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Cord blood screening for congenital infectious diseases and haematological change in Sulaimani Provence

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Abstract

Aims: to determine the seroprevalence of immunoglobulin A (IgA) and immunoglobulin G (IgG) anti-*Toxoplasma*, anti- *Helicobacter pylori* antibodies in cord blood serum and to access the usefulness of haematological parameters in diagnosing of toxoplasmosis and *H. pylori* infection. *Toxoplasma gondii* specific IgA, IgG and *H. pylori* specific IgG antibodies were assessed by Chorus enzyme immune assay. 19 out of 70 (27.1%) cord blood serum samples were found positive for anti-*Toxoplasma gondii* IgG antibody and there was only one (1.4%) positive for IgA. Regarding the detection of specific anti -*H. pylori* IgG, 30 cord blood samples were tested in which 26 (86.6 %) were found positive and four (13.4%) negative. The positive mean with *H. pylori* infection was significantly greater than *H. pylori*-negative mean (88.37 ± 53.69 and 5.8 ± 0.46, respectively, P = 0.005). The comparison of hematological profiles between positive and negative cord blood samples (IgG ≥ 8 and IgG < 4; IgA >1.2 and IgA < 0.8) showed no statistically significant variations in higher and lower value of IgG and IgA titration (P > 0.05).
Conclusions: The Results revealed that all the cord blood serum samples were negative for IgA antibodies except one sample (1.4%), indicate that all these newborn infants were rarely infected with congenital toxoplasmosis. Neonates born from *H. pylori*–infected mother, are provided with a high amount of specific IgG *H. Pylori* antibodies, which are transferred transplacentally. The complet blood picture (CBC) test shows the non-significant effect of *T. gonidii* and *H. pylori* on most of the hematological parameters.

Introduction

Maternal infection during pregnancy still embodies an elusive zone in teratology. It is known that many infections can be transferred from mother to fetus across the placenta during fetal development, and umbilical cord of the newborn infant is a particularly common portal of entry for systemic infection. Certain pathogens including *T. gondii* that causes Toxoplasmosis can have adverse consequences for the fetus if they cross the placenta (e.g. miscarriage, stillbirth or severe complication for baby) [1]. It is among the most prevalent of human infections that affects an estimated to be 30–50% of the world population [2]. Congenital toxoplasmosis (CT) occurs only when a woman becomes infected with *T. gondii* during pregnancy and it can be asymptomatic or can have more sever maternal and neonatal consequences [3]. The risk of mother to child transmission of the parasite increases steeply with gestational age (GA) at maternal infection, with a probability of 10–25% in the first trimester of pregnancy, 30–45% in the second trimester, 60–65% in the third trimester, and up to 80% before childbirth [4]. Infections in the pregnant women or infants born

with congenital toxoplasma can be diagnosed by serological screening for toxoplasma antibodies [5]; if synthesis of specific anti-Toxoplasma antibodies (IgA, IgM, and/or IgG) was proven at birth or during the first year of life, and/ or if specific antibodies were still present after the age of 12 months [6]. The results of previous studies suggest that the mothers play a crucial role in transmitting *H. pylori* infection to their children [7] and [8]. *H. pylori* were recognized as the primary etiological agent in a variety of gastroduodenal disorders, including chronic gastritis and peptic ulcer disease. The route of *H. pylori* transmission is not completely clarified.This microorganism commonly colonizes the stomach [9]. Previously intrafamilial clustering has been documented [10] and [11]. In intrafamilial *H. pylori* infections, the infected parents, mainly the infected mothers, usually have been considered the possible source of transmission [12]. Since then, several reasonable automated hematology analyzers have recently been developed for in-clinic usage. As complete blood count (CBC) is a standard hematological test that mostly used to inspect information from erythrocytes, leukocytes, and platelets indicating infections and disorders in the body. Several studies have demonstrated the *H. pylori* infection and Toxoplasmosis can alter haematological parameter [13] and [14]. The objective of the current study was to determine the seroprevalence of IgA and IgG anti-*Toxoplasma,*anti-*H.pylori* antibodies in cord blood serum and to access the usefulness of haematological parameters in diagnosing of toxoplasmosis and *H. pylori* infection.

Materials and Methods

Ethics statement

This study approved by the ethics committee of the Maternity Hospital of Sulaimani. Informed consent was obtained from each woman and personal data regarding demographic characteristics.

