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Bacterial meningitis: epidemiology, pathology, diagnosis and treatment

A Review Article in
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Abstract

Bacterial meningitis is a medical contingency that needs to be diagnosed and treated as soon as possible. Meningitis is caused by two pathogens: *Streptococcus pneumoniae* and *Neisseria meningitidis*. Antibiotic tolerance is on the rise, and it will be a major issue in the near future. Clinical and laboratory research has contributed to a better understanding of the pathways that cause brain harm, its consequences, and neuropsychological losses. The current pathophysiological understanding of acute bacterial meningitis is reviewed, as well as current treatment options.

Keywords*: bacterial meningitis,. pneumococci,. Meningoencephalitis,. meningococci*

1. Introduction

Bacterial meningitis (brain inflammation) is still an unsolved challenge within clinical medicine although new antibiotics and enhanced critical care. Despite the fact that very efficient antibiotics destroy bacteria effectively, a mortality ratio of up to 34 percent still exists. Long-term consequences affect up to half of the legacy.(Van de Beek et al., 2006) Meningococci or pneumococci, which are the most prevalent reason for society-acquired bacterial meningitis, are the subject of this study. Dexamethasone such as auxiliary therapy of antibiotics was proposed in two seminal trials as a way to increase the result of acute Bacterial meningitis via reducing inflammation.(Weber and Tuomanen, 2007) Several trials conducted in low wealth regions found little positive impact, in comparison to the health evidence obtained in the United States or Western Europe. Other underlying illnesses, especially AIDS, may be contributing to this disparity.(Molyneux et al., 2002) as well as the reality these patients in these trials appeared to contingency departments at later phases of the illness. (Scarborough and Thwaites, 2008) As a result, in addition to the science and medical problems of trying to unravel the molecular foundation of bacterial meningitis, discovering novel therapies & opposite new approaching challenges such as rising pathogen tolerance to commonly used antibiotics (for eg, pneumococci up to 35 percent drug resistance), the widening divide between richer populations and country capital poses an equally significant problem. (Weber and Tuomanen, 2007) It is critical to note that the ratio of resistant isolates is highly influenced by regional and also other factors.

Bacterial meningitis(BM) is an inflammation to the meninges, especially its spidery or pia mater, caused by bacteria invading this subarachnoid space; which has been understood for over a century.(Weber and Tuomanen, 2007) Pathogens multiply or cause inflammation inside the CNS by exploiting various features of an IMMUNE SYSTEM(antibodies) (Simberkoff et al., 1980a) The induction of highly active leukocytes into Cerebrospinal fluid is a defining feature of bacterial meningitis(BM). Inflammation of the meninges can be caused by microbes, influenza, fungi, and noninfectious factors like systemic or neoplastic illness, as well as some medications.

Inflammation generally involves the brain parenchyma as well as the meninges that accompany it [meningoencephalitis].(Swartz et al., 1984) It starts in the ventricles (cardiac chambers) and extends down the spine.(Kastenbauer et al., 2001) Neuronal injury, especially in hippocampal systems, has been reported as a possible cause of chronic neuropsychological losses in legacy in recent years. Bacterial meningitis (BM) is a medical emergency(contingency) that requires prompt diagnosis and care. The sub-Saharan meningitis belt has the highest prevalence, with cyclic epidemics occurring at most once each decade. (Nau et al., 1999)

2. Bacterial meningitis (BM)

Bacterial meningitis is an inflammation (swelling) to the meninges, especially the arachnoid mater, pia mater caused by bacteria invading a subarachnoid space, according to concepts that have been understood for over a century.(Flexner, 1907) Pathogens multiply and cause inflammation within the Central nervous system(CNS) by exploiting different features of the immune system.(Simberkoff et al., 1980b) The induction of highly active leukocytes into Cerebrospinal fluid is a common symptom of bacterial meningitis(BM). Inflammation of the meninges may be caused by (bacteria, influenza, fungi, and non-infectious factors) such as somatic and neoplastic(tumor) disease, as well as some medications. An inflammatory mechanism generally involves the brain parenchyma [meningoencephalitis], the ventricles(cardiac chambers), as well as the spinal cord.(Kastenbauer et al., 2001) Neuronal injury, especially in hippocampal systems, has recently been recognized as a possible cause of survivors' chronic neuropsychological problems. Bacterial meningitis is really a medical emergency that necessitates prompt diagnosis and therapy.(Nau et al., 1999)

