Boffin Access

Open Access Journal | Volume: 1.1

Journal of Emerging and Rare Diseases

ISSN 2517-7397

# Scanning Electron Microscopy of Placental Villi Associated to Four Complications of Pregnancy

Olivar Clemente Castejón[1]*
Angela J López G[1]

[1]Professor, General Coordinator of the CIADANA (Center for Research and Analysis Assistancel Teaching of the Nucleus Aragua) Laboratory of Electron Microscopy, Faculty of Health Sciences, University of Carabobo, Aragua State, Maracay, Venezuela

## Abstract

Placental villi in a case of fetal death associate to complications of pregnancy as preeclampsia severe, megaloblastic macrocitic anemia, infection by Zika virus and marginal insertion of umbilical cord were showed in woman pregnancy of 26 years old at 22 weeks of pregnancy causing severe degenerative changes. Is our objective to explore the cell surface of the trophoblast identifying the changes observed with scanning electron microscopy. Samples of normal trophoblast were compared with samples of the case with the four complications. This case was correlated besides with the features found with light microscopy according with a preliminary study previously published. Groups of villi were seen not following their normal morphological pattern. Immature globular terminal villi rush out of mesenchymal villi in irregular aspect. These presented depressions or prolongations of syncytial plasma membrane. Mesenchymal villi are originating mature intermediate villi without to present terminal villi in their trajectory. Immature villi are covered by fibrinoid deposition as others placental villi. Many villi exhibit deep cracks. With frequency curvilinear mature intermediate villi were observed with proliferation of terminal villi in their extremity. These results indicate morphological bad development of the villous tree to the 22 weeks of pregnancy with deficiency of terminal villi and high risk of normal interchange of gases or nutrients contributing with the fetal death.

## Keywords

Scanning electron microscopy; Placenta; Pregnancy; Complications

## Introduction

Recently were studied in histological slides prepared for light microscopy with H&E the placental changes provoked by preeclampsia severe, megaloblastic macrocitic anemia, infection by Zika virus and marginal insertion of umbilical cord that affected to patient of 26 years old who had a fetal death at 22 weeks of pregnancy [1].

These complications caused severe degenerative changes in the placental villous tree being observed in bi dimensional form mediating cuts of 3 to 4 um with light microscopy. The scanning electron microscopy (SEM) can to demonstrate in a better aspect the grade of maturity reached and the morphological changes that are produced by these four factors on the trophoblast of placental villi.

The mature intermediate villi show in normal condition, the characteristic bends of its longitudinal axis with terminal villi that arise from the convex sides of each bend directly or with a narrow week region. These present rich final branching into terminal villi and can to be observed all them in three-dimensional appearance during the third trimester of pregnancy [2].

Scanning electron microscopy is a standard method of biological investigation used for the explore cell surface. Details of the plasma membrane of the syncytiotrophoblast can be seen with this technique. SEM can aid in our understanding of the trophoblast in these conditions and document structural features of placental villi at the final of second trimester [3].

It has been mentioned in recent update of the pathology of Zikav that remains unclear how the virus is able to cross the placental barrier and trophoblast cells at term were resistant to infection, whose mechanism is still unknown suggesting that depend of the gestational stage [4]. Has been suggested that lysis of the syncytial plasma membrane by the Zikav could produce holes on it and entrance of fluids to the stromal region since the intervillous space disorganize it [5].

SEM shows to us the opportunity to examine a panoramic overview of relatively large areas of external surface in trophoblastic tissue. The bad development of villi affected by these four factors will be object of our study with SEM.

## Article Information

| | |
|---|---|
| **DOI:** | 10.31021/jer.20181104 |
| **Article Type:** | Case Report |
| **Journal Type:** | Open Access |
| **Volume:** | 1 **Issue:** 1 |
| **Manuscript ID:** | JER-1-104 |
| **Publisher:** | Boffin Access Limited |
| **Received Date:** | October 28, 2017 |
| **Accepted Date:** | December 30, 2017 |
| **Published Date:** | January 10, 2018 |

**Citation:** Castejon OC, Lopez GAJ. Scanning Electron Microscopy of Placental Villi Associated to Four Complications of Pregnancy. J Emerg Rare Dis. 2018 Jan;1(1):104.

