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The Adverse Outcomes of Chorioamnionitis on the Pregnant Female and Their Fetus

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ABSTRACT

Introduction: Chorioamnionitis is an intra-amniotic infection caused by an ascending polymicrobial bacterial infection. Triple I (intrauterine inflammation or infection or both) is also proposed to replace chorioamnionitis due to its imprecise definition and variable clinical manifestations. Intraamniotic infection is characterized by inflammation of the amniotic fluid, placenta, fetus, fetal membranes, or decidua. Maternal fever, uterine tenderness, maternal tachycardia, fetal tachycardia, and purulent fluid coming from the cervical os (the cervical opening can be external, near the vagina, or internal, near the uterus) are the key clinical findings that are crucial for the diagnosis of clinical chorioamnionitis. Clinically, intra-amnionitic infections have been linked to severe morbidity and death in pregnant women, fetuses, and newborns.

Method: A systematic search of PubMed, Embase, LitCovid, MedRxiv, BioRxiv, Google Scholar, EBSCO MEDLINE, CINAHL and Scopus electronic database was done using key words like Chorioamnionitis, adverse complication in pregnancy to mother and her fetus associated with intrauterine infection, Intraamniotic inflammation and triple I. Studies have been included from January 2015 to December 2022.

Result: Chorioamnionitis is most typically caused by ascending infection and is frequently linked with premature membrane rupture. Chorioamnionitis is typically caused by a polymicrobial infection, with Ureaplasma urealyticum, Mycoplasma hominis, and Gram-negative anaerobes being common pathogens. Antimicrobial drugs, antipyretics, rapid treatment, and supportive care are the mainstays of management.

Conclusion: Overall, this review provides a better understanding of Chorioamnionitis and its potential impact on public health. By shedding light on this emerging disease, we hope to contribute to ongoing efforts to prevent and treat of this serious disorder.

Keywords: *Chorioamnionitis, Intrauterine Infection, Pregnancy, adverse complication, Triple I, Intraamniotic Inflammation*

INTRODUCTION

The placenta's membranes and chorion are acutely inflamed in chorioamnionitis, also known as an intra-amniotic infection. This condition is often brought on by an ascending polymicrobial bacterial infection following membrane rupture. When very small, fastidious genital mycoplasmas such Ureaplasma species and Mycoplasma hominies are present in the lower genital tract, chorioamnionitis may develop with intact membranes [1].

The term "clinical chorioamnionitis" refers to a broad clinical illness that may include any one or more of the following symptoms:

fever, uterine pain, amniotic fluid that smells bad, or an elevated white blood cell (WBC) count. However, the presence of one (or even more than one) of these symptoms or signs does not always mean that there is histopathologic chorioamnionitis or intrauterine/intra-amniotic inflammation. In a study of patients with preterm clinical chorioamnionitis, 66% of the patients exhibited negative amniotic fluid cultures and 24% of the patients showed no signs of either intra-amniotic inflammation or infection. It's interesting to note that the placenta only had identifiable bacteria in 12% of patients with acute histopathological chorioamnionitis at term [2].

With significant variance in presentation of pregnant women with chorioamnionitis and their newborns, the term "chorioamnionitis" has been used indiscriminately to describe a heterogeneous array of disorders characterized by infection and inflammation or both. The National Institute of Child Health and Human Development (NICHD) expert panel proposed to replace the term chorioamnionitis with the more general, descriptive term "IUI or infection or both," abbreviated as "Triple I," due to the imprecise definition and the variable clinical manifestations. The group suggested a classification for Triple I and suggested methods for evaluating and treating pregnant women with Triple I and their unborn children [3].

An Intraamniotic infection with accompanying inflammation of the amniotic fluid (AF), placenta, fetus, fetal membranes, or decidua is referred to as an intraamniotic infection (IAI). The amniotic sac, which encloses the fetus and is one location of maternal/fetal immunological interaction, is made up of fetal (chorion and amniotic membranes) and maternal (decidua) components. A layer of epithelial cells, a layer of mesenchymal cells beneath them, an extracellular matrix, and collagen with a high tensile strength make up the fetal tissue known as the amnion. A reticular layer, the basement membrane, and trophoblasts make up the chorion. In close proximity to the chorion, the decidua, or modified maternal endometrium of pregnancy, is made up of maternal immune cells, decidual stromal cells, and extravillous fetal trophoblasts [4].

