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Incidence of Congenital Toxoplasmosis in Newborn Infant in the Western Amazon, Brazil

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Abstract: *Toxoplasmosis is a very common infection in our environment. The congenital form is the most worrisome, as it causes neurological and ocular lesions, leading to late sequelae, and may also cause abortions and death of the newborn. The objective of the study was to analyze the incidence of Congenital Toxoplasmosis in newborns in the State of Rondônia, Western Amazonia, in the period corresponding to 4 (four) years. The blood collection method was used on filter paper and the Elisa technique for the capture of Toxoplasmosis IgM. The results found in 102,963 newborns who underwent toxoplasmosis, 126 presented alterations. It is concluded that Congenital Toxoplasmosis in Rondônia is 1: 817 on newborns triates.*

Keywords— Congenital Toxoplasmosis. Newborns. Rondônia. Western Amazon.

I. INTRODUCTION

T. gondii Congenital Toxoplasmosis is a result of an acute asymptomatic infection, which is acquired by the mother during pregnancy. Few cases of Congenital Toxoplasmosis are now known to have occurred in immunocompetent women when they acquired the infection six to eight weeks before conception, so it is understood that vertical infection occurs when infection occurs during pregnancy 1, [2]. Collaborating to the best understanding, they affirm that immunodeficient women with chronic infection, can transmit infection to the fetus [1], [2]. The risk of this occurring is difficult to quantify, it is probably low. The risk of the fetus is not correlated with the fact that it is the infection of the symptomatic or asymptomatic mother during pregnancy [3].

The severity and incidence of Congenital Toxoplasmosis are related to the quarter in which the infection was acquired by the mother. Children born to mothers who acquired the infection in the first and second trimesters often show severe Congenital Toxoplasmosis. In contrast, the majority of children born to women who acquire during the third trimester are born with the subclinical form of the infection. However, if untreated, 85% of these

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children may develop symptoms and signs of this disease, in most cases, chorioretinitis and developmental delay [4], [5].

As the fetal infection occurs when a pregnant woman is infected, for the pregnant woman the disease is usually asymptomatic and may not manifest itself. Infection of the fetus, however, can cause serious problems, and if it occurs early in pregnancy, the severity is much greater. It is therefore seen that Congenital Toxoplasmosis is one of the most severe forms of the disease, in general, provoking varied symptoms, but commonly framed within the Sabin syndrome or tetrad. Thus characterized: chorioretinitis in 90% of the cases, cerebral calcifications in 69% of the cases, neurological disorders in 60% of the cases and alteration of the cranial volume in 50% of the cases [6].

Congenital Toxoplasmosis is transmitted by Toxoplasma gondii, a protozoan of worldwide distribution, with high serological prevalence. The most serious form of the disease is found in newborn infants, when the mother is initially infected, or during gestation through poorly baked meat, poorly washed fruits and vegetables, or by manipulating cat feces contaminated with the cyst. The transmission occurs when the pregnant woman during pregnancy, and, presenting the acute phase of the disease transmits Toxoplasma gondii to the fetus. Epidemiological studies have revealed that in most areas of the world the presence of cats is a primary factor of great importance in the transmission of the parasite. T. gondii infection has not been found where there is no presence of cats. Oocyst excretion occurs in approximately 1% of cats in different areas of the world [7].

Raw or undercooked meats containing viable cyst compared to oocyst ingestion do not change the frequency of infection. Studies conducted in France, where eating undercooked meat is common, the prevalence of infection is high, and meat may be an important cause of infection (it was in Paris in France that the hypothesis of propagation of T. gondii from meat to man has been proven). In contrast, Central American countries have a high prevalence of human infection, but meat intake is not common [8].

The most common clinical manifestation is asymptomatic, but there may also be a febrile syndrome, with complaints of fatigue, malaise, headache and muscle pain. Congenital Toxoplasmosis classically described by Sabin in 1942 is characterized by retinocoriditis, hydrocephalus, intracranial calcification in the form of disseminated nodulations and seizures, and microphthalmia may also occur. In these more severe forms, it is also usual to involve several organs, and pneumonia, myocarditis, cholestasis, anemia, thrombocytopenia, meningoencephalitis and cerebrospinal fluid abnormalities may occur [9].

However, most infected newborns do not present clinical abnormalities at birth. This difficulty in the recognition of the congenital infection generates difficulties in the clinical diagnosis, therefore, the importance of exams such as neonatal screening (test of the foot) soon at the beginning of the birth, to obtain a therapeutic success, not leading to late, mainly visual sequelae and neurological disorders.