3. Epidemiology

The epidemiology of bacterial meningitis also significantly improved over the past two decades. *Haemophilus influenza,* that significant reason for meningitis in the past, is no longer a problem in developing countries, and it acts as an excellent indicator of an effective vaccine program. In both the United States and Europe, pneumococci have become the common produce of (BM)bacterial meningitis in children & adults. Mostly in the United State, the disorder has a prevalence that ranges between 1.1 to 2.(Schuchat et al., 1997) as well as in Western Europe (Berg et al., 1996) In Africa, rise to 12 out of every 100,000 people die each year.(O'DEMPSEY et al., 1996) Individuals under the age of Five and those above the age of 60 have the greatest risk of illness. A previous splenectomy, malnutrition, and sickle - cell disease disorder are also recognized predisposing factors.(Kastenbauer and Pfister, 2003) In certain areas that promote this strategy, the use of such conjugate pneumococcal vaccinations has resulted in a substantial reduction in invasive pneumococcal illness, like meningitis. (Whitney et al., 2003) Pneumococci tolerant to β –lactam antibiotics are becoming more common, which is becoming

a growing concern. Pneumococci persisting in the (CSF) for an extended period of time may cause increased death and severe brain damage in survivors. (McCullers et al., 2000) These live bacteria results compel one to investigate the consequences of bacterial toxins and emerging cell membrane and surface constituents of neuronal damage to a greater depth. *Neisseria meningitides* had replaced *Haemophilus* while one of the much common causes of meningitis in developed countries, but it remains a significant health concern in the United States and Europe. Meningococci may also ultimately cause disease, such as acute gram-negative blood poisoning (sepsis)and disseminated intraarterial coagulopathy, in addition to traditional meningitis. According to the World Health Organization, at least 500 000 new symptomatic diseases occur each year, with approximately 50 000 fatalities.(Stephens, 2007) A sub-Saharan meningitis area has the highest maximum prevalence, with cyclic epidemics occurring at least once every decade.

4. Pathogenesis of Bacteria meningitis

4.1. Bacterial attack (invasion)

The latest theory would be that great groups of bacteremia occurs before meningitis and also that bacteria enter the (CNS) through the bloodstream. Direct entrance to the central nervous system via Dural deficiencies or regional infections is another choice. Such defects should be detected in the clinical surroundings using Computerized tomography (CT) scans or magnetic resonance images (MRI), but the structural location of bacterial invasion from the bloodstream is unidentified. The choroid plexus can be a location of invasion, according to the survey.(Weber and Tuomanen, 2007) Meningococci can be present in both the choroid web(plexus) and the meninges.(Pron et al., 1997) These findings indicate that some highly vascularized places may be possible entry points for pneumococci, which invade the leptomeningeal blood vessels of meningitis. Meningeal infections should bear powerful molecular instruments in order to penetrate a blood or blood-brain Cerebrospinal fluid barrier, as well as to overcome complex mechanisms such as adherens junctions.(Polfliet et al., 2001) Streptococcal proteins including Certified Business Process Associate interact with leukocyte activating factor or glycoconjugate receiver of phosphorylcholine onto eukaryotic cells (real nucleus cell)to facilitate the endocytosis process and blood-brain boundary passing.(Radin et al., 2005) PilC1 attachment from meningococci reacts to CD46 and binds to vitronectin as well as integrins upon on external membrane.(Unkmeir et al., 2002) Bacteria that cause meningitis in infants, especially group B *streptococcal* & *Escherichia coli*, are also well prepared with attaching proteins that enable them to attack the Central nervous system. A thorough understanding as to how bacteria (activate & invade)cells can allow scientists to inhibit these contacts and thus avoid disease progression.(Maisey et al., 2007)

