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Case Report: Meningitis in Postpartum

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ABSTRACT

Introduction: Meningitis is an infectious disease of the brain membranes and spinal cord. Viruses, bacteria, fungi, parasites, or aseptic actions can all cause meningitis. Bacterial meningitis is a severe infection with high mortality and morbidity rates that can induce fever in postpartum patients. The incidence of invasive *Streptococcus agalactiae Group B* (SGB) in pregnant women reached 31.58%; the older the pregnant woman, the greater the risk of invasive SGB. **Case:** The patient came to the emergency room with unconsciousness, fever, and shortness of breath. The patient presented with Ronchi +, neck stiffness +, and GCS E1M1V1. A chest X-ray found cardiomegaly as well as pulmonary edema. A full blood count indicated leukocytosis, increased neutrophils, elevated SGOT and SGPT levels, and lymphocytopenia. The patient was then given antipyretics, anticonvulsants, neurotonics, neurotrophics, diuretics, antihypertensives, and antianginals. **Conclusion:** In this study, it can be determined whether infection during pregnancy or labor can cause postpartum meningitis. The most common pathogen that can cause bacterial meningitis is *Streptococcus agalactiae Group B* (SGB), which causes most cases. Early diagnosis of postpartum meningitis is important in order to begin as soon as possible and achieve the best results for the patient's recovery.

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INTRODUCTION

Meningitis is a worldwide public health issue. This is an infectious disease of the brain membranes and spinal cord. Aseptic procedures as well as viruses, bacteria, fungi, and parasites can all cause meningitis.[1]

Bacterial meningitis is a severe infection with high mortality and morbidity that can induce fever in postpartum patients.[2,3] As of April 2012, there had been 48 reports of bacterial meningitis in pregnant women globally.[4] In 2010, there were 19,381 confirmed cases of meningitis in Indonesia, but the number of postpartum meningitis cases has never been reported.[1]

Streptococcus pneumoniae and *Listeria monocytogenes* are the most common causes of bacterial meningitis.[4,5] In addition, *tuberculosis*, *enterococcus*, and *cryptococcus* can potentially lead to meningitis.[3,6,7] *Tuberculous meningitis* (TBM) occurs in 1% of TB cases and has a mortality rate of 15–40%. The disease progresses through three stages: the prodromal phase, the meningitis phase, and the paralytic phase. These are classified based on neurological signs and symptoms. Complications are prevalent, and outcomes worsen as the disease progresses.[8]

In general, there is a complex immune system response throughout pregnancy, with different responses depending on the stage of pregnancy. Immunosuppression is used during pregnancy to keep the body from rejecting the fetus because of histocompatibility antigens passed down from the father. Downregulation of Th-1 cytokines (e.g., INF-γ) increases a number of natural anti-inflammatory responses.[9,10] CD4+ T-helper responses and numbers decrease during pregnancy, with the greatest drop occurring in the third trimester.[11]

Several hormonal changes occur during pregnancy, including the hormone progesterone, which acts as an immune system during pregnancy. Also, progesterone is one of the most important hormones for regulating the inflammatory response during pregnancy. Its anti-inflammatory effect can improve the uterine environment, which functions for fetal implantation, and reduce inflammation at this stage of pregnancy.

One of the predisposing factors for bacterial development during pregnancy is the changes in maternal T cells and other immature cell activity.[9] The number of T-regulatory cells in peripheral blood rises during early pregnancy, peaks during the second trimester, and declines postpartum. Estrogen decrease T-cell activation, and T-regulatory cell numbers correlate with estrogen levels. Progesterone is also thought to be immunosuppressive. It helps produce the T-regulatory cytokine IL-10, boosts Th2-weighted immune responses, reduces inflammatory cytokine production, reduces Th1-weighted immune responses, and suppresses allogeneic responses. As a result, pregnancy-related hormonal increases in both progesterone and estrogen appear immunosuppressive.[11]

The incidence of meningitis in postpartum results in immunosuppression which can also be associated with an inflammatory response that encourages the onset of several diseases; an appropriate diagnosis is very important so that antimicrobial treatment can be given promptly and effectively. We can consider *Listeria monocytogenes* as one of the possible causes of meningitis in postpartum women. The prevalence of *Listeria monocytogenes* infection is very likely due to the bacteria's widespread spread and the high contamination of raw and industrially processed foods, which usually cause mild illness but can develop rapidly and be associated with severe complications affecting the central nervous system, such as meningitis.[12]