***Corresponding author:**

**Olivar C Castejón**
Professor
General Coordinator of the CIADANA
(Center for Research and Analysis Assistancel Teaching of the Nucleus Aragua) Laboratory of Electron Microscopy
Faculty of Health Sciences
University of Carabobo
Aragua State, Maracay, Venezuela
Tel no: 58-0243-2710520
Fax no: 0243-2713312
E-mail: olivar.ciadanauc@gmail.com

## Material and Methods

Two groups of population of placental villi were taken of placenta study and placenta normal. The group study proceeds from hospitable institution whose placenta was obtained to the 22 weeks of pregnancy. Woman pregnancy of 26 years old in come to the hospital with renal insufficiency, preeclampsia severe, megaloblastic macrocitic anemia, and infection by Zikv at 14 weeks of pregnancy and with eccentric insertion of the umbilical cord observed after delivery.

Zikv was confirmed by nucleic acids detection by RT-PCR in the Hygiene National Institute-Caracas. The serology of patient with placenta study was negative for hepatitis B, C, cytomegalovirus, Epstein Barr virus, rubella and toxoplasmosis.

The newborn was born alive at 22 weeks and died 4 hours after. The infected woman pregnancy persisted with Zikv infection during five days and had knowledge from informative consent and approval by the ethical committee of the hospitable institution for the realization of this investigation according to the Helsinki declaration. The placenta normal was obtained at 38 weeks of patient with an increase of weight of 10 kg without antecedent of disease.

Of each placenta were taken five small specimens of the maternal surface selected at random from the region central parabasal in the vertical plane. Specimens were observed with a Hitachi S2300 scanning electron microscope according to conventional stains of SEM. These images observed were compared or associated with images obtained with light microscopy in a previous report [1]. So, cross sections of placental villi with H&E stain were correlated with similar regions taken with SEM. The concepts of immaturity and fibrinoid deposition are used as mentioned in the literature [2].

## Results

Groups of placental villi are seen in intimate association with terminal villi in curvilinear form. In his trajectory terminal villi rush out of irregular manner of mesenchymal villi which not present bends of its longitudinal axis (Figure 1). These villi are of immature aspect without definite orientation (Figure 2).

Globular terminal villi depressions were observed on the cell surface of the syncytium and extensive prolongations of plasma membrane can to be noted (Figure 3). Mesenchymal villi show transition into mature intermediate villi and these are seen growing with deficiency of terminal villi covered by fibrinoid (Figure 4).

From extensive zones of fibrinoid deposition upon immature villi are originating villi news (Figure 5). Numerous placental villi are covered with fibrinoid (Figure 6). Deep depressions of the cell surface of the trophoblast are exhibited with fissure or cracks (Figure 7). Frequently are observed figures of intermediate placental villi in curvilinear fashion or aspect of letter "s" which present proliferation of terminal villi in their extremity (Figure 8).

## Discussion

During preeclampsia the placenta is exposure to reduced placental perfusion or hypoperfusion, increased branching of villi, area and volume of reduced terminal villus, inflammation and oxidative stress [6].

Maternal anemia results in increased proliferation of trophoblast and stromal cells [7]. Marginal insertion of umbilical cord associated with vasa praevia can to produce rupture of blood vessels during delivery in the placenta with the consequent hemorrhage [8].

Zika virus placental infection induces proliferation and hyperplasia of Hofbauer cells in the chorionic villi but does not elicit villous necrosis, maternal or acute inflammatory cell reaction [9]. Although Rosenberg et al not could to observe necrosis in the placental villi we have demonstrated notable degenerative changes in the syncytium provoked by Zika virus including necrosis [5,9]. It is possible that the differences observed by us are caused by advanced age during pregnancy or by Zikav infection when it is complicated with another sickness [1,5].