4.2. Inflammatory reactivity (response)

Activation of Inflammatory from endothelial cells appears to be a requirement to bacterial invasion, and it also regulates adhesion molecules like(ICAM-1). (Freyer et al., 1999) As a result, these molecules help to facilitate a several step leukocyte invasion mechanisms. Meningitis is diagnosed by the presence of leukocytes, especially granulocytes, with in Cerebrospinal fluid. Early inflammatory responses of bacterial invasion appear to occur in lockstep, as do Matrix metalloproteinases(MMPs), which are produced by activated leukocytes.(Kieseier et al., 1999) Others lead to the early deterioration of the blood-brain barrier as well as the blood- Cerebrospinal fluid barrier. Bacteria duplicate, induce autolysis known as(self-digestion), and induce more inflammation once a reach the subarachnoid area. Many cell kinds appear to be include and as referred endothelial cells, perivascular macrophages as well as labrocyte known(mast cells)could play a crucial function.(Polfliet et al., 2001) High - temperature bacteria and(pamps) pathogen-associated molecular pattern to meningitis infections such as (lipopolysaccharide , lipoteichoic acid , peptidoglycan, and lipoprotein)are produce meningitis differentiated from live bacteria, of immune sequence identification molecules such as CD14 or LBP serve as receptors in recognizing PAMPs. (Beutler et al., 2003) TLR2 inhibitor recognizes pneumococcal (LP- PG), while TLR4 recognizes LPS and, oddly, the pneumococcal poison pneumolysin. TLR alert(signal) are transmitted downward by the endo cellular matcher Myeloid differentiation primary response protein to a variety of inflammatory mark waterfalls, comprising nuclear factor (NFkB) and mitogen-activate protien(MAP) kinases, resulting due to fast inflammatory reaction in meningitis.(Lehnardt et al., 2006)

4.3. Neuronal Harm

Neuropsychological decrease affect up to half of (BM) survivors. (de Beek et al., 2002) The hippocampus appears to be the most much unstable region inside the brain, with synaptic dysfunction resulting in hippocampal necrosis, as shown on MAGNETIC RESONANCE IMAGING scans of survivors of(BM) bacterial meningitis. (Bigler et al., 1996)

An extracellular- fluid surrounding brain cells is adjacent with the Cerebrospinal fluid, or the similarity to a ventricular system enables spread between both compartments, which could distribute dissolved bacterial with inflammatory (inflammatio)toxic intermediaries.(Rennels et al., 1985) Neuronal injury in meningitis was specifically multifactorial, containing bacterial toxins, antibody competent cell cytotoxic compounds, and indirect pathology as a result of intracranial disorders (figer1). Hydrogen peroxide(H2O2) and pneumolysin, a pore- making cytolysin have also been recognized as main poisons in *S. pneumoniae* its pathogen linked with high prevalence of neuronal harm. difference between to wild kind bacteria, synaptic injury was decreased by 50percent in laboratory meningitis caused by carcinogen

(pneumococcal mutants). (Braun et al., 2002) The demonstration of direct bacterial toxicity emphasizes the vital value of eradicating live bacteria and their metabolism quickly with antibiotics. Toxic action can be greatly extended in insufficiently processed patients or immune bacteria, causing neuronal damage. By causing fast mitochondrial destruction, these toxins appear to induce programmed mortality of (neurons or microglia).

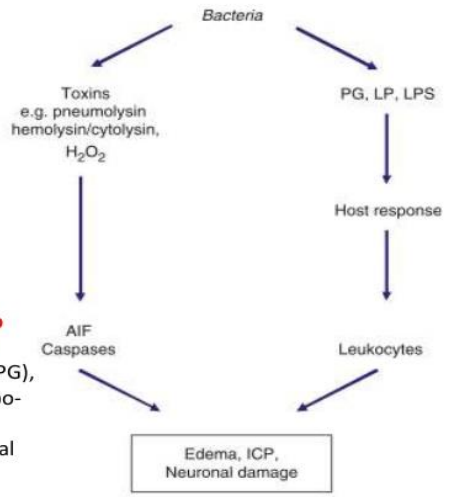

Figure 1. Two major routes involving **bacterial toxins** and **cytotoxic products of the inflammatory response lead to intracranial complications and brain damage**. Peptidoglycan (PG), bacterial lipopeptide (LP), lipopolysaccharide (LPS), apoptosis inducing factor (AIF), intracranial pressure (ICP).

