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Platelia-Toxo IgA, a New Kit for Early Diagnosis of Congenital Toxoplasmosis by Detection of Anti-P30 Immunoglobulin A Antibodies

A. DECOSTER,[1,2]* B. SLIZEWICZ,[3] J. SIMON,[3] C. BAZIN,[2] F. DARCY,[1] G. VITTU,[2] C. BOULANGER,[4] Y. CHAMPEAU,[5] J. L. DEMORY,[2] M. DUHAMEL,[2] AND A. CAPRON[1]

Centre d'Immunologie et de Biologie Parasitaire, Unité Mixte INSERM U 167-CNRS 624, Institut Pasteur, 1 rue du Professeur Calmette, BP 245,[1] and Hôpital Pédiatrique St. Antoine du Centre Hospitalier Feron-Vrau, 329 Boulevard Victor Hugo, BP 255,[2] 59019 Lille, and Diagnostics Pasteur, 92430 Marnes la Coquette,[3] Service de Néonatalogie, Centre Hospitalier, 59056 Roubaix,[4] and Laboratoire de Biologie Médicale, Centre Hospitalier, 50303 Avranches,[5] France

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With the aim of achieving earlier diagnosis of congenital toxoplasmosis, anti-P30 immunoglobulin A (IgA) antibodies were assayed by using a Platelia-Toxo IgA kit with samples from 72 children born to mothers who seroconverted during pregnancy. A total of 148 serum samples and 1 cerebrospinal fluid sample were from 23 congenitally infected children (2 serum samples were collected from fetuses), and 74 serum samples were from 49 uninfected children. Among the 23 infected children, anti-P30 IgA antibodies were present in all infants either at birth or in the following weeks, whereas anti-P30 IgM antibodies were present in 13 from the 23 infected children either at birth or in the following weeks. Serum samples collected in utero from two infected children were also tested. One of these samples was positive for both anti-P30 IgA and anti-P30 IgM antibodies, whereas both children were negative at birth for these antibodies. Neither anti-P30 IgA nor anti-P30 IgM antibodies were detected in 47 of 49 uninfected children. These results suggest that detection of anti-P30 IgA antibodies by the Platelia-Toxo IgA kit is a very effective method for early diagnosis of congenital toxoplasma infection.

Conventional tests for antibodies to toxoplasma are not immediately helpful for serodiagnosis of congenital infection because children's immunoglobulin G (IgG) toxoplasma antibodies cannot be distinguished from maternal IgG antibodies acquired across the placenta during intrauterine life. The maternally transmitted IgG antibodies persist for some months in the child. A specific immune response by the infant which indicates infection is marked by the presence of antibodies which do not cross the placenta, such as IgM or IgA antibodies (4, 5, 8, 10, 12, 13, 14). We have previously shown that detection of IgA antibodies directed against P30, a major *Toxoplasma gondii* membrane protein, was of interest to early diagnosis of a congenital infection in children whose mothers had seroconverted during pregnancy (3).

We have now evaluated Platelia-Toxo IgA, a new immunocapture assay aimed at detecting anti-P30 IgA antibodies and developed by Diagnostics Pasteur (Marnes-la-Coquette, France).

MATERIALS AND METHODS

Patients and samples. A total of 222 serum samples and 1 cerebrospinal fluid sample were assessed. They were collected at birth and in the months following birth from 72 children whose mothers were infected by toxoplasma during pregnancy. No mother had clinical manifestations, and all mothers were treated with spiramycin until delivery as soon as their seroconversion was diagnosed.

All of the children are at present more than 6 months old and are considered congenitally infected or uninfected on the basis of clinical and serological follow-ups. In particular, a rise in specific antibodies was observed after the sixth month of life in all infected children, after disappearance of the passively transmitted maternal antibodies, whereas uninfected children were seronegative at this time. Among the infected children, 19 did not show clinical signs at follow-up. In four children, a peripheral chorioretinitis was diagnosed at birth (Table 1, cases 3 and 5) or developed secondarily at 4 months (Table 1, case 14) and 9 months (Table 1, case 18) of life. Except in three cases (Table 1, cases 14, 18, and 19), infected children had been treated with alternating courses of pyrimethamine sulfadiazine, and folinic acid (four 3-week treatments in the first year of life) and of spiramycin (2). In one case (Table 1, case 14), the child had received only half the dose because of anemia; in case 18, the child had received spiramycin alone, and in case 19, the child had received only one combined dose of pyrimethamine and sulfadoxine and then a dose of spiramycin alone.

