge

3.1 CSF Cultures and Bacterial Isolates

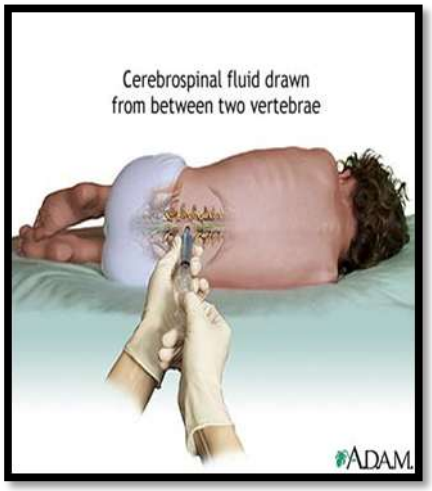
A sterile spinal needle, either 25 or 27 gauge, was utilized to extract cerebrospinal fluid (CSF) from the subarachnoid space located between the fourth and fifth lumbar vertebrae. The CSF was collected into two sterile screw-capped tubes, each containing two milliliters of fluid.

The first tube was designated for microbiological analyses such as direct Gram staining, standard bacteriological culture, and antibiotic susceptibility testing. The second tube

was allocated for physical assessments (e.g., color and appearance), chemical analyses (e.g., glucose concentration and protein levels), as well as cytological examination.

3.2 Colour and aspect, Cytological examination: Leukocytes were rated as negative (less than 10), 1+ (10–50), 2+ (51–290), and 3+ (more than 291 cells/uL). Microbiological examination: Microbiological standards were followed while preparing direct smears stained with Gram. Gram-stained smears were examined under the microscope, CSF's Culture: Blood, chocolate, and Mac-agar Conkey's plates were inoculated, and the plates were then incubated aerobically and anaerobically at 37°C for 72 hours in the presence of 5-10 percent CO2. Bacterial colonies are identified by colony shape and biochemical responses.

3.3 The sensitivity of the recovered clinical isolates to antimicrobial drugs was determined using the disc diffusion method. The sizes of inhibition zones were measured in millimeters with a ruler, and the agents examined were classed as susceptible, moderate, or resistant. In individuals with neurological deficits, seizure, or a brain MRI conducted prior to the lumber puncture . Patients were monitored on a daily basis to see if their symptoms improved or if they developed new ones. For the first 48 hours, vital signs were checked every four hours, then as needed after that. Follow-up on a daily basis using a neurosign chart that comprised the following variables: During the inpatient treatment, tests for (Glasgow coma scale (GCS), seizure, headache, and nuchal rigidity) were performed. At discharge, patients were evaluated for gross neurologic abnormalities (visual issues, hearing loss, and bodily weakness) as well as a mini-mental state test. The epidemiologic characteristics, clinical data and laboratory findings, causal organisms.