Acute chorioamnionitis and funisitis have traditionally been attributed to intraamniotic infection; however, recent research suggests that "sterile" intraamniotic inflammation, which happens in the absence of observable microorganisms and is brought on by "danger signals," is frequently linked to this condition also. Chemokines (such interleukin-8 and granulocyte chemotactic protein) create a gradient during intraamniotic infection that favors neutrophil migration from the maternal or fetal circulation into the chorioamniotic membranes or umbilical cord, as appropriate. Neutrophil chemokines can also be generated as a result of danger signals released during cellular stress or cell death. According to gestational age at birth, chorioamnionitis is found in 3-5% of term placentas and 94% of delivered placentas at 21-24 weeks of gestation [5].

MATERIALS & METHODS

When searching through comprehensive databases like PubMed, Embase, LitCovid, MedRxiv, BioRxiv, Google Scholar, EBSCO MEDLINE, CINAHL, and Scopus, terms like "Chorioamnionitis," "pregnancy," "preterm premature rupture of the membrane," and "Intraamniotic infection" were used to find studies published between January 2015 and December 2022.

The title and abstract of each article were originally checked without regard to

language. Then, a few complete texts of accepted papers were chosen. For each particular subject, systematic reviews that included meta-analysis were first included. Also included were recent RCTs that had been published but had not been covered by systematic reviews. We used experimental, non-randomized prospective, uncontrolled prospective, retrospective, and meta-analyses in addition to meta-analyses and RCTs to support our findings [28].

EPIDEMIOLOGY

About 4% of term deliveries have chorioamnionitis, although preterm births and early membrane ruptures have it more frequently [6].More than 94% of births between 21 and 24 weeks gestation are associated with histological chorioamnionitis [7].Failure to advance is linked to term deliveries in chorioamnionitis-affected moms. Preterm birth is likely to result from chorioamnionitis in preterm labor. According to studies, chorioamnionitis or inflammation of the placenta can be present in anywhere between 8% and 50% of premature births [8,9].

Many studies have found factors that can increase the chances of a woman getting chorioamnionitis. These things include when the water breaks for a long time, labor lasting a long time, never having given birth before, being African American, having devices inside the vagina to monitor labor, having many vaginal exams, the fluid around the baby being dirty, smoking or using drugs, having a weak immune system, getting an epidural for pain, having a certain bacteria called group B streptococcus, having bacterial vaginosis, getting infections from sex, and having a certain bacteria called ureaplasma in the vagina [10-14, 15-20].

The maternal HIV status is not a risk factor for chorioamnionitis, despite the condition being a risk factor for vertical transmission during pregnancy. In a Study by Neman et al, both groups of women in the trial of 298 women with comparable risk factors and demographics, experienced a similar incidence of chorioamnionitis. The frequency of vaginal examinations performed during childbirth was significantly correlated with the greater incidence for each group [21].

MECHANISM:

The pathophysiology of chorioamnionitis is characterized by the transmission of infectious agents to the placental umbilical cord and/or chorioamnion [22,23]. By causing FIRS (Fetal Inflammatory Response Syndrome), bacteria begin attacking the placenta and umbilical cord. This results in funisitis (cord necrosis and inflammation), chorionic vasculitis, avascular villi, and intervillous bleeding, which reduces the baby's oxygen supply. Therefore, the baby's body begins manufacturing cytokines in reaction to infection. The body of the mother also begins to produce cytokines, which eventually reach the baby's body. Therefore, as a result of an excessive generation of cytokines, there was an excessive amount of inflammation, which ultimately caused cerebral palsy or a long-term neurological impairment.

CLINICAL SIGN AND SYMPTOMS

Maternal fever, uterine tenderness, maternal tachycardia, fetal tachycardia, and purulent fluid coming from the cervical os (the cervical opening can be external, near the vagina, or internal, near the uterus) are the key clinical findings that are crucial for the diagnosis of clinical chorioamnionitis [24].