All the sera used for this study were first characterized by an agglutination test with and without β-mercaptoethanol (BioMérieux, Lyon, France), by a direct IgG enzyme-linked immunosorbent assay (Diagnostics Pasteur), and by a double-sandwich enzyme-linked immunosorbent assay for detection of anti-P30 IgM antibodies (Platelia-Toxo IgM; Diagnostics Pasteur). IgM and IgA tests were run concurrently from the same serum dilution.

Immunocapture assay for the detection of anti-P30 IgA: Platelia-Toxo IgA. The immunocapture assay for the detection of specific anti-P30 IgA antibodies was carried out as follows.

(i) Toxoplasma antigens. The antigen was obtained from cultures of human larynx carcinoma cells (Hep2) infected with the virulent RH strain of *T. gondii*. Parasites were harvested at day 5 after infection, and the membrane extract was prepared as previously described (1, 11).

* Corresponding author.

TABLE 1. Detection of specific anti-P30 IgM and IgA antibodies in 23 children congenitally infected with *T. gondii*[a]

Case no.	Gestational term of infection	Age	IgG (IU/ml)	IgA		IgM (FI)
				FI	OD	
1	2	Birth	400	2.2	0.92	0
0		1 mo	1,000	0	0.41	0
		2 mo	1,000	0	0.32	0
		3 mo	1,000	0	0.40	0
2	1	Birth	800	>27.4	>3	0
		1 mo	400	>20.9	>3	0
		5 mo	400	15.5	2.25	0
		7 mo	400	5.7	1.27	0
		9 mo	300	1.1	0.81	0
		13 mo	300	0	0.35	0
		16 mo	250	0	0.29	0
		19 mo	250	0	0.34	0
		23 mo	500	11.8	1.88	0
		28 mo	250	0	0.31	0
3	2	Birth	200	0.9	0.71	0
		3 wk	100	0	0.45	0
		6 mo	800	0	0.35	0
		10 mo	800	0	0.33	0
		13 mo	800	0	0.36	0
		16 mo	400	0	0.41	0
		22 mo	3,000	9.7	1.67	0
4	3	Birth	0	11.4	1.84	19
		1 wk	100	>22.8	>3	17.6
		2 wk	100	>22.8	>3	24
		3 wk	100	>22.8	>3	17.3
		4 wk	100	>22.8	>3	16.6
		5 wk	100	>22.8	>3	16.2
		6 wk	100	11.6	1.86	1
		2 mo	100	>22.8	>3	0.5
		4 mo	200	6.5	1.35	0
		5 mo	200	0	0.48	0
5	2	Birth	100	10.6	1.76	0
		2 wk	100	6.5	1.35	0
		3 wk	100	0	0.42	0
		4 wk	100	0	0.42	0
		5 wk	100	0	0.36	0
		8 wk	50	0	0.41	0
		9 wk	25	0	0.27	0
		10 wk	25	0	0.30	0
		11 wk	25	0	0.35	0
		12 wk	25	0	0.43	0
6	3	Birth	0	0	0.28	0
		1 wk	25	7.3	1.43	10.6
7	3	Birth	0	2.3	0.93	7.6
		2 wk	10	>22.8	>3	3.6
		3 wk	10	18.4	2.54	0
		4 mo	25	0.0	0.7	0
		7 mo	25	0.7	0.77	0
		9 mo	25	0	0.41	0
8	3	Birth	400	>22.8	>3	22.1
		2 mo	400	>22.8	>3	0
9	1	Birth	400	ND		ND
		1 mo	200	>27.4	>3	0
		2 mo	200	>27.8	>3	0
		6 mo	1,600	11	1.8	1
		8 mo	2,240	9.3	1.63	0
		9 mo	3,254	5.8	1.28	0
		10 mo	5,280	<20.9	>3	13.4
		12 mo	2,000	0.9	0.79	0
		15 mo	400	0.3	0.73	0
		18 mo	400	0	0.42	0
		24 mo	4,800	3.4	1.04	0
		26 mo	4,800	4	1.1	0
		28 mo	4,800	6	1.3	0
		30 mo	2,400	0	0.43	0
		33 mo	1,000	0	0.41	0
		39 mo	500	0	0.38	0
		42 mo	500	0	0.42	0
		45 mo	200	0	0.35	0
		46 mo	200	0	0.29	0
10	3	Birth	100	>22.8	>3	23.9
		4 mo	1,300	3	1	3.3
		CSF	0	12.2	1.92	0
11	1	In utero	50	0	0.29	0
		Birth	400	0	0.37	0
		2 mo	1,000	17.5	2.45	0
		3 mo	1,000	5.5	1.25	0
		4 mo	1,000	0	0.36	0
		5 mo	1,000	0	0.42	0
		8 mo	800	0	0.32	0
		9 mo	500	0	0.32	0
12	1	Birth	3,200	11.3	1.83	0
		1 wk	1,600	>20.9	>3	0
		2 mo	400	0	0.35	0
		5 mo	200	0	0.29	0
		8 mo	100	0	0.33	0
		10 mo	200	0	0.36	0
		12 mo	200	0	0.30	0
13	2	Birth	1,600	ND		ND
		2 mo	800	>27.4	>3	0
		4 mo	400	>16.8	>3	0
		5 mo	200	>20.9	>3	0
		9 mo	200	8.6	1.56	0
		13 mo	200	0	0.36	0
		24 mo	3,200	>20.9	>3	0
14	3	Birth	400	>20.9	>3	>29.1
		1 wk	400	>20.9	>3	>29.1
		2 wk	800	>20.9	>3	22.6
		3 wk	800	19.2	2.62	12.8
		4 wk	800	>27.4	>3	12.4
		6 wk	800	>21.5	3	3
		2 mo	800	0.5	0.75	0
		4 mo	1,200	>27.4	>3	0
		6 mo	1,200	>21.5	>3	0
		9 mo	800	12.2	1.92	0
		11 mo	1,200	1.5	0.85	0
		19 mo	2,000	0	0.51	0
		22 mo	1,000	0	0.42	0
		25 mo	500	0	0.36	0
		32 mo	2,000	0	0.43	0
15	3	Birth	1,600	>20.9	>3	5.6
		1 wk	1,600	>20.9	>3	2.4
		8 mo	400	0	0.42	0
		12 mo	100	0	0.36	0
		20 mo	400	4.2	1.12	0
16	1	In utero	50	0.6	0.76	1.2
		Birth	800	0	0.41	0
		1 mo	400	0	0.31	0
		3 mo	200	0	0.36	0

