Immunoflorescent Study of Placenta-Bound IgG in Women with Common Infection at Term

Wifaq Mahmoud Ali AL-Wattar

ABSTRACT:

BACKROUND:

The level of IgG increases during pregnancy reaching a maximal level at term and was clamed to have a role in the immunological initiation of labor at term, some times the presence of infection during pregnancy might increases the level of such antibody as part of the maternal humeral immune response toward invading pathogens, among which U.T.I and chest infection.

OBJETIVE:

The evaluation of the role of placenta- bound IgG as a part of immune response against the infectious agents causing UTI and or chest infection during the second half of pregnancy.

METHODS:

Fifty one women were included in this study delivered normally at Baghdad teaching hospital,17 women had UTI and 18 where complaining of chest infection at time of sample collection compared with 17 women delivered normally and apparently healthy as a control group ,the level of IgG was detected by direct immunoflorescent test (IF).

RESULTS:

The study showed a significant higher level of IgG in the placental biopsy of women with UTI, and chest infection than those of the control group.

CONCLUSION:

The level of placenta –bound IgG is higher in women having infectious disease during pregnancy than those with normal uncomplicated gestation, which could be considered as a reflection for the level of passive immunity donated to the baby during his first six months of life.

KEY WORDS: placenta-bound IgG, direct immunoflorescent, UT I, chest infection.

INTRODUCTION:

It is well established that all maternal subclasses and allotypes of immunoglobulin G (IgG) crosses human placental barrier during pregnancy[1,2] , but there is little if any transfer of IgM or IgA [3,4] to reach the synsytiotrophoblast by active transport [5] .The antibody transport increases rapidly after the 22 weeks of gestation[2,6,7] to reach a maximal level at term [8,9,10,11] . IgG usually binds to the FC receptors on the syncytiotrophoblastic membrane of the normal human placenta, to be actively transported throw that membrane to the fetus providing it with the passive immunity necessary for the protection against the currently prevalent pathogens during the first sixth months of infancy [12,13,14,15].

PATIENT AND METHODS:

Prospective study was done on three groups of pregnant women attending Baghdad teaching hospital and delivered normally by normal vaginal delivery. The first group comprised 17 women who had history of UTI during their second half of pregnancy while the second group included 16 women with a history of chest infection; both were compared with a group of 17 apparently normal women as a control group.

Cases were chosen of normal singleton pregnancy not less than 37 completed weeks of gestation as confirmed by early ultrasound and clinical examination. Women were checked for the medical history during the attendance to the antenatal clinic, they were exposed to complete physical examination, sent for general urine, sputum examination, and culture and sensitivity were done for the isolates from both specimens to prove the diagnosis and treatment. Immediately after normal vaginal delivery, two cm block of tissue were cut from a central cotyledon of placenta, snap frozen in liquid nitrogen stored at -30 C to be sectioned later on by cryostat microtome at thickness of 4 microns. The placental sections were stained by direct IF test, the procedure includes the use of tissue fixative as Triton X-100 and formaldehyde 3%.

Followed by adding fluorescien conjugated antihuman IgG directed against fragment crystalizable portion of IgG molecules bound to the trophoblastic basement membrane [9, 11, and 16]. Slides were examined under U/V microscopy and the scoring was done according to the density of immunoflorescent staining and graded from 1-4as shown in figures (1, 2).

Department of Microbiology, Collage of Medicine , Baghdad University.

Statistical analysis: The student t –test was used to compare the statistical significant difference in the concentration of IgG deposited on the trophoblastic basement membrane of the placental biopsies at α –level of significance.

RESULT:

When the immunoflorescent scores of IgG deposition were studied in the placentae of all 3groups, the results were interesting, as the density of IgG deposition was found to be significantly higher in the placentae of women delivered by normal vaginal delivery and had a history of UTI than the control group (3.64±0.14) Vs (3.1±0.16), as shown in table (1) with p value <0.01. The difference was also significant between the pregnant women with chest infection and the control group (3.56±0.15) Vs (3.1±0.17) at p value < 0.025 level of significance as clearly shown in table (1).

Table 1: The distribution of immunoflorescent score of IgG in theTwo study groups and the control group.

Immunoflorescent score of IgG	Women with UTI	Healthy Control	Women with chest infection	Total %
1	-	-	-	
2	1(5.88%)	2(11.7%)	1(16.6%)	4
3	4(23.52%)	10(58.8%)	5(27.7%)	19
4	12(70.58%)	5(29.4%)	10(55.5%)	27
Total	17(100%)	17(100%)	16(100%)	50
Mean ± SEM	3.588±0.15 p<0.01	3.176±0.16	3.389±0.14 p<0.025	

Figure 1: Direct immunoflorescent staining of score 3, for a placental biopsy at 38 weeks of gestation, delivered by normal vaginal delivery

Figure 2: Direct immunoflorescent staining of score 4, for a placental biopsy at 39 weeks of gestation, delivered by normal vaginal delivery .