Collection of samples

Between February and April2015 cord blood samples of 70 women were collected at birth in the obstetric units of Maternity Hospital of Sulaimani. After delivery, blood samples were collected from cord vessels representing the existence of *T. gondii* and *H. pylori* antibodies in baby's circulation. Serum samples were obtained by centrifugation at 2000 rpm for 10min of clotted blood, stored under -20 °C until utilized.

T.gondii and *H. pylori* antibodies in serum.

Anti-*T. gondii* IgG, IgA and anti- *H. pylori* IgG were detected by a Chorus enzyme immune assay (Chorus Elisa system, Disease Diag., Siena, Italy), which had been previously validated for Iranian,Turkish and Korean populations [15] , [16] and [17]. The assays were performed according to manufacturer's instructions. The cut-off for both *Toxoplasma* and *H. pylori*- specific IgG test positive corresponds to ≥ 8 IU/ml, equivocal corresponds to ≥4 to <8 IU/ml and negative corresponds to <4 IU/ml. Anti-Toxoplasma-specific IgA was considered positive when the index was > 1.2 IU/ml, the result was equivocal when index was from 0.8-1.2 IU/ml and the test was regarded negative if the index was <0.8 IU/ml, according to the manufacturer's instructions. Equivocal samples were retested.

Haematological values

At birth 2 ml of cord blood was collected in EDTA tubes from very recently labored women in Maternity Hospital of Sulaimani City and CBC tests performed by Orphée Mythic 18 hematology analyzer (SA, Switzerland) calibrated daily with standards provided by the manufacturer. It analyzes 18 parameters, including leukocyte, and erythrocyte and thrombocyte count.

Statistical analysis

Statistical significance for differences between categorical data calculated by pairwise two-tailed t-test and chi-square (x^2) using Graph Pad Prism 6 (Software MacKiev, USA). Statistical significance was considered at $P<0.05$.

Results

During delivery, 70 cord blood samples were collected from the participating women between February and April, 2015 at birth in the obstetric units of Maternity Hospital of Sulaimani (Figure: 1). The

pregnant women's mean age was 29±5.6 years old (range 16 to 40 years old). Distribution by age was as follows: 4.28% pregnant adolescents (16-18 years old), 80% pregnant adults (19-35 years old) and 15.71% older pregnant females (>35 years old).

The frequency of specific anti-*T. gondii* IgG and IgA antibodies in cord blood samples.

Nineteen out of 70 (27.1%) cord blood serum samples were found positive for anti-*T. gondii* IgG antibody and there was only one (1.4%) positive for IgA. There was a significant positive correlation between IgG and IgA amongst the cord blood samples (P=0.0001). No statistically significant difference was detected amongst different maternal age groups (P<0.05), the highest infection rate in observed in age groups 21-25 years old (Figure 2).

The frequency of specific anti-*H.pylori* antibodies in cord blood samples.

Thirty cord blood samples were investigated. Maternal-specific anti-*H. pylori* IgG were detected in the cord-blood samples of 26 (86.6 %) newborns (Figure: 3).The positive mean with *H. pylori* infection was significantly greater than *H. pylori*-negative mean (88.37 ± 53.69 and 5.8 ± 0.46, respectively, P = 0.005)

Cord blood haematology

The comparison of haematological measurements between two groups of cord blood samples (IgG ≥ 8 and IgG<4; IgA>1.2 and IgA< 0.8) showed no statistically significant variations in higher and lower value of IgG and IgA titration(P > 0.05).