The manifestations of Congenital Toxoplasmosis are variable, depending greatly on the gestational age at which the mother was infected. The infection mainly involves the nervous system and the muscular and connective tissues. Among the main signs and symptoms are: intracranial calcifications, changes in the central nervous system, microcephaly, hemiplegia, abnormal muscular tonicity and active chorioretinitis (the most common sequel, being that the risks of new complications remain for some years). In most cases, toxoplasmosis occurs asymptomatically, or, with subtle manifestations like fever lymphadenopathies that regress spontaneously. However, when the infection occurs in the gestational period, the fetus may be affected with risks of serious ophthalmologic and neurological lesions, and definitive sequelae. In this way, the identification and early treatment of the disease in the pregnant woman, the fetus and the newborn are fundamental, since they avoid or attenuate the present and resulting lesions of the disease during pregnancy [6].

The consequences to the fetus of maternal toxoplasmosis will depend on the degree of exposure of the fetus to toxoplasms, strain virulence, the ability of maternal antibodies to protect the fetus, and, from the gestational period. It is known that 40% to 50% of infected fetuses die. The most common changes or fetal lesions are: 1st trimester of pregnancy: Abortion; 2nd trimester of pregnancy: Abortion or premature birth; 3rd trimester of gestation: the child can be born normal and present evidence of the disease some days, weeks or months after childbirth [10].

Toxoplasma gondii is a protozoan that parasites birds (chicken, pigeons), mammals, including man and primates [11]. T. gondii infection is a zoonosis of global dimension, this organism infects herbivorous, omnivorous and carnivorous animals, including birds [12].

It belongs to the Apicomplexa phylum that has as characteristic the presence of an apical complex visible only by electron microscopy. This complex is constituted of conóides, polar ring, subpeliculares microtubules, roptrias, micronemas and dense granules. Toxoplasma has

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no host or host cell specificity, developing in almost all organic tissues [9]. It is an intracellular parasite, with tropism by cells of the reticuloendothelial system, muscle, nervous system and retina. Free forms are found circulating only for a short time [13].

The toxoplasmosis parasite has three forms, which are: the tachyzoites, the bradyzoites and the oocysts.

The tachyzoites are found during the acute phase of the infection, being characteristically a mobile form of rapid multiplication by endodiogenia. The parasites penetrate host cells actively, or by phagocytosis, and multiply within the cytoplasmic vacuole (vacuoles parasitophore) of several cells. This multiplication destroys the host cell, but the appearance of the lesions will depend on the ability of the cells to regenerate. These forms can be found in organic liquids, excretions and secretions, hepatic, pulmonary, nervous and muscular cells are little resistant to the actions of the gastric juice that destroys them in a short time. They are presented as a half-moon shape, with one end being tapered and the other rounded, measuring about 4-9x2-4µm, with a core in a more or less central position [9]. The survival of the tachyzoites is due to the formation of a vacuole that protects against the lysosomal function consequently the acidification does not occur. The active invasion of the macrophages by the tachyzoites is not an oxidative killing mechanism of the activator. An effective immune response significantly reduces the number of tachyzoites in all tissues [12], [14], [15].

Bradyzoites are forms of slow multiplication, characteristic of chronic infection. The bradyzoites found within the tissue cysts are known as cytozoites. These forms are thinner and less susceptible to peptic and tryptic destruction when compared to the tachyzoites. Tissue cysts develop within the cytoplasm of the host cell [9]. T. gondii multiplies intracellularly at the site of the invasion; bradizoites are released from the cysts or sporozoites are released from the oocyst they penetrate and multiply within the intestinal cellular epithelium [16], [17].

The nucleus of the cell remains outside the cyst and may degenerate after some time. The cysts grow and remain intracellular as the bradyzoites divide by endodiogeny. The cytoplasmic vacuole membrane becomes a capsule of the cyst. The tissue cyst wall is elastic and resistant, arginofilic, composed of parasite and host cell material [18]. The size of the cyst is variable and depends on the parasitized cell and the number of bradyzoites contained therein [19]. Younger cysts are small, 5µm in diameter and contain two bradyzoites in their interior, while older ones can reach 200µm containing hundreds or thousands of organisms. The most prevalent sites of cystic form of toxoplasma are skeletal and cardiac muscle tissues, nerve tissue and the retina. Finding cysts in visceral organs like lungs, liver and kidneys are rarer. Intact tissue cysts generally do not cause damage to the host and may persist throughout the life of the infected individual [18]. Oocysts are formed in the intestinal cells of felids and are eliminated as sporulated (immature), along with feces. They are spherical, measuring 12.5 to 11µm and sporulate in the environment where they contain two sporocysts, each with four sporozoites, which have a double wall that is very resistant to environmental conditions and resistant to the action of gastric juice [9] .

Toxoplasma has a two-phase life cycle: an asexual phase, in the tissues of several animal and man hosts, and a sexuada, in the intestinal epithelial cells of young nonimmune cats, which are the largest eliminators of oocysts. The host susceptible to ingesting oocysts, tissue cysts and tachyzoites becomes infected. The tachyzoites are destroyed in the stomach for the most part, but may penetrate the oral mucosa or be inhaled, evolving in the same way as other forms. After ingestion the proteolytic enzymes cause degradation of the oocyst and tissue cysts wall, resulting in the release of bradizoites and sporozoites that will invade the host cell [9].