A large-scale cohort study looked at a lot of adult patients who got bacterial meningitis in the community. Infection with *Streptococcus agalactiae Group B* (SGB) was found in 0.4%–7.4% of all patients. *Streptococcus agalactiae Group B* (SGB) is a normal bacterium that lives in the adult gastrointestinal system and genitourinary tract. SGB usually does not cause disease, but under certain conditions, it can infect adults over the age of 18, who are not pregnant and are more than 30 days postpartum, by invading normally sterile areas, such as blood or cerebrospinal fluid.[13] The clinical manifestations of SGB meningitis are similar to those of meningitis caused by other pathogens, in that they are acute in onset and associated with neurologic dysfunction. Patients with SGB meningitis typically arrive within 24 hours of symptom onset, making it one of the most rapidly progressive forms of bacterial meningitis.[14,15]

The incidence of pregnant women with invasive SGB reached 31.58%; the older the pregnant woman, the more at risk of invasive SGB because the body mechanism also decreases. One of the steps to prevent invasive SGB is to vaccinate during pregnancy to protect neonates and pregnant women. Penicillin G must be used as the first antibiotic for Streptococcus agalactiae Group B (SGB) because it is resistant to non-beta antibiotics, such as vancomycin. Antibiotic use is given for a minimum of 10 days; if meningitis is present, then 14 days of therapy are required.[13]

According to research by Ghani *et al.*, a 25-year-old woman was hospitalized due to premature delivery at 35 weeks; the results of the vaginal swab examination did not contain certain organisms; a negative septic examination was performed; and she was discharged. The mother had a fever, headache, and shortness of breath seven days after giving birth. *Streptococcus agalactiae Group B* (SGB) was found

during a lumbar function.[16]

Pastick *et al*. did a study that found 19 cases of *cryptococcus* in pregnant and postpartum women who were infected with HIV. There was also cryptococcal meningitis in all of these women.[7] As many as 100,000 people die each year in developing countries from cryptococcal meningitis, which is common in sub-Saharan Africa.[11] The World Health Organization (WHO) recommends that HIV patients with a CD4+ count of less than 100 cells/µl should be screened for cryptococcal antigen (CrAg). Preventive treatment and screening for CrAg have been shown to reduce the risk of meningitis and death, but not yet in pregnant women.[7]

Based on the discussion above, it is important to determine whether women with postpartum fever have previously undergone regional anesthesia during labor or after delivery, as this can be a source of postpartum meningitis. Women who had epidural analgesia in labor were shown to be significantly more likely to develop a fever than women who used other pain relievers like pethidine. Women who acquired pyrexia as a result of epidural use typically had raised temperatures only during birth or in the postpartum period.[5]

A postpartum headache is often described as a headache and neck or shoulder pain that occur within the first six weeks after giving birth. It is one of the most common symptoms, with up to 39% of postpartum women suffering headaches in the first week postpartum.[17] Post-dural puncture, subdural hematoma, meningitis, and cortical vein thrombosis are the most common causes of postpartum headaches within the first six weeks after delivery with neuraxial anesthesia.[18]

There are two ways for bacteria to enter the cerebrospinal fluid: aseptic mistakes that can exogenously introduce bacteria, and needle-caused microscopic bleeding that allows bacteria to travel from the bloodstream to the subarachnoid space. Aseptic meningitis is a condition where drugs injected into the subarachnoid space directly irritate the meninges or cause a hypersensitivity reaction. Another cause of meningitis that can be postoperative is drug-induced meningitis. Associated drugs are *non-steroidal anti-inflammatory drugs* (NSAIDs), antibiotics, and H2 receptor blockers.[19]

Postpartum meningitis is a rare case, so we are interested in reporting a case of postpartum meningitis in a 28-year-old female patient who was hospitalized at Widodo Hospital, Ngawi, in December 2022.

CASE

Patient M, a female, 28 years old, came to the Emergency Department (ED) of Widodo Hospital, Ngawi, on December 28, 2022, with complaints of unconsciousness. In the last three days, the patient had a fever, dizziness, and a tight cough. The patient had a vaginal delivery for her second child at the hospital 16 days before being admitted. She denied having a past history of diseases like asthma, diabetes mellitus, and hypertension.

Physical examination showed that the patient was unconscious with a GCS of 111, a respiratory rate of 28 beats per minute, a pulse rate of 120 beats per minute with strong and regular beats, a blood pressure of 180/100, and a body temperature of 39.8 degrees Celsius. The patient's oxygen saturation is currently 96% with a nasal canal of 4 liters/minute. The pupils were incorrect, with positive light reflexes in both eyes. On examination, meningeal signs: positive rigidity, negative Kernig sign, negative Lasegue test, negative Brudzinki I and II. Rhonchi and vesicular breath sound were heard in the right and left lungs. The heart, abdomen, back, extremities, genitals, and rectum were within normal limits.