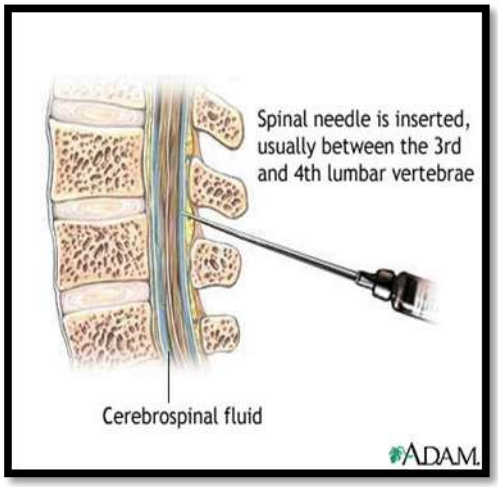


Figure3.1 CSF Sample collection/ Lumber puncture

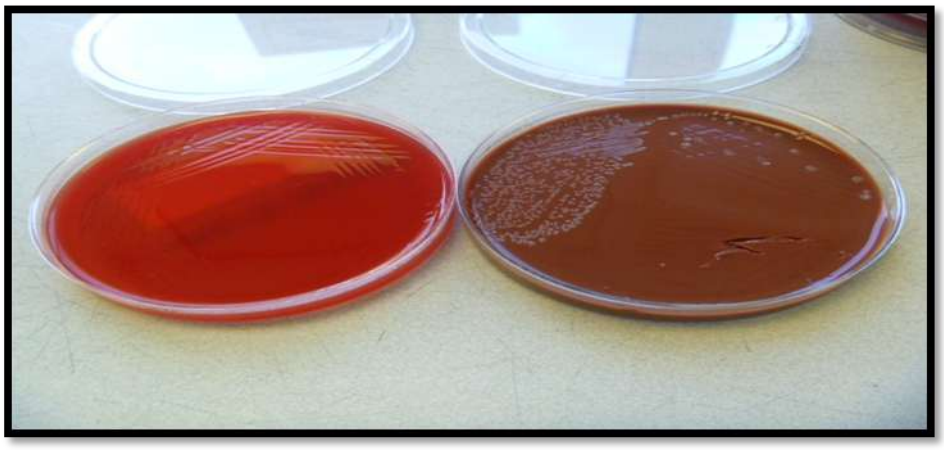


Figure 3.2 Chocolate agar is an excellent Haemophilus growth medium

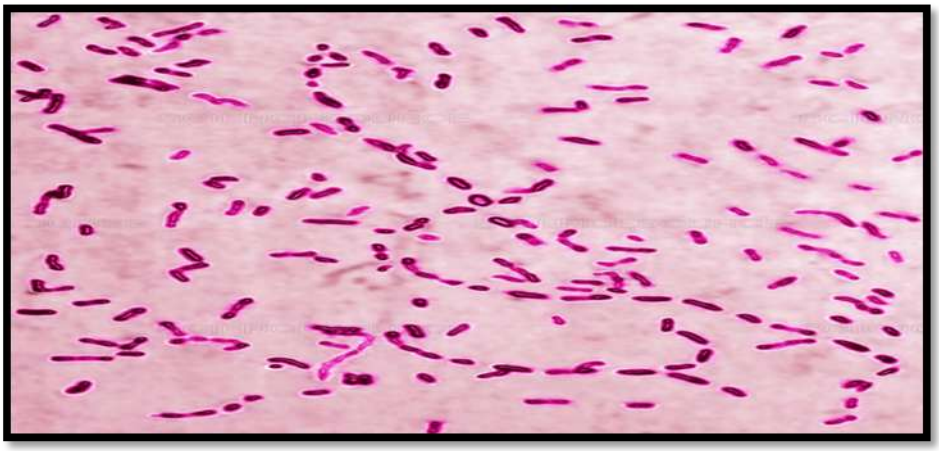


Figure3.3 Haemophilus bacteria are typically small coccobacilli, they are categorized as pleomorphi

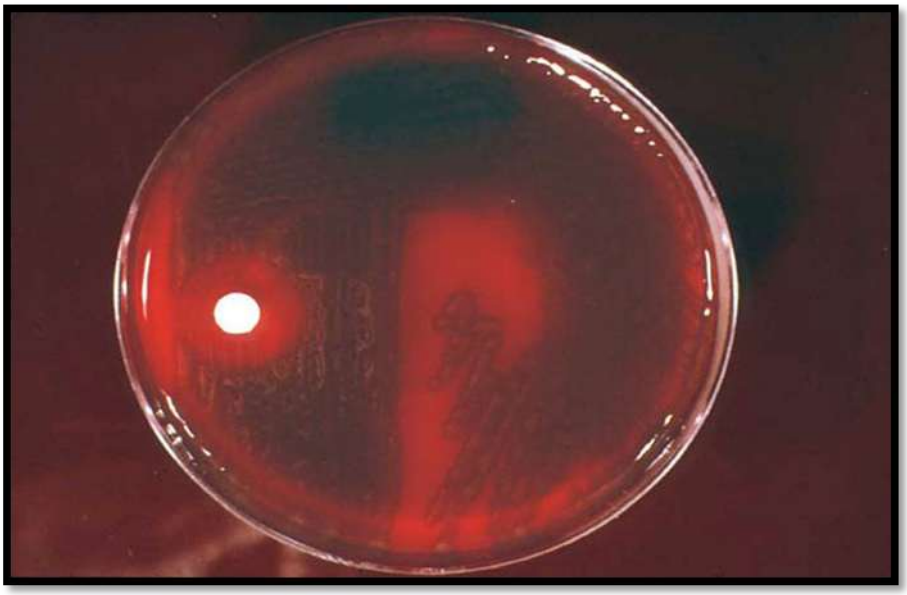


Figure 3.4 *S. pneumonia* on blood Agar sensitive to Optochin antibiotic

Figure3.5 Results of antimicrobial susceptibility patterns of different antibiotics