After a rapid passage through the intestine, the sporozoites and bradizoites will become tachyzoites and will parasitize various cell types, occurring to the formation of cytoplasmic vacuoles containing parasites. In these vacuoles, the tachyzoites are protected from the immune system of the host, multiplying asexually and causing rupture of the infected cell, with the release of new tachyzoites that will invade other cells, with greater tropism by the central nervous system and retina, restarting the cycle . The spread of the parasite in the organism occurs through the blood and lymph, leading to the involvement of multiple organs [19].

The sexuated phase occurs in the young and nonimmune cat that acquires the infection through the ingestion of oocysts, tissue cysts and tachyzoites. The sexual cycle occurs in the intestinal cells with formation and fertilization of the gametophytes and formation of zygote, this originates the immature oocyst that is later released after cell disruption, being eliminated in the feces after one or two weeks of infection 19. The immature, non-infectious oocysts under ideal conditions of temperature, humidity and oxygen, such as those occurring in regions of tropical climate, sporulate and can survive for 12 to 18 months in the soil as long as conditions are maintained [9]. The transmission of oocysts can occur through contaminated water, fruits or vegetables containing oocysts on their surface or by contact with soil or sand as a practice of gardening [20].

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The only form of proven transmission of toxoplasmosis among humans to date is that which occurs through the passage of tachyzoites through the placenta [6]. The pregnant woman can transmit the disease to the fetus most often infected during pregnancy [9]. Toxoplasma can colonize the placenta that remains infected until the end of gestation. Thus, if the treatment is suspended, the placenta may behave like a reservoir, sending live microorganisms to the fetus throughout gestation [6].

Parasitemia characterizes the acute phase of the disease, when there is great cellular destruction and parasite proliferation. The severity of the disease will depend on factors such as the number of infective forms, the parasite strain and host susceptibility [9].

From the development of immunity, the extracellular parasites disappear from the blood and lymph, also occurring to decrease their multiplication in the intracellular environment. In this period there is the formation of tissue cysts in the host, characterizing the chronic form. Periodic rupture of these cysts may occur with cellular destruction and focal reactivation of the infection. However, with the exception of the retina, the parasites released into the tissues are rapidly destroyed by the immune system [10].

Cell destruction caused by parasites in the acute phase is more severe in the brain, eyes and muscles. The severity of the lesions depends on the ability of the tissue to replace the destroyed cells. In lymphatic, epithelial and connective tissues, in the liver and lungs no significant lesion is observed due to the greater recovery of these tissues. The inflammatory reaction consequent to these cell lesions is characterized by infiltration of lymphocytes, monocytes, macrophages and polymorphonuclear cells. Tissue repair is done by fibrosis and, in the brain, by glucose [9].

Due to the immaturity of the immune system, toxoplasma spreads to the fetus in various tissues, causing severe damage to the nervous system, including retina. In the cerebral cortex of an infant, the focal lesion may be so large that the calcified necrotic area is seen by the skull radiograph. Periaqueductal and periventricular necrosis reflects the great cerebral parasitism. Obstruction of the Sylvius aqueduct leads to dilation of the lateral ventricles and the third ventricle. Periventricular necrosis and necrosis due to infarction are characteristic of congenital toxoplasmosis [9].

In the eye, the parasite establishes a focus of infection that progresses from retinitis to a secondary choroid impairment. A conversion of the form tachyzoites to the bradyzoite encystoid appears apparently induced by the host immune system. The resolution of the lesion occurs with the control of the acute infection and the formation of scar tissue. The cyst may remain inactive in the healed lesion or adjacent to it for several years. During this period, there may be slow replication of the bradyzoite, which may result in rupture of the cyst wall, releasing the parasites in the adjacent retina and leading to recurrence of retinitis [21].

The reasons that lead to rupture of the cyst and reactivation of the disease are unknown, but immunosuppression may contribute to this phenomenon. It was thought that retinitis could occur due to a hypersensitivity reaction, but the occurrence of aggressive disease in immunocompromised hosts suggests that retinitis is a consequence of toxoplasma proliferation. Secondary lesions of vitreitis and uveitis may represent a hypersensitivity response [21].

In the retina, congenital infection may lead to the formation of white or unilateral necrotic focal retinocoroiditis areas located in the posterior pole, mainly in the macular region, which may be small or large and simple or multiple and with well defined limits between the areas involved by necrosis and without necrosis. The necrotic regions may exhibit dispersed melanocytic pigments derived from the retinal pigment epithelium. Adjacent active lesions and retinal cicatars may be observed, or in the area of necroses surrounded by retinal edema [21].

The intense inflammatory reaction of mononuclear nature consists of lymphocytes, macrophages and epithelioid cells with plasma cells grouped at the margins of the lesions. This reaction gives rise to the cell in the vitreous and exudation and, in the late stages, there is an increase in the glacial tissue that can invade the vitreous, leading to a vitreíte and subsequent formation of membranes [21].