Pneumolysin in specific,was discovered to transport nutrients to mitochondria and cause the forming of pores in mitochondrial films.(Kruft et al., 2001) Destroyed mitochondria trigger(apoptosis inducing factor) which causes DNA injury moreover apoptosis-like cell killed on a big scale. Caspases are not needed for this form of cell death. As a result, caspase blockers may not be able to save cells exposed to active pneumococci or pneumolysin in laboratory.(Bermpohl et al., 2005) Intrathecal treatment of the (broad-spectrum) caspase inhibitor z(carbobenzoxy-valyl-alanyl-aspartyl-[O-methyl]- fluoro methyl ketone), on the other hand, stops about half of the neuronal injury in clinical meningitis in vivo. Additional research in caspase-3 mice lacking showed that caspase action is needed for delayed neuronal damage and not for early neuronal damage.(Du et al., 2004) These results suggest that soon(caspase is independent)cell death could be triggered through bacterial toxins although late(caspase is mediated apoptosis), is primarily caused through the host resistant response(antibody). In the laboratory, the results in vivo could be established as well as the toleration of various types and on enough of cell death has been established.(Bermpohl et al., 2005) Antibiotic therapy leads to an uptick in bacterial waste, particularly bacterial DNA with highly potent immune signals such as PG and LPS. Much like LPS amounts in parts of the body are related to the incidence of meningococcal infection, the accumulation of PG in the Cerebrospinal fluid is correlated to the treatment diagnosis of (pneumococcal meningitis).(Schneider et al., 1999) Although these this bacterial cell membrane constituents are extremely powerful inflammatory receptors, they had a little clear toxic impact with

cultivated neurons. This tolerance to Toll-like Receptor ligands could be exacerbated by the low of (TLR4 - TLR2) upon those cells. Pathogen-associated molecular patterns, on the other hand, cause indirect neurotoxicity by activating pattern recognition receptors porcine Reproductive and Respiratory Syndrome onto microglia, as seen in coculture models. TLR ligands effectively cause neuronal cell passing in the existence of microglia, but only if the same corresponding TLR, as well as the downstream downward MyD88 cascade in microglia, are present.(Lehnardt et al., 2006) Microglia are activated by bacterial components in a TLR-dependent manner, and microglia emit cell damage signals known as NO to adjacent neurons. Responsive(O & N)species extracted from bacteria and the host coalesce for produce highly sensitive, tissue-harmful intermediates.(Kurpius et al., 2006) Furthermore, killing parenchymal cells emit TLR ligands to endogenous (risk signals) resulting in the inflammatory tissue injury vicious cycle. These processes are significant and contribute to our knowledge of how active microglia can destroy nearby neurons, extending the significance of the results outside meningitis.(Lehnardt et al., 2006)

5. Clinical features and diagnosis

5.1. Clinical characteristic(feature)

Fever, fatigue, and migraine are common early symptoms associated bacterial meningitis are subsequently meningism neck tightness(photo- phono)phobia, and nausea appear as indicators of meningeal inflammation.(Van de Beek et al., 2004) Meningitis and migraine are two conditions that can occur together. 5-HT1B/D/F antibody agonists have been shown to inhibit inflammatory energizing for trigeminal receptive nerve strands(strand) with in meninges triptans. The function to triptans in the management of headaches in cases of(BM) bacterial meningitis, on the other hand, is yet unknown.(Lampl et al., 2000) Meningitis can be missing clinical symptoms in patients who are severely unconscious, in youngsters, or in patients who are immunocompromised, like those with liver damage. The traditional trio of warmth, neck stiffness, with changed psychological condition is observed with lower than half of people with confirmed bacterial meningitis.(Heckenberg et al., 2008) Around 33percent of patients suffer acute neurological symptoms including epileptic convulsions or limb palsy, and high to 69 percent of patients have poor awareness and 14percent have coma. (Van de Beek et al., 2004) Petechiae, which indicate meningococcal disease, or Osler's nodal, which indicate bacterial pericarditis can be seen on the epidermis. Meningitis is often a sign for bacterial pericarditis is roughly 7percent of cases.(Angstwurm et al., 2004) *Staphylococcus aureus*, which is rare to bacterial meningitis, or pneumococci have been the most prevalent pathogens within that setting. Pneumococcal meningitis (PM), endocarditis,, and asthma are sometimes diagnosed at the same time [Austrian's type symptoms]