**Figure 1:** Placental villi are seen not following their normal morphological pattern

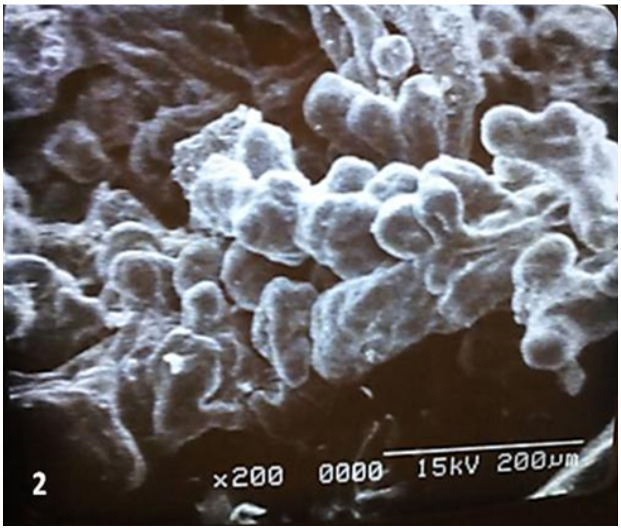


**Figure 2:** Terminal villi rush out disorderly with characteristics of immaturity.


**Figure 3:** Globular villi are noted with depressions and prolongations of the plasma membrane.

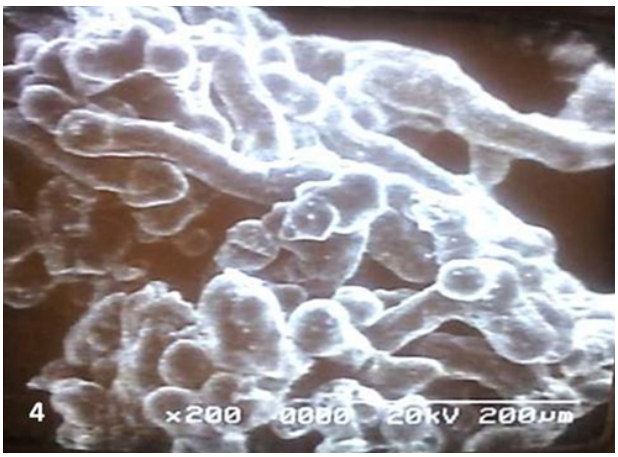


**Figure 4:** Mesenchymal villi are originating mature intermediate villi which not present terminal villi in their longitudinal axis.