A complete blood test was taken when the patient was admitted to the hospital emergency room on December 28, 2022. On that date, hemoglobin was 12.2 g/dl; leukocytes were 19.9 cells/µL; platelets were 411,000 cells/µL; neutrophils were 79.1%; lymphocytes were 18.4%; and erythrocytes were 4.8x106 µL; MCV was 77.7 fL; and MCH was 25.2 pg. The patient's total blood glucose level was 231 mg/dl. The SGOT and SGPT levels were 70 and 37 u/L, respectively. The patient's potassium and calcium values were 2.86 and 0.91 mmol/L, respectively. On December 28, 2022, radiology tests showed cardiomegaly with signs of pulmonary edema.

Meningitis, cardiomegaly, and pulmonary edema were all suspected in this patient. While in the emergency room, the patient was given 10 liters of oxygen per minute through a non-rebreathing mask, a NaCl infusion of 10 drops per minute, a citicolin injection of 500mg, a cefotaxime injection of 1 gram, a furosemide injection of 2 ampoules, and a sanmol injection of 1 gram. The patient was advised to do further laboratory and thorax examinations, as well as a head CT scan.

The results of consultation with a neurologist obtained further therapy for citicolin injection (2 x 500 mg), piracetam injection (3 x 3 gr), and mecobalamin injection (2 x 1 gr). After seeing a cardiologist, the patient was given more furosemide injections (5 ampoules at 50 cc at a speed of 2.5 cc/hour), oral spironolactone (1 x 25 mg), ramipril (1 x 5mg), and nitrocaf (2 x 2.5 mg). The results of consultation with an internal medicine specialist obtained further therapy for injection of sanmol infusion (3 x 1 gr), hepatin (3 x 1), and KSR (2 x 1).

Urinalysis was performed in the Intensive Care Unit (ICU) on December 29, 2022, and resulted in a

yellow color, slightly cloudy, with a PH of 5.0 and a specific gravity of ≥ 1.025. Macroscopic examination found one positive protein and a positive reduction, while urobilinogen, ketone, and nitrite were negative. Microscopic examination revealed 10–12 epithelium cells per visual field (PLP), 8–10 leukocytes PLP, 2–3 erythrocytes PLP, amorphous crystals, and positive bacteria. Ca oxalate crystals, uric acid, fungi, granular cylinders, hyaline cylinders, lekocyte cylinders, erythrocyte cylinders, and thyrosin were all found to be negative. The HbA1C examination was found to be 5.7%.

On December 29, 2022, a complete blood test was performed again in the ICU room, and the results were as follows: hemoglobin 13.4 gr/dl; leukocytes 12.9 cells/µL; platelets 344,000 cells/µL; neutrophil type count 93.9%; lymphocyte type count 3.6%; erythrocytes 5.3x106 µL; MCV 75.1 fL; and MCH 25.2 pg. The patient's total blood glucose was 123 mg/dl. The SGOT and SGPT showed 82 and 71 u/L, respectively. The patient's potassium and calcium values were 2.2 and 0.72 mmol/L, respectively. On December 30, 2022, the CT scan result revealed no abnormalities.

DISCUSSION

Bacterial meningitis can be indicated by the triad of meningitis symptoms (fever, stiffness, and changes in mental status), as well as by a physical examination to find a stiff neck, Kernig's sign, or Brudzinski sign.[16,20] The patient in this case presented with a fever for three days before admission, arrived at the hospital unconscious (GCS E1M1V1), and revealed positive rigidity on physical examination. Bacterial meningitis, in general, also causes cranial nerve defects. This is consistent with what happened to this patient, who had a decrease in consciousness as a result of the GCS E1M1V1 examination results.[1,11] On physical examination, there was hypertension, hyperpyrexia, hypercapnea, and tachycardia. A positive finding of rhonchus in both lung fields on physical examination supported the thorax X-ray results showing pulmonary edema.