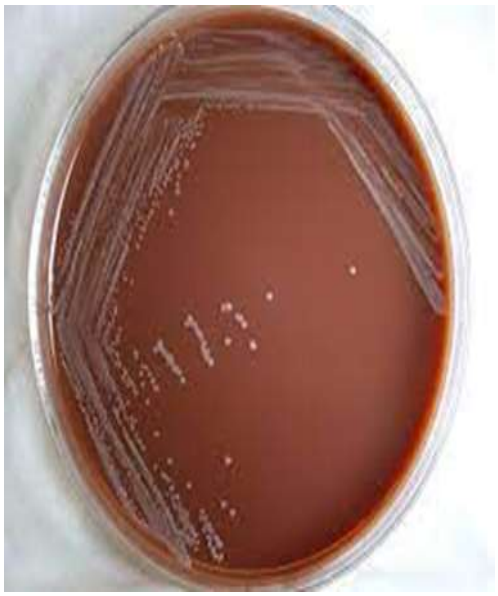


Figure 3.6 Neisseria meningitides.

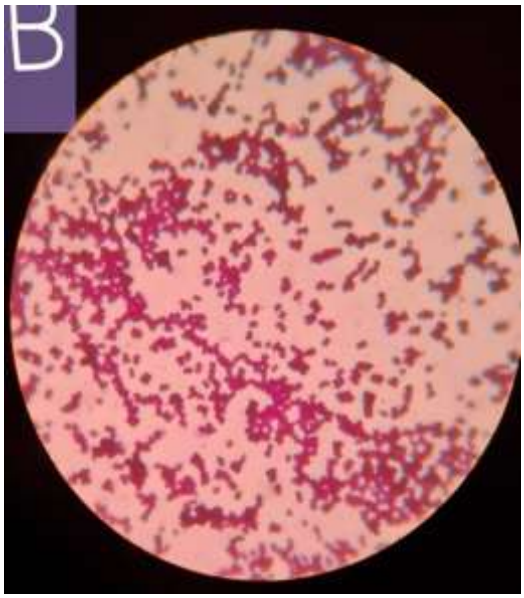


Figure 3.7 Gram-negative cocci.

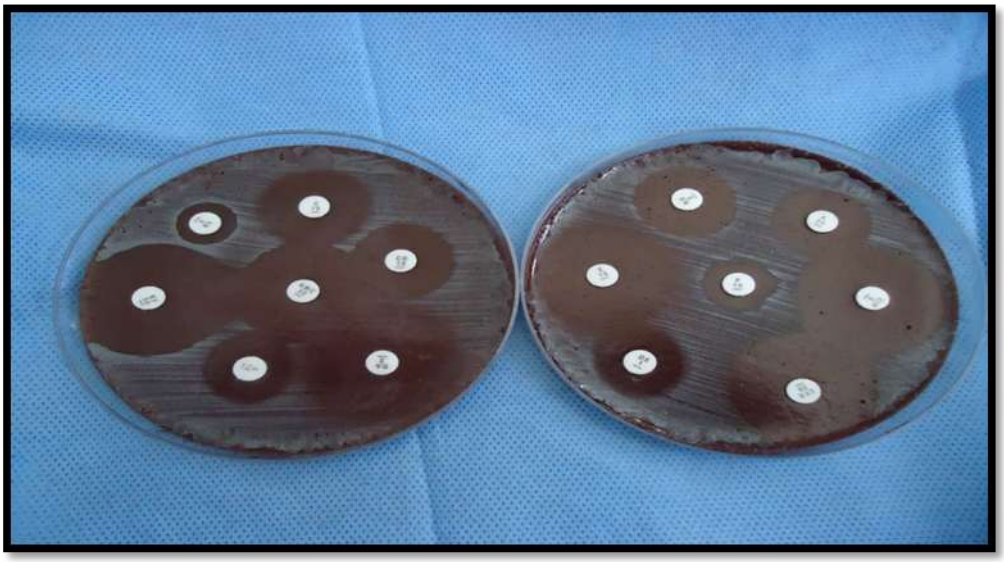


Figure3.8 Results of antimicrobial susceptibility patterns of different antibiotics antibiotics against Neisseria meningitis