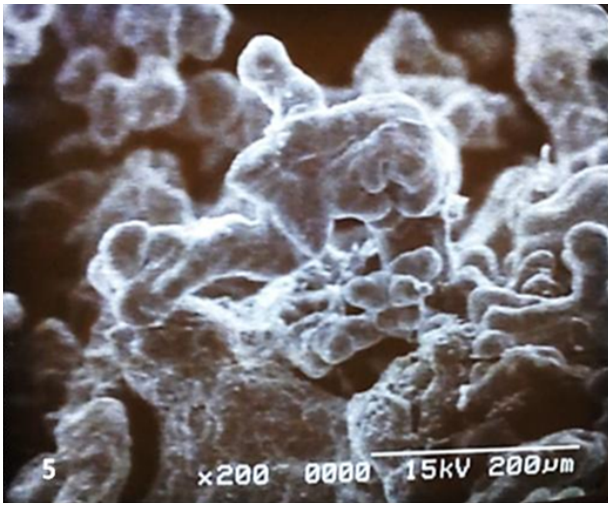


**Figure 5:** Immature villi cover by fibrinoid is seen in the central and lower region.

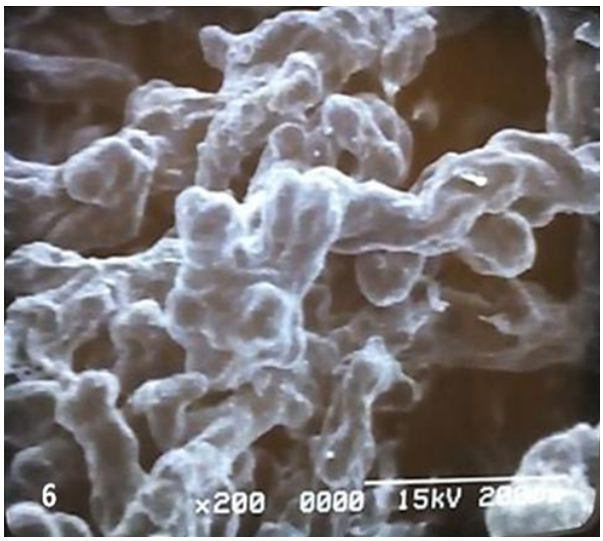


**Figure 6:** Deposition of fibrinoid is exhibited on the cell surface of many villi.

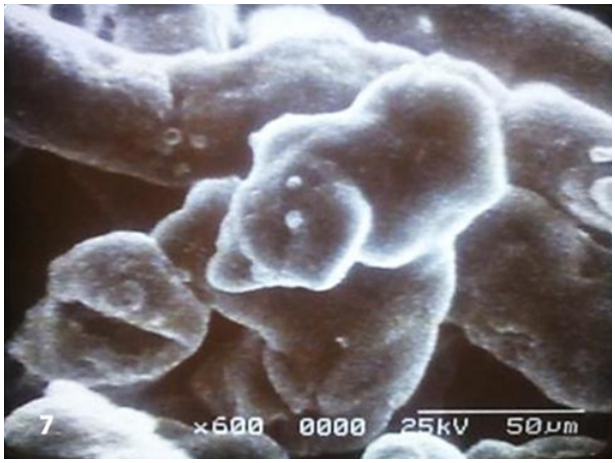


**Figure 7:** Detail of fig4.Cracks or deep depressions are showed in the trophoblast of placental villi.


**Figure 8:** Bad development of villi with curvilinear trajectory and proliferation of terminal villi in their extremity.

Curvilinear aspect of terminal villi is provoked by degenerative changes observed in stromal region as has been noted in previous work [5]. When placental villi maintain the organization of the stromal region entire; these are straight and they are curved when there is disorganization of placental stroma. Curved villi are own of preeclampsia.

Placental immaturity feature has been seen in villi being affected only by Zika virus infection, by Chikungunya virus and placenta infected by HIV and HPV [5,10-12]. It appears that the virus affect noticeably the immature intermediate villi provoking diminished development of the placental villi.

In preeclampsia it has been described a reduction of the microvilli of the syncytium, excessive syncytial knothing or budding sprouting, which is known as Tenney-Parker change, and alterations in their thickness which can be the origin of the depressions observed on the syncytium [1,2].

The intervillous space is obliterated in many villi that are in intimate association and lack of circulation in hypoxic condition. It leads to degenerative changes described in previous work [1]. This condition is increased by the megaloblastic macrocytic anemia that could to produce mesenchymal villi which originate villi with deficiency of terminal villi and placental villi ramifying from immature villi in irregular aspect.

Fissure or cracks seen in the syncytium correspond with regions where the trophoblast was deportated to the intervillous space into the maternal circulation in this preeclamptic pregnancy [13,14].

In the formation of these fissures the activity of Zikav could to be contributing with lysis of the syncytial plasma membrane as has been suggested [1].

Figures of intermediate placental villi in curvilinear appearance were also seen in sickle cell disease associate to depressions and fibrinoid deposition [15].

Placental villi as observed in Figure 8 or with aspect of letter "S" are examples of bad development. Marginal insertion is related with thrombosis, fewer penetrating artery, fewer cotyledons and abnormal blood flow [16,17]. In our case, preeclampsia progress with reduction of blood flow and Zikav provoke damage to blood vessels that transport blood associate to megaloblastic macrocytic anemia [1].

These events combined carry to bad development of the villous tree. In this case terminal villi are small, being of 50um when compared with normal terminal villi of 60um [18]. Zikav and high blood pressure have produced a general disorganization of the structure of the villi. High blood pressure, viral activity, and the hypoxia induced by marginal insertion and megaloblastic macrocytic anemia have originated this morphological disorder in the villous ramifications.

It has been described that Zikav infection has a low fatality rate in adults and few cases occurred of death are associate with another conditions such as lupus and sickle cell anemia [17]. In our case associate to fetal death Zikav infection has been interacting with preeclampsia, macrocitic anemia and marginal insertion of the umbilical cord which have created a severe case of hypoxia. And this condition has been considered as very important cause of mortality [19].