Several hormonal changes occur during pregnancy, including the hormone progesterone, which acts as an immune system during pregnancy. Besides that, progesterone is one of the most important hormones for regulating the inflammatory response during pregnancy. Its anti-inflammatory effect can improve the uterine environment, which functions for fetal implantation, and reduce inflammation at this stage of pregnancy. One of the predisposing factors for bacterial development during pregnancy is the changes in maternal T cells and another immature cell activity.[9]

Immunosuppression in postpartum conditions can also be associated with inflammatory responses that support the onset of several diseases, such as meningitis. Postpartum immunity changes over a long period of time and can last up to one year after delivery.[12]

There are two ways for bacteria to enter the cerebrospinal fluid: aseptic mistakes that can exogenously introduce bacteria, and needle-caused microscopic bleeding that allows bacteria to travel from the bloodstream to the subarachnoid space. Aseptic meningitis is a condition where drugs injected into the subarachnoid space directly irritate the meninges or cause a hypersensitivity reaction. Another cause of meningitis that can be postoperative is drug-induced meningitis.[19] The patient in this case had a vaginal delivery for her second child at the hospital 16 days before being admitted. The woman's immune system isn't as strong as it should be after giving birth, which means that various kinds of possible bacteria can get in.

Streptococcus agalactiae Group B (SGB) is a normal bacterium that lives in the adult gastrointestinal system and genitourinary tract. SGB usually does not cause disease, but under certain conditions, it can infect adults over the age of 18, who are not pregnant and are more than 30 days postpartum, by invading normally sterile areas, such as blood or cerebrospinal fluid.[13] The suspected bacterial cause of postpartum meningitis in this case was *Streptococcus agalactiae Group B* (SGB), with rapid disease progression, despite the fact that fluid analysis and cerebrospinal fluid culture were not conducted because the patient died the next day.

Listeria monocytogenes infection is very common due to the bacteria's widespread spread and the high frequency of contamination of raw and industrially processed foods, which usually cause mild disease but can progress rapidly and be associated with severe complications affecting the central nervous system. *L. monocytogenes* can be a possible cause of meningoencephalitis in postpartum, which can be considered a transient underlying immunosuppressive condition.[12]

Streptococcus agalactiae Group B (SGB) invasive disease is defined as SGB isolation from a normally sterile site (e.g., blood or cerebrospinal fluid) in a woman more than 30 days postpartum.[13] *E. faecalis* may also be the cause of meningitis in these patients as a result of infection transmission during invasive procedures such as, neuraxial anesthesia, either epidural or spinal, inducement of labor, catheter insertion, and so on.[3]

A lumbar puncture (LP) is essential to confirm the diagnosis of meningitis. A meningitis diagnosis necessitates obtaining a cerebrospinal fluid (CSF) sample from the space around the brain and spinal

cord. However, it was not possible for this patient due to the patient's rapidly deteriorating condition and the inadequate facilities available at the hospital. The presence of white blood cell levels can be used as a clue.

The patient frequently undergoes antenatal care (ANC) examinations with negative HIV findings, ruling out the potential for immunocompromised diseases like HIV in this patient. There was no history of previous disease in this patient's anamnesis for cardiomegaly and pulmonary edema, but blood pressure was 180/100 based on vital sign examination results. In the right and left lungs, vesicular and rhonchi sounds were heard. On December 28, 2022, radiology results showed cardiomegaly with signs of pulmonary edema. These tests cannot rule out the possibility due to the presence of other disorders like pre-eclampsia or postpartum heart disease, such as postpartum cardiomyopathy.

This patient had a complete blood test and urine, which revealed leukocytosis, increased neutrophils, and elevated SGOT and SGPT levels. Proteinuria and increased leukocyte sediment were found during urinalysis. Patients who are immunocompromised, have decreased consciousness, and are suspected of having a central nervous system require a computed tomography of the brain.[19] CT scans of this patient revealed no abnormalities.

Meningitis must be diagnosed rapidly so that antimicrobial treatment can begin as soon as possible for the best chance of a full recovery.[12] Patients at high risk, such as postpartum women, are advised to get ampicillin in addition to ceftriaxone and vancomycin as first-line empiric treatment for suspected bacterial meningitis.[8,12] Cefotaxime 1 g was injected to this patient. Pregnant women can protect themselves against invasive SGB disease and prevent meningitis by receiving a vaccination.[2] Aseptic precautions must also be observed by healthcare staff, from catheter insertion to invasive operations such as neuraxial anesthesia, either epidural or spinal, or induction of labor.

CONCLUSSION

Prevention of postpartum meningitis can be done by quickly treating concerns about infection by administering prophylactic drugs when performing risky medical procedures such as infusion, catheter insertion, anesthesia, caesarean section procedures, or normal delivery, especially to susceptible subjects. Besides that, routine checks or monitoring during pregnancy are also needed by conducting antenatal care (ANC) checks at health facilities so that early screening can be done for at-risk pregnant women.

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Conflict of Interest
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Author Contribution
The author contributed to the collection of data, data analysis, and writing the manuscript.

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