Discussion

To the best of our knowledge, this is the first study in Sulaimani city to report the detections of anti-toxoplasma IgA and anti-*H. pylori* IgG in cord blood sera. Most congenitally infected newborn infants do not show any clinical symptoms of infection at birth.Diagnosis of congenital infections in newborns is, therefore, relying on biological investigations. In the present study, we evaluated the *T. gondii* (IgG and IgA) and *H. pylori* IgG antibody from cord blood serum using a commercial chorus enzyme immuno assay test kit. Seropositivity for IgG anti-*T. gondii* antibodies were observed in 27.1% (19/70) cord blood serum. For IgA assay, only 1.4% (1/70) cord blood serum present specific anti-IgA positive, the presence of anti-*T. gondii* IgG alone suggests a chronic infection in congenital infection in babies. IgG present in cord blood samples indicates maternal exposure to toxoplasmosis during her lifetime. Moreover, the presence of IgA in cord blood reflects CT. In comparison to previous studies, the prevalence IgG found in this study was lower than the prevalence found in 1998 in Bogotá (43%) and that reported in Bosa (45%) [18]. On the other hand, our results agree with previously reported in a study conducted in Bogota, Colombia (28.2%) [19]. In 1999, Robert-Gangneux and his colleagues used immunofluorescence assay and ELISA to determine specific IgG in cord blood sera. The serological tests identified specific IgG in 54 of 57 uninfected newborns and 18 of 20 infected newborns with CT [20]. An Iraqi study performed by Al-haris *et al*. showed that 105 neonates were positive for IgG (35%) and only one neonate with a positive IgM (0.33%) [21]. Detection of specific anti-*Toxoplasma* IgG and IgM antibodies in cord blood is not quite satisfactory for early diagnosis of CT. Many studies have been published on this topic. Most of these studies confirmed the superiority of IgA testing compared to IgM testing [22] and [23]. In a similar study conducted in Lyon, France, on 41 congenital toxoplasmosis infants, 38 had IgA positive reaction at the 1: 20 dilution, 30 had IgA positive reaction at the 1:100 dilution. Among the 155 infants without congenital toxoplasmosis, nine had IgA at the 1:20 dilution and two had IgA at the 1:100 dilutions [24].Wallon *et al*. identified five infants with Toxoplasma-specific IgA but not IgM in a group of 89 Toxoplasma-infected infants [25]. Stepick-Biekand his collaborators observed that anti-Toxoplasma IgA antibodies were demonstrable in 8 of 9 infants/fetuses with CT. In certain, IgM antibodies could not be demonstrated [26]. In studies which have evaluated the two assays (IgM and IgA) in parallel, IgA assay has been considered to be the more sensitive [27], [23] and [26].
For the detection of serum anti-*H. pylori* IgG, we routinely take thirty cord blood. According to our results out of 30 cord blood samples 26 (86.6%) were positive, and 4 (13.4%) were negative. Ashorn *et al*. (1996) reported that anti-*H. pylori* IgG were detected in the cord blood samples of 21 children (10-6%) [28]. Kuo *et al*.(2014) have reported similar results, who showed that only 89 (84.7%) out of 105 *H.pylori*-infected

mothers had positive cord serum antibody detection from their babies [29]. It is well-known that specific anti-*H. pylori* IgG antibodies are transplacentally transported from mothers to fetuses [30] and a close relationship between maternal and cord specific IgG levels were established [31] and [32].

The CBC test shows the non-significant effect of *T. gonidii* on most of the hematological parameters. However, no significant up and down in both monocytes and PCT% parameters have found. These results agree with the tests made on cats by [14]. They also found that most of the blood parameters have not significantly changed except the PCV, RBC and monocyte that were significantly high in cats with IgM ≥1/64. During the research, we found that there is no significant relation between CBC and *H. pylori* infection. As in [13] results, they observed that *H. pylori* do not affect haemoglobin (Hb) and MCV significantly. In contrast to our study Zuberi *et al*. (2007) noticed the significantly low level of haemoglobin in male and non-pregnant female patients with *H. pylori*. [33], also found significantly low levels of Hb, ferritin and vitamin B12 in patients with *H. pylori* infection. Moreover, our result may be due to oral supplementation of Folic acid and Iron during pregnancy period.

Conclusions

In summary, this study revealed that the most of the cord blood serum samples exhibited negative IgA level which indicated that there is infrequent acute infection with *T. gondii*, whereas anti-*T. gondii* IgG was found in 27.1% serum samples of neonates born could be of maternal origin. We also conclude that neonates born from *H. pylori*–infected mother are provided with a high quantity of specific IgG *H. Pylori* antibodies, which are transported transplacentally. Regarding haematological parameters, all the values were within the normal limits and, therefore, we believed that unhelpful in this situation.