The occurrence of retinal vasculitis may lead to local hemorrhage. Iridocyclitis is frequently seen in patients with toxoplasmic retinocoriditis. Another finding frequently associated with a previous granulomatous uveitis and, more rarely, panuveitis or optic neuritis may progress to optic atrophy. The cure of retinochoroiditis is associated with a decrease in the inflammatory reactions of the iris, ciliary body and vitreous [21].

Other ocular changes that can be found associated with retinocoriditis are microphthalmia, glaucoma, cystoid macular edema, cataracts, posterior synechia, retinal perivasculitis, chorioretinal vascular anastomosis, microcornea and retinal displacement. Strabismus and nystagmus may arise as a result of central retinocoriditis lesions [22].

The diagnosis of Congenital Toxoplasmosis can be made by means of the neonatal screening exam, already mentioned, by dosing with blood collected on special filter paper the fraction IgM, and in case of positivity

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confirm with IgG and IgM serological test, using the ELISA methods [20]. Also by polymerase chain reaction (PCR) in the aminiotic fluid, which can be performed from the 12th week of pregnancy, with few complications [23]. This examination though not available throughout the public health network is less risk to the fetus and if positive one can begin intrauterine treatment.

The newborn with suspected toxoplasmosis should undergo a complete physical examination, including a thorough neurological examination. Other exams such as transfontanel ultrasound, ophthalmologic examination and serological examination may be performed to complement the diagnostic investigation [24].

Prenatal treatment can be done with the use of antibiotics, and the use of spiramycin in the initial treatment of the pregnant woman with toxoplasmosis is recommended until the confirmation of the fetal infection as it is devoid of important side effects and well tolerated. When fetal infection is proven, the association of sulfadiazine with pyrimethamine and folinic acid is recommended from the second trimester of pregnancy, as spiramycin does not treat fetal infection. The treatment of the pregnant woman throughout the pregnancy should be continued with alternating or continuous therapeutic regimens. For prudence, it is recommended to substitute sulfadiazine for spiramycin or clindamycin at the end of gestation to avoid the possibility of kernicterus caused by sulfadiazine in the newborn [6].

Treatment of infected newborns should be performed even in the absence of clinical manifestations, since most infected newborns are asymptomatic. The therapeutic regimen should be started as early as possible and maintained for one year with the association of sulfadiazine, pyrimethamine and folinic acid [6].

The prognosis depends on the severity of the clinical picture and the treatment. Cases with apparent clinical manifestations have a worse prognosis, with high mortality (12%) and patients who can survive have sequelae such as mental retardation (85%), seizures (75%) and visual injury (50%) [6].

In patients who have subclinical and undiagnosed infection early, there may be late visual, auditory, or other neurological changes. In these cases, 85% will present episodes of chorioretinitis, reports of hypocausia occur in 10 to 30% of cases and psychomotor retardation in 20 to 75% of these children. Fetal infection is less when the mother is treated during pregnancy. Similarly, treatment during pregnancy may modify the severity of fetal infection [6].

Ocular lesions are predominant in Congenital Toxoplasmosis, even in subclinical forms, and may lead to late sequelae that are responsible for many cases of subnormal vision. Thus, in order to ensure adequate prevention of injuries, it is important to emphasize the importance of effective prenatal care for the early detection of acute infection in the pregnant woman and the institution of early treatment. At the same time there should be coverage in the fetus for fetal detection and also at birth through neonatal screening, as treatment will avoid or attenuate future repercussions.

Currently, the detection of Toxoplasma gondii infection, DNA / RNA analysis of the parasite in the aminiotic fluid through the PCR technique and new serological techniques of high sensitivity and specificity, such as IgA, IgM, IgE, and avidity of IgG.

However, despite the advances in diagnostic techniques used in suspected toxoplasmosis, the certainty of infection remains a major challenge, since infection may be present even in cases where placental examination was negative, IgM was not found and that IgG showed a transient decrease in their levels [6].

Several investigations have shown that after inoculation of SHIV virus in monkeys the presence of opportunistic pathogens such as: pneumocystis, cytomegalovirus, cryptosporidium, toxoplasma and candidiasis. Other researchers found a prevalence of toxoplasmosis in 120 HIV-positive patients with retinal lesions compared to Mycobacterium avium [25]. They also observed that reinocoroiditis lesions had scars with less pigment than those found in immunocompetent patients. When analyzed 33 HIV positive children found ocular toxoplasmosis as the most common manifestation [26].

