Pathogenesis of neonatal sepsis pdf

Infectious agents associated with neonatal sepsis have changed since the mid-20th century. In the 1950s, S aureus and E coli were the most common bacterial pathogens among newborns in the United States. In the following decades, Group B Streptococcus (GBS) replaced S aureus as the most common gram-positive organism causing sepsis at first. Currently, GBS and E coli continue to be the most commonly identified microorganisms associated with neonatal infection. In neonatal sepsis, other organisms such as coagulotase-negative Staphylococcus epidermidis, L monocytogenes, Chlamydia pneumoniae, H influenzae, Enterobacter aerogens, as well as types of bacteroids and Clostridium were also found. Meningoencephalitis and neonatal sepsis can also be caused by infection with adenovirus, enterovirus or coxakivirus. In addition, sexually transmitted diseases (eg, gonorrhea, syphilis, herpes simplex virus [HSV] infection, cytomegalovirus [CMV] infection, hepatitis, human immunodeficiency virus [HIV] infection, rubella, toxoplasmosis, trichomoniasis and candidiasis) have been implicit in neonatal infection. Bacterial organisms with increased antibiotic resistance appeared and further complicate the treatment of neonatal sepsis. [7] Colonization patterns in nurseries and staff are reflected in organisms currently associated with nosocomial infection. In neonatal intensive care unit (NICUs), infants of lower birth weight and younger gestational age have increased sensitivity to these organisms. Epidermids, coagulase-negative Staphylococcus, are increasingly seen as the cause of nosocomial or late sepsis, especially in premature babies, in which it is considered the leading cause of late onset of infections. Its spread is probably associated with several intrinsic properties of the organism, which allow it to easily adhere to the plastic environments found in intravascular catheters, which are usually required for the care of these children. The bacterial capsule polysaccharide adheres well to the plastic polymers of catheters. Also, proteins found in the body (AtlE and SSP-1) increase attachment to the surface of the catheter. Adherence creates a capsule between a microbe and a catheter, preventing deposition of C3 and phagocytosis. [8, 9] Biofilms are formed on infusing catheters by aggregation of organisms that have multiplied under the protection provided by adhesiveness to the catheter. As a result of the extracellular material formed by the body, infields are produced, which provides a barrier to protect the body, as well as antibiotic action, which makes coagulase-negative staphylococcal blood infection (BSI) more difficult to treat. Toxins formed by epidermis are also associated with necrotizing enerocolitis. In addition to being a cause of Coagulate-negative Staphylococcus is ubiquitous as part of the normal flora of the skin. Therefore, it is a frequent contaminant of blood and cerebrospinal fluid (CSF) cultures. When the culture grows this organism, the clinical presentation, the number of colonies and the presence of polymorphonuclear neutrophils (PMN) per Gram coloration of the presented specimen often help to differentiate the true infection from infected culture samples. In addition to the specific microbial factors mentioned above, numerous hospitality factors predispose the newborn to sepsis. [10] These factors are particularly important in premature babies and include all levels of host protection, including cellular immunity, humoral immunity and barrier function. Immature immune protections and environmental and maternal factors contribute to the risk of newborn newborns, morbidity and mortality, especially in preterm birth and/or very low birth weight (VLBW). [10, 11] There may also be a genetic association. [10] PMNs are vital for effectively killing bacteria. However, neonatal PMNs are insufficient in hemotaxis and killing capacity. Reduced adherence to the endothelial mucosa of blood vessels reduces their ability to margate and allow the intravascular space to migrate into tissues. Once in the tissues, they may not degranculate in response to hemotak factors. In addition, neonatal PMNs are less deformable and thus less able to move through the extracellular matrix of tissues to reach the site of inflammation and infection. The limited capacity of neonatal PMN for phagocytosis and killing bacteria is further impaired when the baby is clinically ill. Finally, neutrophils are easily depleted due to reduced bone marrow response, especially in premature babies. [12] Neonatal monocyte concentrations are at adult levels; however, macrophages have impaired function and continue to exhibit decreased function in infancy. The absolute number of macrophages decreases in the lungs and probably decreases also in the liver and spleen. The propertyactic and bactericidal activity and the presentation of the antigen by these cells are also not fully competent at birth. The production of cytocline by macrophages is reduced, which may be associated with a corresponding decrease in the production of T-cells. [13] Although T cells occur in the early gestational system in the bloodstream of the fetus and increase the number from birth to about 6 months, these cells represent immature populations. These naïve cells do not multiply as easily as adult T cells when activated, and they do not effectively produce cytokines that support with B-cell stimulation and differentiation and granulocyte proliferation/ monocytic. Formation function of antigen-specific memory after primary infection is delayed, and the cytotoxic function of neonatal T cells is 50% - 100% as adult T cells. At birth, newborns are insufficient in memory T cells. Since the newborn is exposed to antigenic stimuli, the number of these memorable T cells increases. Natural killer cells (NK) are found in small amounts in the peripheral blood of newborns. These cells are also functionally immature because they produce far lower levels of interferon gamma (IFN-γ) at primary stimulation than adult NK cells. This combination of findings may contribute to the severity of HSV infections in the neonatal period. The fetus has some pre-formed immunoglobulin (Ig), which is acquired primarily by nonspecific plaque transfer from the mother. Most of this transfer occurs in late pregnancy, so lower levels are detected with increasing premapromacity. The ability of the newborn to generate