Meningococcal illness can manifest is the severe(gram-negative) sepsis and severe cardiovascular dysfunction and disseminated intravascular coagulation, posing a risk of ischemic tissue injury. A

petechial dermatitis is not specific to meningococcal illness; it can also occur in septicemia produced by *streptococci* and *Staphylococcus aureus*.(Dalal and Ahmad, 2008)

5.2. Laboratory diagnosis

The presence of bacteria therein Cerebrospinal fluid, as shown by Gram staining (Figure 2) or +ve bacterial cultivation, is essential for the diagnosis of (BM) bacterial meningitis. Cerebrospinal fluid detection rates have been reported to be more than 90 percent, whereas blood cultures have yielded roughly 50 percent positive findings. Centrifugation of to the bigger sample or expertise can increase the diagnostic yield using CSF microscopy. When microscopic & cultural diagnosis of the pathogen fail, (PCR) may be used, but it is not currently a regular diagnostic. In meningococcal illness, Polymerase chain reaction (PCR) plays a crucial function in strain detection.(Fox et al., 2007) For important meningitis infections, latex (agglutination-based) fast data are administered, but their low accuracy and specificity make them unsuitable for routine diagnostic usage at this moment. For bacterial meningitis (BM)the Cerebrospinal fluid has a high white blood cell (WBC)count smaller than (500)cells per ml with predominant neutrophils or a high protein level smaller than1gram per liter, indicating a significant blood Cerebrospinal fluid barrier disruption. Lactate levels over (0.3 gram per liter) and a glucose (CSF per blood) level of less than 0.4 confirm the identification of severe bacterial meningitis. For resource-constrained situations when extended CSF investigations or microscopy weren't accessible, urine dipsticks to semiquantitative identification of glucose --leukocyte levels within CSF have been recommended.(Moosa et al., 1995) With(*Listeria monocytogenes*, *Mycobacterium tuberculosis*, and fungi,)reduced cell numbers and mixed pleocytosis are seen; they can also be present in meningitis that has been partly or inadequately treated. With endemic regions, cerebral malaria also isn't frequently accompanied by severe CSF pleocytosis Low Down CSF (WBC)white blood cell numbers may complicate the diagnostic in(immune-compromised) leukopenia individuals with bacterial meningitis and in cases with an excessive bacterial infection (an ulceration bacterial meningitis), stressing the necessity to Gram stains. In bacterial meningitis central (WBC) serum (CRP) C-reactive protein ,erythrocyte sedimentation rate, and procalcitonin, or other severe period proteins are frequently raised, although they have limited diagnostic utility, particularly in unusual cases. Furthermore, the above-mentioned usual CSF values might change early in the illness and in individuals who have not been well treated with antibiotics.(Michel et al., 2001)

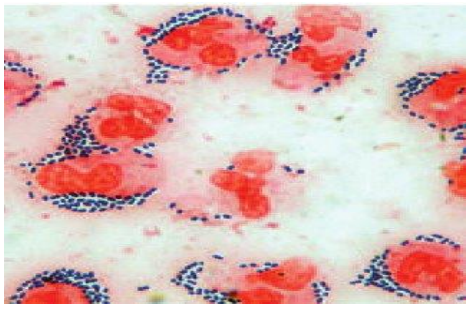

Figure 2. Typical Gram stain of CSF in a case with [PM] pneumococcal meningitis. (Neutrophils) stained red is enclosed by Gram +ve (diplococci) stained blue.

5.3. CT scan

Intracranial problems like cerebral swelling, hydrocephalus, & infarcts (necrosis)can be detected via a cranial computerized tomography (CT) scan. In addition, imaging of bone window detects parameningeal foci like sinusitis, mastoiditis, and odontogenic cyst. Local diseases are usually into pneumococcal meningitis or may necessitate surgical intervention. The use of a cranial COMPUTERIZED TOMOGRAPHY (CT) before is still debatable, doppler ultrasound with the main issue being the danger of brain herniation due to increased peripheral resistance. Patients with severe neurological impairments or seizures, as well as those with a confused state of awareness should obtain a cranial CT before doppler ultrasound. If the technology is not accessible, therapy must be started on the basis of clinical diagnosis alone, without the need for a CSF investigation. (CT) abnormalities are observed in less than 3percent of patients never any focal symptoms or seizures as well as a normal state of consciousness, therefore CSF could be taken without previous CT screening. A normal CT scan, on the other hand, may not law out intracranial pressure and the possibility of herniation.(Hasbun et al., 2001)