In conclusion, the villous tree was found with small terminal villi, deficiency of terminal villi and bad development of villi which have provoked high risk for the interchange of gases or nutrients contributing with the fetal death.

## Acknowledgement

We are grateful to Dr. Nora López of the service of Obstetrics and Gynecology of Clinic Medicine "The Floresta" in Maracay by the obtention of the placental material and to our secretary Laury Rosely of Ovalles by to transcribe the manuscript.

## References

1. Castejón S OC, López GA (2017) Four complications of pregnancy affecting the placental villi. Adv Biol Biomed 3: 1-8.
2. Benirschke K, Kaufmann P (2000) Pathology of the human placenta. 4ed. New York: Springer-Verlag 123, 558.
3. Hodges GM, Car KE (1983) Biomedical research applications of scanning electron microscopy. Vol 3. London: Academic Press, 1983.
4. Ramos da Silva S, Gao SJ (2016) Zika virus: An update on epidemiology, pathology, molecular biology and animal model. J Med Virol 88: 1291-1296. doi: 10.1002/jmv.24563.
5. Castejón OC (2016) Zika virus affects the placental villi. BMR Medicine 3: 1-6.
6. James MR, Escudero C (2012) The placenta in preeclampsia. Pregnancy Hypertens 2: 72-83. doi: 10.1016/j.preghy.2012.01.001
7. Kosanke G, Kadyrov M, Korr H, Kaufman P (1998) Maternal anemia results in increased proliferation in human placental villi. Placenta 19: 339-357. doi: 10.1016/S0143-4004(98)80024-6
8. Bohiltea RE, Cirstoiu MM, Ciuvica AI, Munteanu O, Bodean O, et al. (2016) Velamentous insertion of umbilical cord vasa praevia: case series and literature review. J Med Life 9: 126-129.
9. Rosenberg AZ, Yu W, Hill DA, Reyes CA, Schwartz DA (2017) Placental pathology of Zika virus: Viral infection of the placenta induces villous stromal macrophage (Hofbauer cell), proliferation and hyperplasia. Arch Pathol Lab Med 141: 43-48. doi: 10.5858/arpa.2016-0401-OA
10. Castejón OC (2015) Immaturity in placental villi affected by chikungunya virus. Electron J Biomed 3: 21-29.
11. Castejon Sandoval OC (2016) The placenta in a case of pregnant woman infected by Chikungunya virus. J Virol Retrovirol 2: 1-4.
12. Castejón SOC, López GAJ (2013) The placenta infected by HIV and HPV. Elect J Biomed 3: 28-35.
13. Sargent IL, Germain SJ, Sacks GP, Kumar S, Redman CW (2003) Trophoblast deportation and the maternal inflammatory response in preeclampsia. J Reprod Immunol 59: 153-160
14. Knight M, Redman CWG, Linton EA, Sargent II (1998) Shedding of syncytiothrophoblast microvilli into the maternal circulation in preeclamptic pregnancies. Br J Obstet Gynaecol 105: 632-640
15. Castejón S OC, Molinaro VMP, Rivas AE, Scucces MMG (2003) Vellosidades placentarias terminales filiformes en la anemia drepanocitica. Gac Med Caracas 111: 17-22.
16. Nordenvall M, Ulberg V, Laurin J, Ligman G, Sandstedt B, et al (1991) Placental morphology in relation to umbilical artery blood velocity waveforms. Eur J Obstet Gynecol Reprod Biol 40: 179-190
17. Alvarado MG, Schwartz DA (2017) Zika virus infection in Pregnancy, Microcephaly and Maternal Fetal Health: What we think, what we know and what we think we know. Arch Pathol Lab Med 141: 26-32. doi: 10.5858/arpa.2016-0382-RA
18. Baergen RN (2011) Manual of pathology of the human placenta. 2ed. New York: Springer. 13-21.
19. Stanek J (2013) Hypoxic patterns of placental injury: A review. Arch Pathol Lab Med 137: 706-720. doi: 10.5858/arpa.2011-0645-RA