Continued on following page

TABLE 1—*Continued*

Case no.	Gestational term of infection	Age	IgG (IU/ml)	IgA		IgM (FI)
				FI	OD	
		6 mo	200	0	0.41	0
		8 mo	300	0	0.37	0
		10 mo	300	0	0.42	0
		12 mo	300	0	0.43	0
		14 mo	300	0	0.29	0
17	3	Birth	100	18.9	2.59	13.5
		2 mo	400	13.1	2.01	0
		4 mo	400	0	0.51	0
		5 mo	400	0	0.42	0
		7 mo	400	0	0.44	0
18	2	Birth	2,000	>22.6	>3	0
		2 mo	825	>22.6	>3	0
		9 mo	2,000	>22.6	>3	0
		11 mo	1,200	>22.6	>3	0
19	2	Birth	150	0	0.28	0
		1 mo	100	0	0.35	0
		4 mo	100	0	0.39	0
		7 mo	600	2.7	0.97	0
		8 mo	600	11.6	1.86	0
		10 mo	600	0	0.38	0
20	3	Birth	0	0	0.38	0
		1 mo	15	1.8	0.88	15
		3 mo	80	21	2.80	7
		6 mo	200	2.6	0.96	0
21	3	Birth	16	0	0.43	8.1
		3 wk	250	20	2.7	19
22	3	Birth	40	7.2	1.42	20
		1 mo	75	17.4	2.44	0
		3 mo	100	2.2	0.92	0
23	2	Birth	140	6	1.3	0
		1 wk	100	4.5	1.15	0
		3 wk	80	0	0.41	0

[a] Abbreviations: FI, fixation index; ND, not determined; CSF, cerebrospinal fluid.