Johnson et al [27] diagnosed acute toxoplasmosis encephalitis in 10 (20.4%) of the 49 autopsied patients, concluding that the large number of CNS toxoplasmosis cases in AIDS patients shows the need for the disease to be included in the differential diagnoses and seven (7) immunocompetent patients in the 69-82 age group with an average age of 74 years. Although treatment with antiparasitic drugs was instituted early in the disease, treatment had to be longer than normal and that 4 (four) patients had reactivation of retinitis after drug withdrawal. There was a visual drop in most cases. Silveira, Belfort Jr, Burnier Jr [28] concluded that toxoplasmosis in elderly patients is more severe due to the low cellular immunity of these patients, they reported a case of Fuchs, associated with toxoplasmic reinocoroiditis and Desmont coefficient, positive. They considered it highly probable that the etiology of Fuchs's syndrome in this case was toxoplasmosis due to the high level of antitoxoplasma antibodies found in the aqueous humor.

Moraes Jr [29] demonstrated that toxoplasmosis can be located in the outer layers of the retina in patients with AIDS, and not only in immunocompetent patients as

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previously demonstrated. Bosch-Driessen, Karimi, Stilma, Rothova [30] studied 150 patients with ocular toxoplasmosis who were examined between 1990 and 1997 and found 6% retinal displacement in patients with ocular toxoplasmosis. However, they concluded that a careful examination of the retina of patients with ocular toxoplasmosis should be done, especially those who have associated other risk factors such as myopia, and very severe intraocular inflammation.

The objective of the study is to analyze the incidence of Congenital Toxoplasmosis through the Elisa test in blood drops using the filter paper methodology in newborns in the Municipalities of the State of Rondônia, attended by the Neonatal Screening Program, in the period of 4 years.

II. MATERIALS AND METHODS

The present descriptive, quantitative character study will contribute to the knowledge of the incidence of Congenital Toxoplasmosis in newborns in the 4 year period in the State of Rondônia.

The material - The material used for the collection of blood in newborns was S & S 903 filter paper, which presents greater sensitivity in the analysis of the pathology by the Elisa method in the capture of IgM antibodies.

The collection of blood on filter paper - The collection was performed between 2 and 7 days after the first feeding, until the 30th day, and can be performed after this period, but classified as late collection, remembering that the earlier better collect the result of the work. In order to expedite the sending of blood samples on filter paper, postage-paid envelopes are available, which must be sent once or twice a week. These will be accompanied by a Listing, containing: RN / Mother's name, date of birth and date of collection of the Pezinho Test. When necessary, for any reasons (incorrect collection, improper handling of the collection card, insufficient material or Diagnostic Confirmation, etc.), a ricochet is requested through a document explaining the reason for the request.

The results of the analyzes - The results of the analyzes were issued individually, by computerized system, in an average term of fifteen days from the receipt, and immediately sent to the collection points from which they come. Any newborn suspected of being a carrier of one of the diseases will be recruited to perform the confirmatory tests. Suspected cases are confirmed by serum levels and, when positive, immediately reported to those responsible for the patient's location. Every live newborn identified / confirmed as having one of the pathologies has the right to adequate follow-up, guidance and treatment. The organization of the sample collection system for the National Neonatal Screening Program - PNTN requires special care in order to obtain desired results. All activities involved directly or indirectly are important, from the choice and training of the professional who will collect the sample transport system to the laboratory that will carry out the analyzes.

The collection point - The professional designated as responsible for the collection at each post is the person who will be activated by the Reference Service in neonatal screening whenever contact with the family becomes necessary. Generally, it is a nursing professional (nurse, nursing technician or nursing assistant), whose activity is regulated by specific legislation, and in the Collection Office has the responsibility to: guide the parents of the child regarding the procedure to take the child in a collection point of the network, in the case of the impossibility of performing the collection (early discharge) in the Hospital / Maternity; managing the storage and stock of filter paper, as well as request for replacement of material; administer the shipments collected to the Laboratory to which it is linked, as well as the receipt of results (Control of shipments sent / received); keeping records of the active search actions of those recruited: to locate the recalled children whose material has been returned for being inadequate, for requesting a new retake test, or for scheduling an appointment at the SRTN; administer and maintain record of delivery of normal or altered results to families; ensure the documentation and registration of information requested in Administrative Rule GM / MS nº. 822; file the proof of collection and delivery of results.

Collection environment - The collection room was a cozy place to quiet, suitable for the purpose. The use of refrigerated air is not recommended as cooling the baby's feet will make it difficult to absorb the blood. Before starting the collection, the professional should make sure that all necessary material, mentioned below, is available in the workbench that must be properly cleaned: procedure gloves (no surgical gloves are necessary); sterile disposable lancets with a triangular tip of approximately 2.0 mm; container (piss) with alcohol 70% for asepsis; cotton and / or small, sterile gauze; PNTN filter paper. In the stand, a small shelf or other device must be available to allow the distribution of already collected filter papers until the samples are completely dry.