4. RESULTES

A total 100 children were admission in hospital with a diagnosis of meningitis, This stud y was Conducted an children admins in pediatric department in Elmegriaf hospital in Ajdabyia city from March 2024 August 2025 . The majority of cases were within the 1-2 year old age group (35%). While the smallest age group is 5-6 years old (20%) old. For all races, males were more commonly infected (60%) than females(Fig 4.1). The commonest presenting complaints necessitating admission to the hospital (Tab 4.1) were fever (80%), consciousness (30%), seizures (23%), and loss of cranial nerve (10%). Cerebrospinal fluid (CSF) cultures were positive for Neisseria meningitidis in (45%), S. pneumonia(30% and Haemophilus influenzae in (25%)(Fig 4.3).

Types of antibiotics prescribed for the treatment of meningitis

All three major causative organisms were 100% sensitive to ceftriaxone. S. pneumoniae was 100% sensitive to penicillin and H. influenzae and N. meningitidis was 90% sensitive to penicillin and ampicillin . Third-generation cephalosporins (ceftriaxone or cefotaxime) were the only antimicrobial treatment administered to the majority of patients meningitis

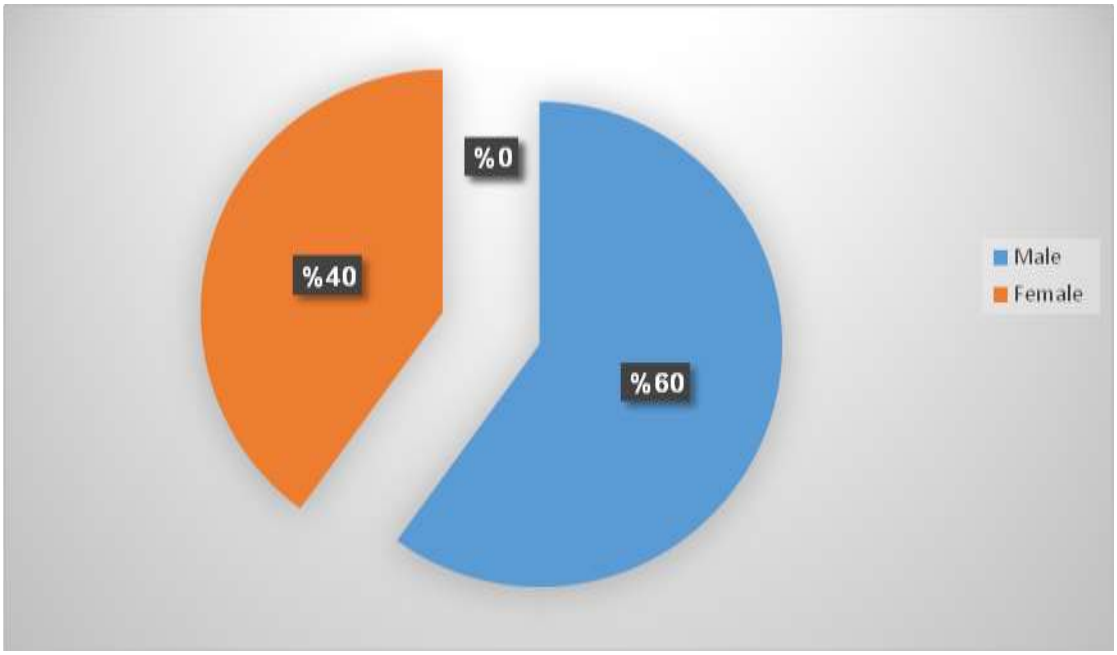


Figure 4.1 Distribution of patient's according to gender

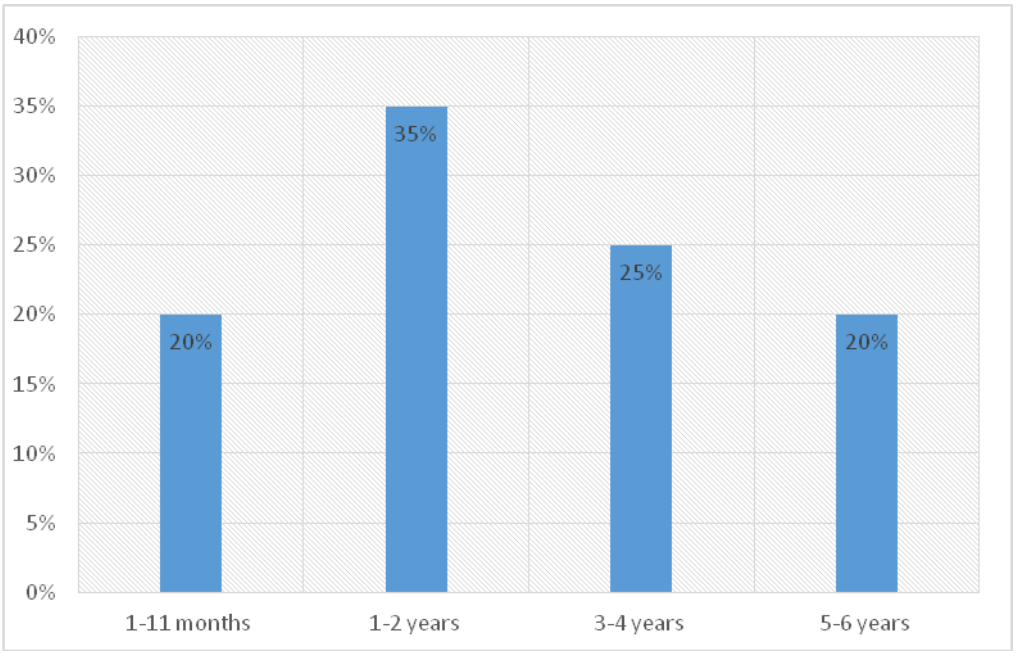


Figure 4.2. Distribution of bacteria causing childhood acute bacterial meningitis in different age groups

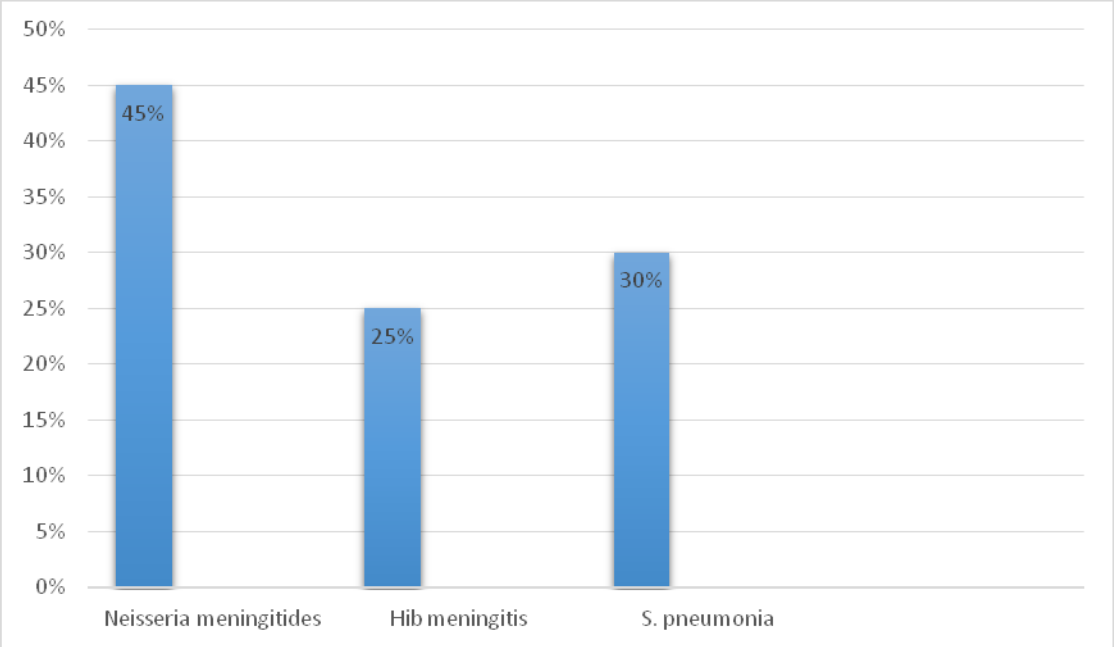


Figure 4.3. Distribution of etiology of acute bacterial meningitis

Table 4.1: Lethality related to neurological signs in 100 cases pyogenic meningitis