(ii) Immunocapture assay. A goat anti-human alpha chain (Diagnostics Pasteur) was used to sensitize microtiter plates at a concentration of 5 μg/ml. The human samples were added (200 μl) at a dilution of 1/20 and were incubated for 1 h at 40°C. After three washes with a 1% Tween 20–0.01% Merthiolate-Tris-NaCl buffer, a *T. gondii* antigen-conjugate anti-P30 complex was added (200 μl). Antigen was used at a concentration of 100 μg/ml, and the horseradish peroxidase-conjugated anti-P30 was the monoclonal antibody against P30 used in Platelia-Toxo IgM (1, 11). After an additional 1 h of incubation at 40°C followed by four washes, substrate buffer (0.05 M citric acid [pH 5.6] and sodium citrate containing 0.03% hydrogen peroxide and 0.01% sodium Merthiolate) and 5% chromogen (orthophenylenediamine-HCl) were added (200 μl), and the reaction was stopped after 30 min at room temperature by the addition of a 4 N sulfuric acid solution (100 μl) before the optical density (OD) was measured at 492 nm. Results are given both as OD and as fixation index, using control sera from adults: a highly positive serum (R5), a cutoff serum (R4), and a negative serum (R3) were used to validate the test: fixation index = [(OD of serum − OD of R4)/(OD of R5 − OD of R4)] × 10. Validation criteria of the assay were established as follows. The ratio OD of R4/OD of R3 should be greater than 1.5, the OD of the positive control (R5) should be higher than 0.5, and the ratio OD of R5/OD of R4 should be greater than 1.5.

TABLE 2. Detection of specific anti-P30 IgM and IgA antibodies in children tested at birth and during follow-up

Status (time of test)	No. of infants			
	IgA^+ IgM^+	IgA^+ IgM^-	IgA^- IgM^+	IgA^- IgM^-
Uninfected (birth)	0	1	1	47
Infected (birth)				
1st term[a]	1	2	0	1
2nd term	0	5	0	1
3rd term	8	0	1	2
Infected (follow-up)	30	54	2	137

[a] Period of maternal infection during pregnancy.

RESULTS

The detection of anti-P30 IgM and IgA antibodies in 223 samples collected from 23 congenitally infected and 49 uninfected newborns was compared. Anti-P30 IgA antibodies were detected in 84 samples (in 54 samples, they were detected in the absence of anti-P30 IgM antibodies), and anti-P30 IgM antibodies were detected in 32 samples (in 2 samples, they were detected in the absence of IgA); anti-P30 IgA and IgM antibodies were simultaneously present in 30 samples and simultaneously absent from 137 samples.

A comparison of the results described below for anti-P30 IgA and IgM antibodies was performed (Table 2). For anti-P30 IgM and IgA antibody detection in children tested at birth, the specificity was the same (48 of 49 [98%]), but the sensitivity was better for anti-P30 IgA antibody detection (10 of 21 [48%] for IgM antibody versus 16 of 21 [76%] for IgA antibody). Differences between results observed at birth in the congenitally infected children (Table 2) are not statistically significant (chi-square test, $P = 0.07$), but results observed during the follow-up period (Table 2) are statistically highly significant (chi-square test, $P < 0.001$).

Uninfected children. At birth, the serum from one uninfected child was weakly positive for anti-P30 IgA antibodies (fixation index, 1.3) but negative for anti-P30 IgM antibodies, and the serum from another uninfected child was negative for anti-P30 IgA antibodies but weakly positive for anti-P30 IgM antibodies (fixation index, 3). These two false-positive data are likely due to a contamination by maternal blood, since sera from the two children were negative at day 7 for both anti-P30 IgA and anti-P30 IgM antibodies. In 47 of the 49 uninfected children, neither anti-P30 IgA nor anti-P30 IgM antibodies were detected at birth or in the following months (data not shown).