DISCUSSION:

Considerable amount of IgG were detected in the placentas by various methods using different techniques [2, 4, 13, 16, 17]. The trophoblastic bound IgG was found to be of maternal origin crossing the placental barrier through the FC receptor s on the synsytiotrophoblast [5, 17, and 18]. An interesting finding was observed in this study that IgG deposition in the placenta of women with urinary tract infection and or chest infection were significantly higher than the apparently normal women for the same gestational age ,parity and mode of delivery ,since these three factors are the main factors that affects the placenta-bound IgG at term ,which is comparable to the many other studies[14,15,11,16]. This result could be due to rapid increase of synsytiotrophoblast activity and\or the development of FC receptors specific for the IgG molecule transport after mid trimester [2, 6]. Which is the same time of exposure to such microbes causing UTI or chest infection? Since the human placenta regulate the transfer of antibodies from mother to the fetus in a selective manner and restricted only to IgG ,so the maternal immune system will respond by the production of the specific antibodies in amount sufficient to be detected by direct IF technique applied to the placental biopsies [14, 15] . That will provide the new born baby with passive immunity enough to protect him during the first six months after birth [19, 20].

CONCLUSION:

The use of direct immunoflorescent technique is a good indicator for the maternal IgG against the currently prevalent pathogens causing UTI and chest infection during third trimester of pregnancy, which is passively donated to protect the fetus from serious sepsis after birth.

REFERENCES:

1. Griffith GD,Kershaw D,Booth AG.Rabibit-peroxidase –antiperoxidase complex (PAP),as a model for the uptake of IgG by the placenta,(1985).Histo-chem.J,17,867-881.
2. Sidirropoulus D,Herrmann U,Morell A,Von Muvate G, Brandun S. Transplacental passage of intravenous immunoglobuline in the last trimester of pregnancy ,(1986).J.Pediatr.,109,5050508.
3. Nevard CHF,Gaunt M.,Ockelford C.D.The transfer of passive and active immunity .In the immunology the fetus. 1990; 193-214.
4. Bright NA,Ockelford CD,and Anewar M. Ontogeny and distribution of FC receptors ,in the human placenta, .Transport of immuno-surveillance J.Anat, 1994; 184,297-308.
5. Jalali GR,Underwood JL and Mawbary J.F.IgG on normal human placenta is bound both at antigene and FC receptors. Transplaentation proceding, 1989; 21,572.
6. Papadopoulou E,Sifakis S,Giahankis E,Fragouli Y,Karkavitsas N.Increased human placental growth hormones at midtimeser pregnancy may be an index of intrauterine groth retardated to preeclampsia .(2006),16(5-6);290-6 Epub.
7. J ohenson PM,Natving JB,Ystehede UA,Falk ,WP.Immunological studies of human plaentae ,the distribution and character of immunoglobulines in chorionic villi,.Clin.Exp..Immunol., 1977; 30:145-153.
8. Gibbs RS,SweetRI.Maternal and fetel immune system, .Creasy RK,Resnik R,eds. Maternal –fetel medicine ,priciplees and practice .Philadelphia :WB Saunders 1984,605.
9. Akin JW,Conover WB and Deprist PD .Increasing quantity of maternal IgG in trophoblastic tissue before the onset if normal labor, .AML of Obestet and Gynicol. 1990; 162,1154-1157.
10. Charlis R.Whitefeild .Dewharts textbook of Obest and Gynae .for prostagradutes, fifth edition, .The fetus, placenta and amniotic fluid. 1995; 37-85.
11. Al-Ani HA.,Mussawy Kh.,Kubba K., The role of transplacental passage of IgG in the initiation of labor.(1997) . Thesis submitted to the Iraqi commission for the requirement for fellowship of specialization in obstetrics and gynecology.
12. Friden –BE,Makiya R,Nilsson BM.The human placental IgG receptor and IgG transport ,.Am.J. Obestet-Gynecol, 1994; 171 :258-63.
13. Regan L. L.In Bread RW and sharp (eds).Early pregnancy loss,mechanism and treatment,. ,London rcog, 1998; 27-36
14. Ohlsson A, Lasy JB. Intravenous immunoglobulin for preventing infection in preterm and or low birth weight infants. , (2000).Cochrane-Database –System –Rev .,(2):CD000361.
15. Nagao AT,Friedland DN-D,Arslian C,Carnero –Sampaio mm.Elevated level of repertoire of colostral anti –LPS antibodyies may have a significanct role in compensating newborn immunity. .Scand –j- Immunol 2001 ; 53,602-9.
16. AL-Wattar W.M, Mussawy Kh., Omari W.R.The role of transplacental passage and deposition of IgG in the initiation of labour in normal and abnormal pregnancies (1998).Thesis submitted to the collage of med. as part of requirement of degree of Master of Science in medical microbiology (clinical immunology).

17. Gray W.,Wood KB andBarbara J. Detection of IgG bound with in human trophoblastic ..J. of Immunol , 1989; 129,1479-1484,.

18. Leach L,Brun ME,First A,J.and Solif C.Immmunogold localization of endogenous IgG ultra thin frozen section of the human placenta ,cell and tissue research ,1989;257, 603-607.

19. Niloofar R.,Fatuma N ,Arnuad C and Walhlgern R.Non immune immunoglobuline binding and multiple adhesion characterize *Plasmodium* *Falciparum* infected erythrocytes of placental origin. 2006 ;103,13795.

20. AL-Mussawy Kh. M. Detection of IgG bound with in trophoblast of women vaccinated with complete and incomplete tetanus toxoid . J. Fac. Med .Baghdad , 2004 ; 46, 208-212.