Data recording on filter paper - All information requested on filter paper is important and necessary to achieve the desired results of the National Neonatal Screening Program. The collection card must fill out all the information. Incorrect, changed or illegible data delay or impede the examination, delay the diagnosis, harming the child. Only clear and well-readable information has

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allowed the rapid localization of children whose examination results have changed, requiring urgent medical attention. The activities at the Collection Station, although considered very simple, are of fundamental importance for the Neonatal Screening Program. The collection point is the gateway to the program. Your organization and the identifying information contained therein are critical and essential to the location of children who need special attention.

Methods - The technique for diagnosis in the Navelida of Congenital Toxoplasmosis is ELISA capture of IgM antibodies because it is of great sensitivity and specificity. The choice of techniques for confirming altered toxoplasmosis results that allow detecting IgG and IgM class antibodies are most useful. The presence of IgM antibody, and a significant increase of IgG in the newborn's recoleta evidenced a recent infection. In the recovery of newborns, the mother's serum is also collected, and the result is delivered to the mother to take to her doctor.

III. RESULTS AND DISCUSSION

The information presented was extracted from the information system database of the Neonatal Screening Program of the State of Rondônia at Natividade - Neonatal Screening Service.

Of the 52 municipalities that compose the State of Rondônia, in 32 municipalities notifications of the change of Congenital Toxoplasmosis occurred.

During the study period, the number of live births was 114,793. Of these, 102,963 newborns were screened with 90% coverage, and diagnosed with Congenital Toxoplasmosis, 126 children, who received treatment and follow-up.

The municipality of Porto Velho (Capital) was where there was a greater number of Congenital Toxoplasmosis with 33 cases, that is, 26.19%. In 9 municipalities there was 1 case of Congenital Toxoplasmosis. In the municipality of Ji-Paraná, 9 cases were identified (7.14%) and in Cacoal 8 cases diagnosed (6.35%). In the municipalities of Ariquemes, Guajará-Mirim, Ouro Preto D'Oeste and Vilhena, 6 cases (4.76%) were confirmed in each municipality.

In the State of Rondônia, 114,793 children were born in the 4-year period, according to information from the Sistema de Nacidos Vivos - SINASC - of the State Department of Health, and 102,963 newborns were screened. Of these, 126 newborns were diagnosed with Toxoplasmosis, in which a incidence of 1: 817 of live births.

A study cited by Vidotto [31], presents the results of serological surveys on canine and feline species carried out in Brazil. From the Public Health point of view, infection in the canine population means that the area involved represents an ecological niche for the parasite and, consequently, a risk for the human population.

Table.1: Triad Newborns and Change of Congenital Toxoplasmosis in the State of Rondônia by Municipality in the period of 4 years.

COUNTIES	*1º year RNT**	*TC**	*2º year RNT**	*TC**	*3º year RNT**	*TC**	*4º year RNT**	*TC**
Alta Floresta d'Oeste	549	1	497	0	537	0	530	1
Alto Alegre dos Parecis	86	0	209	0	207	0	215	0
Alto Paraíso	31	0	173	0	226	1	249	1
Alvorada d'Oeste	317	0	319	1	275	1	308	0
Ariquemes	1.428	2	1.393	1	1.571	2	1.483	1
Buritis	717	0	681	0	772	2	831	0
Cabixi	109	0	96	0	95	1	94	0
Cacaulândia	115	0	80	0	63	0	79	0
Cacoal	1.480	3	1.381	0	1.492	4	1.355	1
Campo Novo de Rondônia	114	0	110	0	113	1	126	0
Candeias do Jamari	245	0	310	0	326	0	292	0
Castanheiras	31	0	39	0	45	0	65	0
Cerejeiras	149	1	325	0	350	1	308	0
Chupinguaia	18	0	44	0	67	0	62	0
Colorado do Oeste	347	0	354	0	315	0	329	0
Corumbiara	0	0	9	0	0	0	24	0