Infected newborns. Serological results concerning the 23 congenitally infected children are reported in Table 1. Among the 23 infected children, anti-P30 IgA antibodies were present in all infants but not always at birth. They were detected at birth in 16 of the 21 children tested, during intrauterine life but not at birth in 1 child, and in the weeks following birth in 6 other children. Anti-P30 IgM antibodies were detected in 13 of the 23 infected children. These antibodies were present in 10 of the 21 infected children tested at birth. Therefore, like anti-P30 IgA antibodies, they appeared later in some cases.

Children were classified on the basis of the results obtained at birth (two children [cases 9 and 13] were not tested at birth).

(i) IgA^- IgM^- infected newborns. Two of the five congenitally infected children negative at birth for both isotypes (IgA^- IgM^-) were born to mothers infected during the first term of pregnancy. One (case 11) was negative both in utero (fetal blood sample collected at week 21 of gestation) and at birth for anti-P30 IgM and anti-P30 IgA antibodies, but anti-P30 IgA antibodies could be detected in his serum in the absence of anti-P30 IgM response when he was 2 and 3 months old. The other child (case 16) was IgM^+ IgA^+ at week 22 of intrauterine life (whereas the other signs of fetal infection were not present) and was negative at birth for both anti-P30 IgA and anti-P30 IgM antibodies. In this case, anti-P30 IgA and anti-P30 IgM antibodies were not detected in the sera collected during the following months.

In one of the congenitally infected newborns (case 19), who had no anti-P30 IgM or anti-P30 IgA antibody at birth, we noticed the appearance of anti-P30 IgA but not of anti-P30 IgM antibodies when the child was 7 and 8 months old. This occurred concurrently with the rise of antitoxoplasma IgG. Two other congenitally infected children (cases 6 and 20) were born to a mother infected at the end of the last month of pregnancy. These newborns were seronegative at birth, but both anti-P30 IgA and anti-P30 IgM antibodies were present in the serum collected after 1 (case 6) or 2 (case 20) weeks and continued to rise simultaneously; in case 20, anti-P30 IgM antibodies were absent from the sample collected at 6 months, whereas anti-P30 IgA antibodies were still detected at this time.

(ii) IgA^+ IgM^+ infected newborns. All eight congenitally infected children positive at birth for both anti-P30 IgA (IgA^+) and anti-P30 IgM (IgM^+) antibodies (cases 4, 7, 8, 10, 14, 15, 17, and 22) were born to mothers infected during the third term of pregnancy. In one child (case 10), anti-P30 IgA and anti-P30 IgM antibodies were also detected in the cerebrospinal fluid collected at birth. In these children, anti-P30 IgM and IgA antibodies disappeared in some months, depending on the antibody level at birth, but the IgA-specific antibodies always persisted longer than IgM-specific antibodies. In addition, these IgA antibodies appeared again in the second year of life, whereas anti-P30 IgM antibodies were absent at that time (case 15).

(iii) IgA^- IgM^+ infected newborn. For the IgA^- IgM^+ child (case 21), anti-P30 IgA was detected when he was 3 weeks old, and, like those of IgM antibodies, titers of the IgA antibodies continued to rise. This child and his mother were infected very late in the last month of pregnancy.

(iv) IgA^+ IgM^- infected newborns. IgA^+ IgM^- infected children (seven cases) were born to mothers infected during the first or second term of pregnancy. In one congenitally infected child (case 18), presently 11 months old, a high level of anti-P30 IgA antibodies persisted, whereas an anti-P30 IgM response has never been detected. His mother was infected in the second term of pregnancy and treated only with spiramycin until delivery. Because of the absence of specific IgM antibody at birth (anti-P30 IgA antibody was not assayed at this time), this child was treated only with spiramycin. When he was 9 months old, a chorioretinitis was obvious. This development justified a more intensive treatment (pyrimethamine and sulfadiazine). In the other children (cases 1, 2, 3, 5, 12, and 23), anti-P30 IgA antibodies disappeared within a range of some days to 5 months, depending on the antibody level at birth. In addition, these IgA antibodies appeared again in the second year of life, whereas anti-P30 IgM antibodies were absent at that time (case 2), as was also observed in infants 9 and 13, who were not tested at birth but were highly positive for anti-P30 IgA antibody in the following months. In case 5, the demonstration at birth of a chorioretinitis proved the congenital toxoplasmosis, but the follow-up was stopped in our laboratory before the rise in titer of specific IgG antibody.