Neurological Alteration	Presence	Absence
Seizures	30(30%)	70(70%)
Irritability	40(40%)	60(60%)
Diminished consciousness	60(60%)	40(40%)
meningeal signs	80(80%)	20(20%)
fontanel bulging	20(20%)	80(80%)
cranial nerve	10(10%)	-

Table 4.2 : The relationship between the incidence rate of neurological signs according to age groupsin 100 children

Neurological Alteration	0-11month	1-2yr	3-4yr	5-6yr	Total
Seizures	15%	5%	10%	-	30%
Irritability	10%	15%	5%	10%	40%
Diminished consciousness	20%	15%	15%	10%	60%
meningeal signs	40%	20%	15%	5%	80%
fontanel bulging	10%	8%	2%	-	20%
cranial nerve	5%	5%	-	-	10%

5.DISCUSSION

Determination of the etiology of bacterial meningitis and estimating cost of disease are important in guiding vaccination policies. To determine the incidence and etiology of meningitis in Ajdabyia city cerebrospinal fluid (CSF) samples were obtained prospectively from children (1 month–5 years of age) with a clinical diagnosis Children less than five years old were the most prevalent group in this study. . The majority of cases were within the 1-2 year old age group (35%). This is also true for most other studies done on the prevalence of bacterial meningitis in Malaysia (Urwin et al, 2010). Factors such as poverty, overcrowding and the lack of use of health care may contribute to the increased incidence (Bedford et al, 2001). The most common presenting complaints on admission were fever were fever (80%), consciousness (30%), seizures (23%) McIntyre PB et al also reported similar findings in a study done in (2012) on hospitalized children in Kelantan in Malaysia. [(2)] The three major organisms responsible for bacterial meningitis in this study were *N. meningitidis* (45%). *S. pneumoniae* (30%),

H. influenzae (25%) . Grandgirard D et al (2016) reported H. influenzae as the most common spp (50%) followed by *S. pneumoniae* (22%) and *N. meningitidis* (45%). *S. pneumoniae, H. influenzae* were responsible for 47% of cases. [6] Risks of complications and sequelae do vary by causative agent, being higher for meningitis due to N. meningitidis than for other causative agents **[1-3]** . As expected on the basis of national vaccination coverage rates and epidemiological data, most of our cases were due to *N. meningitidis* and *S. pneumoniae*. *S. pneumonia* cases were less frequent but more severe than other meningitis, with a 17.6% reported rate of neurological sequelae at discharge. The proportion of pneumococcal meningitis sequelae is consistent with results from meta-analysis, reporting a median of 24% .Current literature reports that complications and sequelae are less common in meningococcal meningitis , and our results confirm this finding. *N. meningitidis* caused however the majority of our cases, as well as most of meningitis cases reported at the national level . The availability of conjugate meningococcal B vaccine, licensed in the European Union in January 2013 but recommended in Libya since 2018 only in a minority of Regions and not at a national level, can guarantee high degree of protection against meningococcal meningitis . Accurate laboratory identification of the cause of acute bacterial meningitis is crucial for ensuring effective patient treatment, managing contacts appropriately, and implementing informed public health measures.