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Costa Marques	202	1	219	1	198	0	288	0
Cujubim	139	0	126	0	146	1	147	0
Espigão d'Oeste	558	0	553	1	563	3	548	0
Governador Jorge Teixeira	128	0	125	0	153	0	150	0
Guajará-Mirim	525	3	650	1	716	1	764	1
Itapuã do Oeste	73	0	136	0	178	0	178	1
Jaru	1.184	1	1.170	3	1.147	1	1.187	0
Ji-Paraná	2.169	4	2.162	2	2.102	1	2.240	2
Machadinho d'Oeste	544	1	576	0	616	0	566	0
Ministro Andreazza	199	0	163	0	194	0	172	0
Mirante da Serra	203	0	249	0	211	0	221	0
Monte Negro	300	0	314	0	354	0	317	0
Nova Brasilândia d'Oeste	453	0	406	1	426	1	380	0
Nova Mamoré	208	0	285	0	264	1	356	1
Nova União	120	0	108	0	120	0	109	0
Novo Horizonte do Oeste	128	0	162	1	190	2	161	0
Ouro Preto do Oeste	943	1	846	4	780	1	740	0
Parecis	0	0	13	0	53	0	69	0
Pimenta Bueno	708	1	632	1	711	0	583	0
Pimenteiras do Oeste	22	0	57	1	38	0	27	0
Porto Velho	6.024	8	6.098	8	6.111	11	6.552	6
Presidente Médici	410	0	412	0	417	0	362	1
Primavera de Rondônia	60	0	86	0	64	0	68	0
Rio Crespo	28	0	80	0	46	0	41	0
Rolim de Moura	977	1	947	2	855	0	835	0
Santa Luzia d'Oeste	217	1	212	0	159	0	152	2
São Felipe d'Oeste	63	0	222	0	96	0	91	0
São Francisco do Guaporé	299	0	203	0	350	0	301	0
São Miguel do Guaporé	410	1	263	0	365	2	360	1
Seringueiras	227	0	240	0	259	0	256	0
Teixeirópolis	11	0	86	0	98	0	88	1
Theobroma	101	0	98	0	99	0	158	0
Urupá	235	2	203	0	256	0	200	1
Vale do Anari	0	0	0	0	0	0	61	0
Vale do Paraíso	105	0	121	0	102	1	130	0
Vilhena	1.202	2	1.203	1	1.224	1	1.283	2
TOTAL	**24.911**	**34**	**25.446**	**29**	**26.115**	**40**	**26.491**	**23**

Fonte: Natividа

Legend:RNT * = Triad Newborns. TC * = alteration by congenital toxoplasmosis

Table.2a - Prevalence of anti-Toxoplasma antibodies in dogs, registered in several serological surveys in Brazil.

DOGS				
State	**Serological test**	**Nºof animals**	**% Positive**	**Reference**
São Paulo	RIFI	276	46,1	Domingues et al (1996)
Goiás	SF	35	57,1	Fernandes & Barbosa (1972)
São Paulo	RIFI	47	63,8	Salata et al (1985)
Minas Gerais	RIFI	40	35,0	Silva et al (1997)
São Paulo	RIFI	80	94,0	Ishizuka et al (1974)

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Rio de Janeiro	SF	101	79,2	Coutinho (1968)*
Paraná	SF	66	51,5	Giovanoni (1958)*
Minas Gerais	HAI	218	52,7	Duran et al (1996)[8]
Amapá/Rondônia	HAI	19	68,4	Ferrarone & Marzochi (1978)*
Rio Grande do Sul	HAI	43	21,0	Chaplin & Silva (1984)*
São Paulo	RIFI	1256	63,8	Ishizuka & Yasuda (1981)*
Rio Grande do Sul	HAI	64	3,1	Chaplin et al (1980)*
São Paulo	SF	20	90,0	Sogorb et al (1976)
São Paulo	RIFI	657	91,0	Germano et al (1985)

Citado por Vidotto (1992)

Table. 2b - Prevalence of anti-Toxoplasma antibodies in cats, registered in several serological surveys in Brazil.

CATS				
Locality	**Serological test**	**Nºof animals**	**% Positive**	**Reference**
São Paulo	SF	130	50,8	Sogorb et al (1972)
Amapá/Rondônia	HAI	32	90,6	Ferrarone & Marzochi (1978)*
Rio Grande do Sul	HAI	100	24,0	Mendez (1983)*
Rio Grande do Sul	HAI	27	40,7	Chaplin & Silva (1984)*
São Paulo	RIFI	27	25,9	Rosa et al (1987)
São Paulo	RIFI	350	37,7	Camargo et al (1998)
São Paulo e Paraná	RIFI	191	19,4	Langoni et al (1998)

Cited by Vidotto (1992)

Because Natividа is the referral service of the aforementioned States, and the examination of IgM toxoplasmosis in all newborns was performed in the body of the neonatal screening exams, it facilitated this work. However, the difficulty in obtaining incidence on Congenital Toxoplasmosis in other States, makes difficult an extended discussion of the comparative data of Congenital Toxoplasmosis.

Currently, it is estimated that 40% of the world's population is infected with Toxoplasmosis. Brazil has an index that is among the highest, where registered serological surveys show a prevalence ranging from 37% to 91%. Citing research conducted by Domingues [32], between November 2001 and January 2002, Brazil recorded the largest outbreak of Toxoplasmosis in the world, occurred in the municipality of Santa Isabel do Ivaí - PR, a total of 462 people presented suggestive serology for toxoplasmosis (IgM - Reagent) seven were pregnant, of these, six had their children infected, a severe congenital anamolia and a spontaneous abortion occurred. Also comparative studies with other states that have already done a pilot plan to show the incidence of congenital toxoplasmosis in Brazil, we can mention the studies in Minas Gerais by the NUPAD (Neonatal Screening Reference Service in the State of Minas Gerais) that showed an incidence of 1 / 1,500 live births [33].

In the State of Mato Grosso do Sul, IPED - APAE-MS Research and Diagnosis Institute, which is a reference in Neonatal Screening in that State, has been conducting Screening for Congenital Toxoplasmosis since 2000, with an incidence of 1: 820.