DISCUSSION

Clinical interest in the detection of specific antitoxoplasma IgA antibodies for early diagnosis of acute acquired and congenital toxoplasma infections has been evaluated during the past few years and is now well established (4, 5, 8, 10, 12, 13, 14). This study presents a new commercial kit which confirms the great value of the detection of IgA antibody response directed against P30, the major surface antigen of *T. gondii*, for the early diagnosis of congenital toxoplasmosis (3). As recently demonstrated by immunoblotting, IgA antibodies to a variety of antigens are present at various stages of toxoplasmosis (7). The predominant antigens recognized have molecular weights of 30,000, 35,000, and 5,000. Interestingly, only the antigens with molecular weights of 35,000 and 30,000 (P30) were recognized by the IgA antibodies from congenitally infected infants. In the present work, we have shown that anti-P30 antibodies were always present in infected children in the antenatal, neonatal, or postnatal period. Detection of these antibodies is confirmed to be very helpful, especially in the absence of clinical signs. This is of utmost importance for better management of an efficient antitoxoplasmic therapy (2, 6).

In congenital toxoplasmosis, anti-P30 IgA antibodies were detected more frequently than anti-P30 IgM in infected fetuses and newborns. The presence of anti-P30 IgA antibodies proves the congenital infection, since, like IgM, IgA was reported to be stopped by the placental barrier. Nevertheless, because of the possibility of contamination by maternal blood at delivery, newborns' samples positive for IgA or IgM antibodies (or both) must be tested again after 1 week. Anti-P30 IgA antibody was present at birth in the majority of cases, whereas anti-P30 IgM antibody was detected in only half of the cases. In all children with both IgA and IgM antibodies, anti-P30 IgA antibodies persisted longer than anti-P30 IgM antibodies.

In two congenitally infected children whose mothers were infected by *T. gondii* in the first term of pregnancy, fetal blood was collected during the second term of gestation. In one child, anti-P30 IgA and IgM antibodies were not detected, probably because the cord blood was collected before the fetus was infected. In similar cases, the possibility of congenital toxoplasma infection cannot be excluded, and the classical management and treatment with spiramycin of infected pregnant women must be continued until delivery (2). In the second child, both anti-P30 IgA and anti-P30 IgM antibodies were detected in fetal cord blood at week 22 of gestation but were absent at birth. Probably because of the progressive development of a functioning immune system during intrauterine life, anti-P30 IgM humoral fetal response seems weaker than anti-P30 IgA response, this latter being more intense and persisting. The serological profile at birth depends on the date of infection in utero. When infection occurred during the third trimester of gestation, the infant had, either at birth or in the following weeks, specific IgA and IgM antibodies simultaneously; in contrast, when infection occurred in the first term, the infant at birth was at the end of the acute phase of fetal infection, characterized by the

absence of IgM antibodies (which probably appeared during fetal life for a short time and disappeared before birth) and the presence of IgA antibodies. This explains the detection of the two isotypes more frequently in the case of a late infection, unless infection was contracted very late in the last month of pregnancy. In this case, anti-P30 IgA antibodies were detected later than anti-P30 IgM antibodies, as observed in the onset of an acute acquired toxoplasmosis. This indicates that additional serodiagnosis for toxoplasmosis must be performed in the first month of life in seronegative newborns, especially if maternal serodiagnosis is in favor of a starting infection (9). As observed in some cases of congenitally infected children, the late detection of anti-P30 IgA antibodies (particularly if neither anti-P30 IgA nor anti-P30 IgM were detected at birth) can constitute an additional criterion of infection.

The data obtained with the Platelia-Toxo IgA kit extend and confirm the interest in anti-P30 IgA in early diagnosis of congenital toxoplasmosis. Anti-P30 IgA antibodies were present more frequently than anti-P30 IgM antibodies in congenitally infected children during fetal life, at birth, or in the weeks following birth. Furthermore, it is clearly indicated by our study that combined testing for both IgM and IgA in the fetus and the newborn is essential for a more efficient diagnosis of infection and that Platelia-Toxo IgA is a simple and reliable method of detecting anti-P30 IgA antibodies. This new test could easily allow a more accurate diagnosis of congenital infection by all laboratories.

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