immunoglobulin in response to antigenic stimulation is intact; However, the magnitude of the response initially decreased, rapidly increasing with increasing postpartum age. [14] The newborn is also able to synthesize IgM in the womb of the 10-week pregnancy. However, IgM levels are usually low at birth, unless the baby has been exposed to an infectious agent during pregnancy, which would stimulate an increase in IgM production. [10] IgG and IgE can also be synthesized in the womb. Most IgG is acquired by the mother during late pregnancy. Newborns can receive IgA from breastfeeding, but do not secrete IgA until 2-5 weeks after birth. The response to bacterial polysaccharide antigen is reduced and remains so for the first 2 years of life. The production of protein complement can be detected as early as 6 weeks of pregnancy; However, the concentration of the different components of the complement system varies widely from one newborn to another. Although some babies had complement levels comparable to those in adults, the disadvantages appeared to be greater in the alternative path than in the classic way. [16] The final cytotoxic components of the complement cascade that lead to the killing of organisms, especially gram-negative bacteria, are insufficient. This deficiency is more pronounced in premature babies. Adult supplementation activities are not achieved until babies reach 6-10 months of life. Neonatal serum levels are reduced with ofsson effectiveness against UBB, E coli and Streptococcus pneumoniae due to reduced levels of fibronectin, a serum protein that helps adhere to neutrophils and has opsonic properties. Physical and chemical barriers to infection in the human body are present in the newborn, but are functionally deficient. The skin and mucous membranes are easily broken down in the prematurely born baby. Newborns who are ill, premature or both are at additional risk due to invasive physical barriers to infections. Due to the interdependence of the immune response, the different shortcomings of the different different immune activity in the newborn plot to create a dangerous situation when the newborn is exposed to infectious threats. The intestines are colonized by organisms in the womb or when delivery by ingestion, and exposure to, amniotic fluid and genic-sex tract secretions. Immunological protections of the gastrointestinal tract are not mature, especially in the prematurely born baby. Lymphocytes are proliferated in the intestine in response to mitogenic stimulation; However, this proliferation is not fully effective in response to a given microorganism, since the antibody response and the formation of cytokines are immature to approximately 46 gestational. Necrotizing enerocolitis is associated with the presence of a number of types of bacteria in the immature intestines. Overgrowth of these organisms in neonatal lumen can be a component of multifactorial pathophysiology of necrotizing enerocolitis. Ventriculitis Ventriculitis is an initiating event in meningitis, with inflammation of the ventricular surface. The exudative material usually appears in the choroid plexus and is external to the plexus. Then an epindimite occurs, with a violation of the ventricular lining and bulges of glynal tufts in the ventricular lumen. Near these tuphs can develop glave bridges and cause blockage, especially in the aqueduct of Silvius. Lateral nitricles can be loculated, a process that is similar to the formation of abscesses. Multifunctional chambers can lead to the development of localized pockets of infection, making treatment more difficult. Meningitis is likely to occur in the choroid plexus and extends through the entricles through aqueducts and into the subarachnoid space to affect the cerebral and cerebellar surfaces. The high content of glycogen in the neonatal choroid split provides an excellent environment for bacteria. When meningitis develops from ventriculitis, effective treatment is complicated, since adequate levels of antibiotics in the brain ventricles are difficult to achieve, especially if ventricular obstruction is present. Arachnoid arachnoiditis is the next phase of the process and is a hallmark of meningitis. Arachnoid is infiltrated by inflammatory cells producing exudate, which is usually thick above the base of the brain and more uniform over the rest of the brain. At the beginning of infection, exudate contains mainly PMN, bacteria and macrophages. It is prominent around blood vessels and can expand into the brain's parenchyma. In the second and third week of infection, the proportion of PMNs decreases; dominant cells are histiocytes, macrophages, and some lymphocytes and plasma cells. Exudate infiltration can occur in the cranial roots 3-8. After this period, exudate decreases. Thick strands of collagen form together with arachnoid fibrosis, eventually leading to blockage of csf flow. Hydrocephalus UBB's early meningitis is characterized by much less arachnoiditis than with a late onset of GBS meningitis. Vasculitis vasculitis extends inflammation of the arachnoid and entricles to the blood vessels around the brain. Blockage of the arteries rarely appears; participation can be severe. Phlebitis can be accompanied by thrombosis and complete blockage of the vessels. Multiple fibrin thrombi are especially associated with hemorrhagic infarction. This vascular involvement occurs in the first days of meningitis and becomes more noticeable in the second and third week of infection. Cerebral edema can occur during the acute state of meningitis and can be severe enough to reduce ventricular lumen significantly. The cause is not known, but it is likely to be associated with vasculitis and increased permeability of blood vessels. It can also be associated with cytotoxins of microbial origin. Herniation of edematous suppression structures usually does not occur in newborns, due to the apartness of the skull. Heart attack is an important and serious feature of advanced neonatal meningitis, which occurs in 30% of infants who die. Lesions appear due to multiple venous oculus, which are often hemorrhagic. Locusts of heart attacks are most often in the cerebral cortex and basic white matter, but can also be subependimaлен within deep white matter. Neural loss occurs, especially in the cerebral cortex, and periventricular leukemia can subsequently occur in areas of neural cell death. [17] [17]

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