The authors Maciel, Philocreon, Leite [34] present the result of the systematic investigation of toxoplasma in aborted concepts, nati and neomortos, from the Hospital of Charity São Pedro D'Alcântara in the city of Goiás-GO, during a period of 5 years. After a brief review of current knowledge on congenital toxoplasmosis, the authors report the finding of a case of congenital toxoplasmosis in 121 concepts.

Prevalence of congenital toxoplasmosis of 1 in 110 births, with only 50% of births resulting in births, was described in Goiânia. Using the numbers obtained in this study, it is possible to infer incidence of approximately 5 per 1000 live births in that city. A study conducted in Rio Grande do Sul found an incidence of 8 cases for 10,000 live births. Mathematical model, developed in the city of São Paulo, shows incidence of congenital toxoplasmosis from 0.8 to 1000 births, which would mean 280 new cases per year, in that city [35].

In the city of Ribeirão Preto, a study was carried out at the Hospital das Clínicas of the Faculty of Medicine (HCFMRP) USP, where they found an incidence of 1/723 live births [36]. This shows that in all of Brazil, because it

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is a tropical country, where the toxoplasmosis cyst finds a favorable environment, the toxoplasmosis index must follow the same proportion in all states. The number of confirmed cases of newborns due to toxoplasmosis in the two states concerned is a cause for concern, as it confirms the inefficacy of prenatal care and also the prevention of infectious diseases in these states, taking into account that Toxoplasmosis is a disease in the majority of cases, asymptomatic and can be avoided with a good elucidation to all the pregnant women in their first consultation, as well as the serology test for toxoplasmosis, both in the 1st trimester and in the third trimester.

It was observed that the incidences found in the other States of Brazil that have done research on Congenital Toxoplasmosis are equivalent to the results of the survey carried out at the Neonatal Screening Reference Service, Natividа - Neonatal Screening Unit, which is the incidence in the State of Acre 1: 638 and Rondônia 1: 861.

In France and Austria the incidence of congenital toxoplasmosis is 3-4 cases per 100 births, and in the United Kingdom 91 cases were reported between 1975 and 1980 [32]. Malm et al. [37] performed a prospective study to define the incidence of congenital toxoplasmosis in Sweden, where blood samples were collected on filter paper of 40,978 newborns, which were analyzed for IgM and IgG anti-toxoplasma. A preliminary report showed 3 children with congenital toxoplasmosis, defined by the detection of IgM antibodies. Two children were asymptomatic at birth. They had normal development until 12 and 15 months of life, respectively. The third child had confirmed congenital toxoplasmosis and treatment was instituted. The child had microphthalmia and peripheral retinochoroiditis in one eye. Despite the medications, he developed hydrocephalus, requiring neurosurgery at 3 months of age. Its development up to 14 months was normal. The incidence of congenital toxoplasmosis in Sweden, detected by the specific IgM in blood on the filter paper is less than 1: 10000.

Comparing the incidence with other countries, such as those mentioned above, it is observed that the incidence of Congenital Toxoplasmosis is lower than that registered in Brazil, as it can be said that in developing countries the incidence is higher than those developed. This may be because prenatal prevention is effective in these countries. Neonatal screening in Brazil took a major step forward after the Brazilian Ministry of Health included as a public health program. In establishing the guidelines for conducting the program in the state and municipalities and for the referral services that are selected by the state health secretariats, being inspected and empowered by the Ministry of Health. As a result, complicity was created among those involved in the consequently, its improvement in the evolution of the children screened, increasing the coverage of live births, and thus, there was an increase in the incidence of the triaged diseases, including congenital toxoplasmosis. However, in the Amazon, although coverage in Rondônia is 94% of live births, and in Acre 62%, the difficulties are greater than in the other states of the federation due to the geographical location of the Amazon, where we have a large riverine population, and also a a very extensive rural area, and lack of infrastructure on the roads connecting municipalities. However, coverage in these states has increased significantly with the creation of the neonatal screening program because it not only performs the examinations but also provides care for children who present some congenital alteration through their multidisciplinary team.

IV. CONCLUSIONS

The study shows that in the State of Rondônia the incidence of Congenital Toxoplasmosis is 1: 817. It is possible that the warm and humid climate of the Amazon region is conducive to the reproduction of the oocyst and with that the contamination becomes greater in this region.

Due to the cat being the natural host of toxoplasmosis and contaminating the environment with the elimination of oocysts, which is the resistance form of toxoplasmosis, and that in hot and humid climates it resists up to five days after its elimination the proliferation of toxoplasmosis by felines high rates, according to Vidotto's research [31].

The only form of transmission of human toxoplasmosis is vertical and depending on the gestational period the sequels may be larger or smaller. The importance of inclusion in the neonatal screening of this pathology is due to the earlier the disease is diagnosed in the newborn and consequently the treatment is started the smaller the complications for the neonatal.

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