6. Treatment of Bacterial meningitis

6.1. Antibiotics

Antibiotic treatment should be started right away and should not be delayed due to medical delays, such as waiting for a (CT) scan. in these cases of discredited meningococcal infection, before comes to the hospital antibiotic therapy is recommended, although it is dependent on the local immunity condition and the medical setting.(Franklin et al., 2008) The blood culture must be collected prior to surgery. Although a microbiological diagnosis of the disease is not always possible, the first selection of antibiotics is generally based on guesswork. Geographic antimicrobial resistance level, patient gender, predisposing circumstances, and services are all causes to consider. Effective antibiotic therapy depends on the microbiological diagnosis and drug susceptibility of a causative factor. Antimicrobial chemotherapy must be tailored based on the cultural outcomes in order to have extremely successful

but closely focused coverage in this face of emerging resistances. Penicillin G or (BENPEN) monotherapy to (meningococci and pneumococci) on the other hand, should be used only after susceptibility has also been established. Most pathogens need ten to fourteen days of care; for uncomplicated meningococcal disease, a faster course of five to seven days is appropriate, whereas (*L. monocytogenes* - Enterobacteriaceae) need three to four weeks for treatmentTreatment periods have very little evidence and are often dependent on expert judgment. Patients with suspected or confirmed meningococcal meningitis should be isolated within the first one day of treatment, and near contacts should be given chemoprophylaxis. In patients that do not recover clinically after 2 days of antibiotic therapy, imaging of cerebral and a recurrent lumbar puncture must be considered to determine antibiotic failure.(Tunkel et al., 2004)

6.2. Corticosteroids for Bacterial meningitis

In laboratory patterns of bacterial meningitis (BM), corticosteroids suppress brain swelling, intracranial hypertension, and meningeal inflammation. Following clinical trials, findings on the possible effects of steroid employ in cases with meningitis have been mixed. The current research suggests that children from *Haemophilus influenzae* meningitis have a lower risk of permanent hearing loss.(Odio et al., 1991) Such pediatric diseases, but at the other hand, have little knowledge. In young people, alone double-blind Randomized controlled trial of 301 patients found lower death, as well as a lower risk of hearing passing and neuropsychological complications.(de Beek et al., 2002) According to subgroup study, dexamethasone's therapeutic effects was restricted the pneumococcal meningitis(PM) mortality: 34 percent versus 14 percent ; unfavorable result : 52 percent versus 26 percent (Van de Beek et al., 2006) Expert advice and many societal recommendations prescribe dexamethasone therapy for infants (%15 mg per kg every 6hr for 24 days) and grown-ups with society acquired meningitis [10 mg every 6 hrs. for 4 days] If Haemophilus influenzae [children]and streptopneumoniae [children-adult] are ruled through as the main pathogens, this therapy should be stopped. In countries that promote immunization (*Haemophilus influenzae* - *S. pneumoniae*) pathogens are diminishing in pediatric people. A primary Hydrogenatet cyclopentanophenanthrene(steroid) dose must be given 1020 minutes before starting antibiotic therapy, or at the very least at the same time. Dexamethasone does not cure current brain swelling and intracranial pressure in the final phases of meningitis so waiting is not a good idea. On the other hand, there is uncertainty for worsened neurotoxicity, which seems to must little therapeutic significance. (Weisfelt et al., 2006) As a result of dexamethasone therapy, antibiotic entry into the CSF can be hampered. Current evidence does not justify the widespread the use corticosteroids in resource-constrained countries.(Weisfelt et al., 2006)

7.SideEffect(Complications)