During the antepartum and postpartum periods, a persistent maternal temperature of more than 38 degrees Celsius, or 100.4

degrees Fahrenheit, is regarded as abnormal. Infections like chorioamnionitis can raise a woman's body temperature, but non-infectious causes including epidural anesthesia and high ambient temperatures have also been linked to raised maternal temperatures [25,26]. 95–100% of clinical chorioamnionitis patients include fever, which is often necessary for the diagnosis. Epidural fever is a common complication of epidural anesthesia, especially in nulliparous women who have labored for a long time. This condition makes it difficult to diagnose chorioamnionitis [25].

Furthermore, maternal tachycardia with a heart rate (HR) greater than 100 beats per minute and fetal tachycardia with a fetal heart rate (FHR) larger than 160 beats per minute are both strongly related with chorioamnionitis [27]. According to one study, 40–70% of chorioamnionitis patients had fetal tachycardia whereas 20–80% of cases had maternal tachycardia [11]. Tachycardia may be present even in the absence of chorioamnionitis, therefore it has to be carefully evaluated for other possible causes. Ephedrine, antihistamines, and beta agonists are examples of drugs that can increase the heart rate of the mother or the fetus. However, the presence of both maternal fever and maternal and/or fetal tachycardia strongly suggests an infection of the uterus, and this should be treated as chorioamnionitis, until proved otherwise [27].

Uterine discomfort and purulent fluid emanating from the cervical os are two additional, more ambiguous indications of chorioamnionitis. Physical examination is used to determine uterine tenderness, which might be confused by contraction discomfort or covered up by epidural anesthetic [11]. Even while purulence or an unpleasant odor of amniotic fluid may be organism-specific and more likely to be present in cases of severe or protracted infection, doctors may or may not be able to detect it [27].

DIAGNOSIS

i) Clinical Signs And Findings:

There are no set standards for diagnosing chorioamnionitis. The diagnosis is a clinical one based on maternal and fetal signs of intrauterine infection. The most common symptoms can be fever, tachycardia, uterine pain, foul-smelling amniotic fluid, and maternal leukocytosis which are seen in maternal manifestations and the fetal symptoms might show up as a fetal heart rate rhythm that is unsettling, including tachycardia and diminished variability [28.]

However, maternal fever is the most important and common observation, and the majority of studies see it as the diagnostic identifier. In fact, intrapartum fever nearly usually indicates the presence of intraamnionic infection in a woman with ruptured membranes when there are no other clear explanations [29].

ii) Laboratory studies:

Leukocytosis may occur in chorioamnionitis patients, but it may also be a common occurrence in laboring women who show no overt indications of infection [29]. The presence of a left shift, bandemia (>9%), maternal leucocytosis (variously defined as WBC >12,000/mm3 or >15,000/mm3), or both might help to confirm the diagnosis of chorioamnionitis. In around 70–90% of instances of clinical chorioamnionitis, leucocytosis is recorded [27].

An inflammatory reaction is typically accompanied by an increase in C-reactive protein, an acute-phase reactant [30]. Though its specificity is limited, it has been hypothesized that an increased C-reactive protein may be related to chorioamnionitis. Numerous factors might contribute to the elevation of this acute phase response. In practical terms, this examination carefully help's with chorioamnionitis clinical diagnosis [29].

The most accurate test is a culture of the amniotic fluid, but its usefulness is restricted because it might take up to three days to receive the findings. Additionally, amniocentesis is not carried out in the majority of situations, which take place during delivery, due to the intrusive nature of the treatment. To confirm chorioamnionitis that has been clinically suspected and to evaluate if preterm delivery is necessary, some practitioners employ amniocentesis [31].

DIFFERENTIAL DIAGNOSIS

Females usually present to the hospital with abdominal pain, fever and tachycardia during their pregnancy. Often this can be mistaken as appendicitis, urinary infection, pneumonia and influenza.

COMPLICATIONS

The chance of cesarean delivery is raised by 2 to 3 fold by chorioamnionitis, and the risk of endomyometritis, wound infection, pelvic abscess, bacteremia, and postpartum hemorrhage is increased by 2 to 4 fold [32]. The rise in postpartum bleeding appears to be brought on by uterine muscle contractions that aren't working properly because of inflammation. If not treated timely, infection can spread and lead to bacteremia, sepsis, multiorgan failure and Disseminated Intravascular Coagulation (DIC). Clinically, intra-amnionitic infections have been linked to severe morbidity and death in pregnant women, fetuses, and newborns [33].