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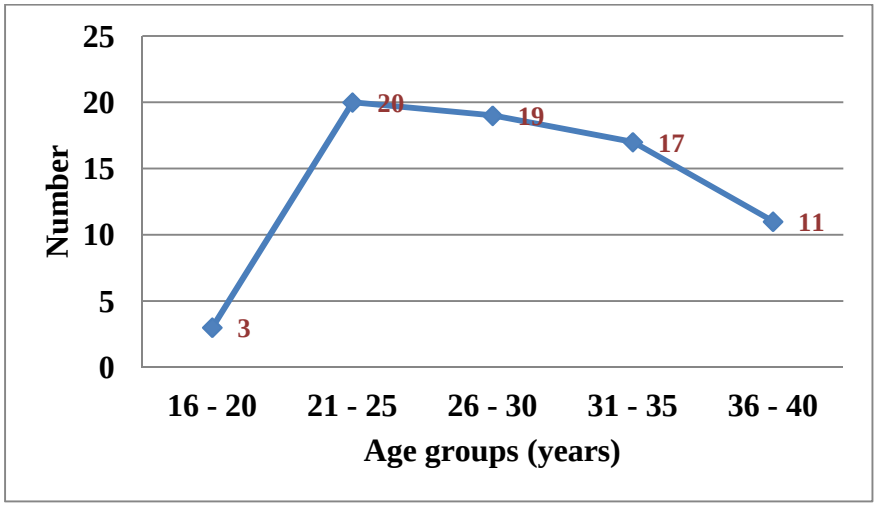

Figure 1:- Number of participants in each age group

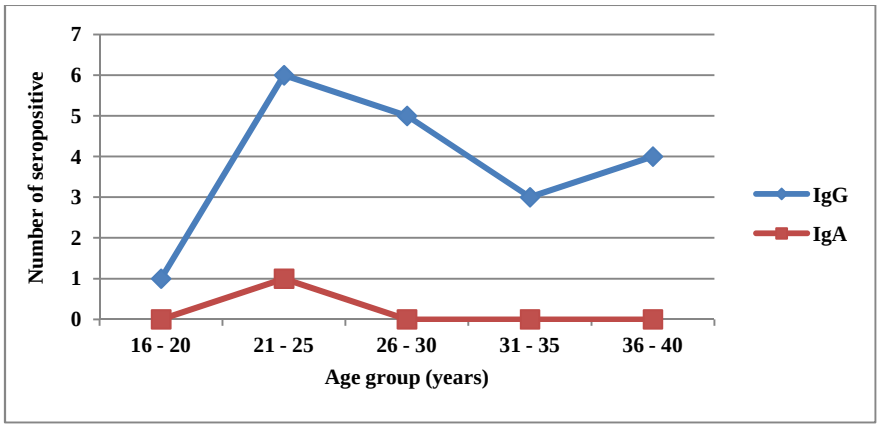

Figure 2: Frequency of anti-T. gondii IgG and IgA antibodies in cord blood by maternal age group.

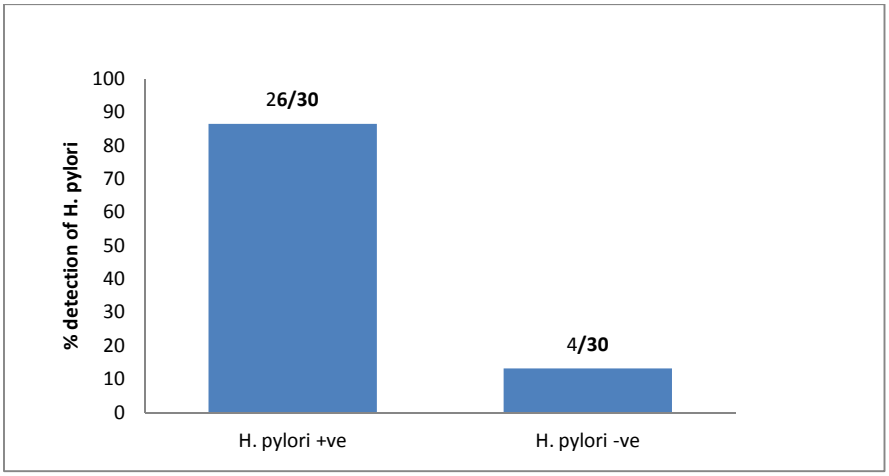

Figure 3: Percentage of cord blood samples tested positive for IgG. The number above each bar indicates the number of positive samples/number of tested samples.