Bacterial meningitis(BM) has a 34 percent death rate.(Van de Beek et al., 2006) streptococcus pneumoniae or L. meningitidis have the greatest levels of virulence. more than a 50percent remain individual (long-term) neurological sequelae may be observed.(Weisfelt et al., 2006) This poor result is due both to intracranial including systemic complications. A first three days in treatment are when complications are far more likely to result. The most common issues are sensorineural silent disease and vestibular dysfunction. *Haemophilus influenzae* - *Streptococcus pneumoniae* have been the most common pathogens. Adjunctive dexamethasone treatment, as previously mentioned, lowers the risk of these problems. Brain swelling vascular changes, or hydrocephalus, all of which lead to elevated pressure of intracranial and parenchymal trauma, have been the most dangerous intracranial problems.(Pfister et al., 1992) Patients may experience a sustained or substantial change in their state of mind or state of consciousness through clinical settings. If patients do not recover after 48 hours of antibiotic therapy and if light effects symptoms appear(CT) imaging must be done. In general, elevating your head is a good idea. (30) In individuals with meningitis, getting out of sleep is advised. Osmotherapy is one of the treatment methods for brain swelling (edema). Therapeutic hypothermia, which has been shown to be beneficial in(BM) bacterial meningitis cases in the lab.(Angstwurm et al., 2000) While it's not been studied in patients, it seems that lowering an elevated blood heat is a good idea.

Up to 15percent of patients experience hydrocephalus, which normally manifests as malabsorption caused by elevated CSF discharge flow resistance. Add computerized tomography may be carefully supervised in patients with hydrocephalus and diminished consciousness they will need outer ventricular discharge in the future (EVD). ICP tracking is an added advantage of EVD. , clinical progress and (CT) scan follow-up are both used to assess the level of drainage. When CSF protein or leukocyte levels are normalized, EVD is typically no longer necessary; therefore, a ventriculoperitoneal shunt must be implanted. In patients through makes brain edema are comatose, invasive (ICP) monitoring must be considered. Vasculitis)inflamation of the blood vessels vasospasm, or blood clot (septic thrombosis) of the Dural sinuses or cortical veins are both vascular problems.(Haring et al., 1998) Broad cerebral regions are often infarcted as a result of this condition. In the context of meningitis, medically new focal neuronal deficits should prompt some diagnostic concerns. Magnetic resonance imaging (MRI), computed tomography (CT) MR or CT scan angiograms are all useful diagnostic tools. In the lack of randomized trials, the complications and advantages of preventing blood clotting in toxic sinus thrombosis remain unknown. Similarly, there are no scientific proof treatments for meningitis-related vasculitis or vasoconstriction. In the case of possible vasculitis, hemolysis and nimodipine have been proposed as alternatives to subarachnoid internal bleeding. Extracranial risks involve sepsis, distributed coagulopathy, multiorgan dysfunction, arthritis, or electrolyte inequality,

which are normally caused by SIADH release. Survivors with bacterial meningitis (BM)are often observed to have neuropsychological problems.

Long-term cognitive dysfunction is more common in adults following pneumococcal meningitis although meningococcal meningitis having a lower occurrence.(de Beek et al., 2002) Adult humans 112 years later bacterial meningitis have serious problems with a short period and memory function executive works and associative education of verbal information.(Schmidt et al., 2006) Other writers place a premium on psychomotor delay as a distinguishing characteristic.(Hoogman et al., 2007) Learning disabilities, diminished bad memory, and behavioural problems in children can also contribute to low academic success.(Grimwood et al., 2000)

8.CONCLUSION

New therapies, in addition to antibiotics, are being developed as our knowledge of the physiopathology of (BM)bacterial meningitis improves. The development of cytokines throughout the Cerebrospinal fluid, the magnitude of a chronic inflammation in the (CSF) and focal neuological deficits have all been shown to be reduced by adjunctive glucocorticoid treatment in both clinical and experimental research. More recent research gives promise to the idea of molecularly targeted treatment to decrease inflammation and increase outcomes. Combination regimens affecting steps involved through the inflammatory phase for example; cytokine synthesis or action blockers and cloning a unique (WBC) against adhesive proteins) are a realistic assumption for the forthcoming. If the genetic web of inflammation becomes more intricate, that will the possibilities for action. Polymerase chain reaction techniques will undoubtedly become a common approach as they improve and become more widely available, however more research is required to confirm their diagnostic precision Furthermore polymrase chain reaction including multiplex (PCR), will only identify pathogens that have been identified & are ivolvedn in primer mix. In a world when resistance to antibiotics is on the rise and new diseases are developing, culture coupled with serological tests maintains the gold standard for diagnosis.

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