Additionally, it serves as a foundation for decisions related to immunization strategies, particularly in countries without routine vaccination against the primary pathogens responsible for acute bacterial meningitis. While bacterial culture remains the gold standard for diagnosis, its sensitivity can be significantly reduced by prior use of antimicrobial drugs, highlighting the need for alternative diagnostic methods that do not rely on culture. [4.5] Selecting the appropriate antibiotic is a key consideration in managing acute bacterial meningitis. For suspected cases in infants under 1-3 months, the first-line recommended treatment includes cefotaxime combined with amoxicillin or ampicillin. In infants and children aged 1-3 months or older, ceftriaxone is typically preferred. In regions where antimicrobial-resistant isolates are prevalent or in cases involving children with prolonged prior exposure to antibiotics, vancomycin combined with a third-generation cephalosporin is suggested as an empirical treatment approach. This study highlights the importance of maintaining high-quality and consistent surveillance of bacterial meningitis cases, along with a thorough understanding of the

etiology and epidemiology of the responsible bacteria. Accurate tracking of meningococcal trends is essential for devising effective vaccination strategies. The epidemiological dynamics of bacterial meningitis are ever changing, with causative agents varying over time and across different geographical areas. [9.10]

6. CONCLUSION & RECOMMENDATIONS

Acute bacterial meningitis continues to pose a significant burden in our setting, with Neisseria meningitidis , Streptococcus pneumoniae, and Haemophilus influenzae identified as the primary causative agents. Mortality rates and acute complications remain alarmingly high, particularly in cases associated with poor prognostic factors such as age below 12 months, delayed medical intervention, pneumococcal meningitis, coma, brain abscesses, and intracranial hypertension. Prioritizing efforts to enhance routine immunization programs targeting vaccine-preventable diseases in infants and children specifically those caused by Haemophilus influenzae, pneumococcus, and meningococcus is critical to reducing the impact of this condition

1.The use of vaccines to prevent the disease is the most effective measure to prevent its spread. Currently, there are many types of vaccines available to prevent meningococcal meningitis, including vaccines against common bacteria **.**

2.Maintaining personal and environmental hygiene, as well as avoiding contact with infected individuals, also plays a crucial role in preventing the spread of bacteria or viruses causing the disease.

3.Residences, schools, and classrooms should be well-ventilated and well-lit. In areas with outbreaks, monitoring should be increased to detect early cases of fever and sore throat to serve monitoring purposes. If conditions permit, throat swabs of patients and nearby people should be taken for testing for meningococcal bacteria.

4.Give the proper antibiotics with the proper way .

5. Collected samples should be send to the laboratory soon as possible .

7.References

1. Ministério da Saúde. Guia de Vigilância em Saúde. Brazil: Ministry of Health; 2016. Portuguese.

2. McIntyre PB, O'Brien KL, Greenwood B, van de Beek D. Effect of vaccines on bacterial meningitis worldwide. Lancet. 2012;380(9854):1703-11.

3. Namani S, Milenkovic Z, Koci B. A prospective study of risk factors for neurological complications in childhood bacterial meningitis. J Pediatr (Rio J). 2019;89(3):256-62.

4. Saez-Llorens X, McCracken GH Jr. Bacterial meningitis in children. Lancet. 2020;361:2139–48.

5. Baraff LJ, Lee SI, Schriger DL. Outcomes of bacterial meningitis in children: a meta-analysis. Pediatr Infect Dis J. 1993;12:389–94.

6. Grandgirard D, Leib SL. Strategies to prevent neuronal damage in pediatric bacterial meningitis. Curr Opin Pediatr. 2016;18:112–8.

7. Horizonte B. BEM (BOLETIM EPIDEMIOLÓGICO MINEIRO) Análise Epidemiológica das Doenças e Agravos Transmissíveis PANORAMA DO ANO DE 2015. Brazil: Minas Gerais; 2019. Portuguese.
8. de Jonge RC, van Furth AM, Wassenaar M, Gemke RJ, Terwee CB. Predicting sequelae and death after bacterial meningitis in childhood: a systematic review of prognostic studies. BMC Infect Dis. 2010;10:232. /10.1186/1471-2334-10-232.
9. Olson D, Lamb MM, Gaensbauer JT, Todd JK, Halsey NA, Asturias EJ, et al. Risk Factors for Death and Major Morbidity in Guatemalan Children with Acute Bacterial Meningitis. Pediatr Infect Dis J. 2015;34(7):724-8.
10.Vasilopoulou VA, Karanika M, Theodoridou K, Katsioulis AT, Theodoridou MN, Hadjichristodoulou CS. Prognostic factors related to sequelae in childhood bacterial meningitis: data from a Greek meningitis registry. BMC Infect Dis. 2016;11:214.