Neonatal depression at delivery, neonatal sepsis, the requirement for mechanical breathing, intraventricular hemorrhage, fetal inflammatory response syndrome (FIRS), and newborn mortality are some of the fetal and early neonatal effects of chorioamnionitis [34-35]. Compared to term newborns, preterm neonates have a greater risk of problems [35].In general, up to 40% of instances of early-onset newborn sepsis are linked to chorioamnionitis. Another well-known risk factor for long-term neurodevelopmental disabilities is chorioamnionitis, especially when it develops before term [36-37].

MANAGEMENT

In the case of clinical chorioamnionitis, prompt antibiotic medication beginning is crucial to prevent problems for both the mother and the fetus [38]. Antibiotic treatment is the main care of chorioamnionitis. Ampicillin and gentamicin are the most widely used antibiotics. For women allergic to penicillin, other antibiotics include clindamycin, cefazolin, and vancomycin [39].

Clindamycin every 8 hours (or metronidazole) is frequently administered for anaerobic coverage if cesarean birth is done. A single intravenous extra dose of antibiotics should be administered after delivery (5% failure rate) [40]; subsequent oral antibiotic therapy is often not helpful [41]. The normal treatment is to provide gentamicin every 8–24 hours and ampicillin every 6–6 hours intravenously till birth [42].

The common pathogenic organisms causing intra-amniotic infection are likewise covered by second- and third-generation cephalosporins, extended-spectrum penicillins, and, like ampicillin and gentamicin, these medications cross the placenta and have equivalent maternal and cord blood levels [43]. These antibiotics produce therapeutic serum concentrations in the fetus and have high efficacy against group B streptococcus and E. coli, the two main causes of neonatal sepsis.

Traditionally, gentamicin was given every eight hours. Daily gentamicin dose in the non-obstetric population seems to be less harmful, more practical, and cost-effective. Gentamicin's bactericidal action is concentration-dependent, meaning that higher peak concentrations have a stronger bactericidal effect. Additionally, greater peak dosages result in the post-antibiotic effect, which is defined as the continuation of the inhibition of bacterial growth at residual drug levels below the minimum inhibitory concentration. Gentamicin has been linked to renal and ototoxicity, however it may not be due to transiently high peak blood

concentrations that lead to excessive drug accumulation in the renal and cochlear systems, but rather to persistent moderate drug levels [44].

It is debatable whether or not to provide corticosteroids when there is a clinical intra-amniotic infection. Between 24- and 34-weeks' gestation, instances with a higher risk of premature birth are treated with corticosteroids to encourage fetal lung maturity. Theoretically, corticosteroids might make the maternal or neonatal infectious process worse even if there is no data proving damage in the context of intra-amniotic infection. Chorioamnionitis is a contraindication to the administration of corticosteroids, the National Institutes of Health consensus conference determined. Historically, randomized studies of corticosteroid treatment have excluded women with intra-amniotic infection [45].

The majority of chorioamnionitis clinical therapy strategies are based on observational or cohort research, with just a small number of randomized controlled trials. Antibiotics should be administered as soon as possible to reduce maternal and newborn morbidity. The most often researched antibiotic regimen consists of gentamicin and ampicillin, and new research favors daily gentamicin dose rather than three times daily dosing.

CONCLUSION

During pregnancy, chorioamnionitis is a serious medical illness that needs close monitoring. The hazards it might provide to both the mother and the child highlight how crucial early discovery, swift treatment, and preventative actions are. The frequency and severity of chorioamnionitis are significantly decreased by prenatal care, regular checkups, and adherence to infection control practices during labor and delivery. Expectant moms and healthcare professionals may collaborate to promote the greatest results for both maternal and newborn health by remaining aware about the signs, causes, and consequences of this illness.

Healthcare professionals cannot, however, fight this battle alone. Active participation from society is crucial. To ensure that policies prioritize maternal and newborn health and that research has the resources it needs to discover ground-breaking diagnostic tools and treatment modalities, we must cultivate a culture of support at the individual and systemic levels. Together, we can build a wall of defense that is supported by wisdom, empathy, and a steadfast dedication to the welfare of expectant mothers and the babies.

Declaration by Authors

Ethical Approval: Not Required

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