Table 1: Statistical data and serological results of the two groups of the 30cord blood based on IgG titration (IgG**≥ 8**,n=26; IgG**<4, n**=4, **P > 0.05**)

		H. pylori IgG (IU/ml)	WBC 103/ml	Lym 10^3/ml	Mon 10^3/ml	Gran 10^3/ml	Lym%	Mon %	Gran %	RBC 10^6/ml	HGB g/dl	HCT %	MCV µm³	MCH pg	MCH C g/dl	RDW %	PLT 10^3/µl	MPV µm³	PCT %	PDW %
≥ 8	Mean	88.37	11.24	4.05	1.38	5.83	36.52	12.37	51.09	4.24	14.15	44.94	106.06	33.41	31.52	15.27	218.31	7.60	0.16	12.07
	SD	53.69	3.57	1.25	0.43	2.42	6.91	3.0	8.15	0.83	2.70	8.89	7.34	2.51	1.57	3.78	93.78	0.56	0.06	2.64
	SEM	10.53	0.76	0.26	0.09	0.51	1.47	0.63	1.73	0.17	0.57	1.89	1.56	0.53	0.33	0.8	19.99	0.12	0.01	0.56
<4	Mean	5.8	13.52	4.97	1.7	6.82	36.87	12.52	50.6	4.14	13.9	44.75	107.92	33.57	31.15	13.45	240.5	7.67	0.18	13.55
	SD	0.46	2.06	3.57	2.24	0.37	2.33	11.7	2.4	13.67	0.08	0.29	2.41	5.07	0.34	1.22	2.28	0.853	0.032	1.44
	SEM	0.09	1.03	0.76	1.12	0.18	1.16	5.85	1.2	6.83	0.04	0.14	1.2	2.53	0.17	0.61	1.14	0.42	0.01	0.72

WBC: White blood cells; Lym: Lymphocytes; Mon: Monocytes; Gran:Granulocytes; RBC: Red blood cell; HGB: Hemoglobin; HCT: Hematocrit; MCV: Mean corpuscular volume; MCH: Mean corpuscular hemoglobin; RDW: Red cell distribution width; PLT: Platelets; MPV: Mean platelet volume; PCT: Platelet hematocrit; PDW: Platelet Distribution Width.

Table 2: Statistical data and serological results of the two groups of the 70cord blood based on IgG titration (IgG≥ 8,n=19; IgG<4, n=51, P > 0.05).

		T.gondii IgG (IU/ml)	WBC 103/ml	Lym 10^3/ml	Mon 10^3/ml	Gran 10^3/ml	Lym%	Mon %	Gran %	RBC 10^6/ml	HGB g/dl	HCT %	MCV µm³	MCH pg	MCHC g/dl	RDW %	PLT 10^3/µl	MPV µm³	PCT %	PDW %
≥ 8	Mean	69.75	12.26	4.39	1.45	6.5	37.05	12.16	50.78	4.37	14.59	45.68	96.83	33.48	32.01	15.46	259.25	7.44	0.19	11.94
	SD	49.46	4.02	0.97	0.34	3.06	6.40	3.43	7.98	0.51	1.44	5.35	27.96	2.23	1.46	4.28	51.9	0.48	0.04	1.84
	SEM	14.28	1.11	0.27	0.09	0.84	1.78	0.95	2.21	0.14	0.4	1.48	7.75	0.61	0.4	1.18	14.39	0.13	0.01	0.51
<4	Mean	0.07	11.51	3.95	1.37	6.17	35.09	11.9	52.92	4.23	13.99	44.78	105.9	33.09	31.27	14.52	206.11	7.66	0.15	12.35
	SD	0.03	3.96	1.37	0.48	2.67	8.17	2.19	9.23	0.9	3.02	9.88	7.04	2.25	1.45	3.15	99.1	0.51	0.07	2.87
	SEM	0.009	0.96	0.33	0.11	0.64	1.98	0.53	2.23	0.21	0.73	2.39	1.7	0.54	0.35	0.76	24.03	0.12	0.01	0.69

WBC: White blood cells; Lym: Lymphocytes; Mon: Monocytes; Gran:Granulocytes; RBC: Red blood cell; HGB: Hemoglobin; HCT: Hematocrit; MCV: Mean corpuscular volume; MCH: Mean corpuscular hemoglobin; RDW: Red cell distribution width; PLT: Platelets; MPV: Mean platelet volume; PCT: Platelet hematocrit; PDW: Platelet Distribution Width.