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ORIGINAL ARTICLE

Smoking parental as risk factor for the development of Neonatal Respiratory Distress Syndrome

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Abstract

The Respiratory Distress Syndrome (RDS) is one of the most frequent pathologies in the premature neonates and a major cause of morbidity and mortality. The objective of this study was to determine the association between parental smoking and the development of this syndrome. This study was an observational, longitudinal, retrospective, analytical, prolective type, with the neonates in the Neonatal Intensive Care Unit (NICU) of the Hospital Regional Monterrey (HRMI), who developed RDS (cases) and those that do not (controls), during the period January 2012 - April 2015, in both groups were determined the smoking habits of the father and the statistical analysis using SPSS (v. 14). The total sample was 85 RN, of which 46 developed SDR and 39 didn't develop it, predominantly the genre male (56%). Seventy per cent of the group of parents who denied smoking, their children developed SDR, while 64% of parents who reported smoking, their children did not manifest this syndrome. The p-value was of 0.002, OR = 0.245, with 0.099 to 0.607 range. Conclusion: Parental smoking isn't a risk factor for the development of RDS in the neonates, it could be considered a protective factor. (Gac Med Mex. 2016;152:554-6) **Corresponding author:** Mario Alberto Arrieta-Mendoza, oiramx5@hotmail.com

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ntroduction

Respiratory distress syndrome (RDS) of the newborn (NB) is the most common complication of prematurity¹. It consists in lung immaturity of the preterm infant that is not only biochemical –lung surfactant deficit–, but also morphologic and functional, since pulmonary development has not yet been completed in these immature infants². It is the cause of higher immediate and long-term mortality and morbidity in spite of great advances for its prevention and perinatal treatment³. Its incidence is inversely related to gestational age and birth weight. It is observed in 60%-80% of children with gestational age under 28 weeks, in 15%-30% of those with 32-36-week gestational age and in 5% of those who are born with more than 37 weeks of gestation (WOG)⁴.

Factors increasing both RDS incidence and seriousness such as, for example, maternal diabetes, male gender and cesarean delivery without labor, have been described. The factors that decrease its incidence are: Hypertensive disease of pregnancy, premature membrane rupture, addiction to narcotics, smoking and,

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Figure 1. Distribution by gender in the entire sample and by group. RDS: respiratory distress syndrome; No RDS: not developing respiratory distress syndrome.



Figure 2. Distribution by WOG at birth in the entire sample.

very important, exposure to corticoids soon before preterm birth⁵. Chronic stress conditions have been described to be associated with an acceleration of fetal lung maturation, through a possible increase in the concentrations of fetus-produced endogenous cortisol⁶.

The direct association between maternal smoking and its adverse effects at birth has been repeatedly demonstrated. However, little is known about indirect exposure to tobacco smoke during pregnancy and its adverse effects. Jaakola, et al. reported an increase in the risk for prematurity, low birth weight and small size for gestational age.

In one meta-analysis, the relationship between passive smoking and its effects at birth was examined in non-smoker women and it was concluded that maternal passive smoking at early, intermediate or late pregnancy influences to a larger extent on fetal growth; however, it is not reported as a risk factor or to be related to the development of RDS⁷.

Other studies have suggested that the association between paternal smoking and low birth weight and/or prematurity is not significant⁸.

Since smoking is one of the most common addictions in the general population, it is of great interest both for the physician and parents of NBs who develop RDS knowing if there is or not a relationship between paternal smoking and the development of this syndrome. By demonstrating a relationship between both, prevention measures will be redirected in order to avoid the development of RDS and this way contribute in decreasing morbidity and mortality of patients in this age group.

Material and methods

A longitudinal, retrospective, analytical, prolective, observational-type study was conducted, taking as sample the totality of premature, younger than 37 WOG, male and female newborns diagnosed with RDS (cases) and those without RDS (controls) admitted to the neonatal intensive care unit (NICU) of the Hospital Regional Monterrey ISSTE (HRMI) during the January 2012-April 2015 period. Exclusion criteria: NBs whose mother referred positive smoking. Elimination criteria: Fathers who refused to participate in the study, lack of a telephonic reference in the NB medical file, fathers not located at the moment they were looked for to obtain the data required for the study. Fathers who met the inclusion criteria received a telephone call and were applied a short questionnaire about their tobacco-consumption habits. The obtained data were recorded in Excel spreadsheets and processed with the SPSS statistical package (V. 14).

Results

Total study sample was comprised by 85 NBs, out of which 46 had RDS and 39 did not. Of this total, 44% were females and 56% males (Fig. 1).

With regard to WOG at birth, the total sample mean was 32.5 WOG and median was 34.0 WOG (Fig. 2).

Among the fathers, 46% of them referred being positive to tobacco consumption and 54% denied it (Fig. 3).



Figure 3. Paternal smoking distribution in the entire sample and by group. (+): positive for smoking; (-): smoking denied. RDS: respiratory distress syndrome; No RDS: not developing respiratory distress syndrome.



Figure 4. Packs/year distribution among all studied fathers.

In the calculation of the packs/year index, the mean in the total sample was 1.8 (Fig. 4).

In 70% of the group of fathers who denied consuming tobacco, their children developed RDS, whereas in 64% of the parents who referred being smokers, this syndrome did not occur to their children. The p-value was 0.002, with an OR = 0.245 and an interval of 0.099 to 0.607.

Discussion

When the results obtained in this study are analyzed, we can observe that there is a predominance of the male gender in NBs who develop RDS in the NICU, which is consistent with findings referred by López-Candiani from his study on this syndrome in a tertiary care public hospital⁵. Similarly, we can corroborate reports by North American authors indicating that the lesser the WOG at birth, there is higher predisposition for the development of RDS⁴. In addition, we can also confirm what Jaakola JJ et al. reported in their study about prematurity risk increase in mothers with passive or indirect smoking⁷. The p-value obtained indicates that the association is statistically significant, which in this case, it was not the expected association, since our alternative hypothesis was refuted when the percentage of smoker fathers of NBs who did not develop RDS was found to be higher than that of parents of NBs who did develop it. This supports one of the little studied theories on chronic stress and maternal-paternal smoking during pregnancy increasing the release of fetal cortisol, which favors earlier maturation of fetal lungs, thus preventing the development of RDS at birth⁶.

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References

- Velaphi S. Tratamiento con surfactante selectivo temprano versus tardío para el síndrome de dificultad respiratoria neonatal. 2010. Consultado el 27-05-2015, de OMS. Disponible en: http://apps.who.int/rhl/newborn/ cd001456_velaphis_com/es/.
- López de Heredia J, Valls A. Síndrome de dificultad respiratoria. Hospital de Cruces, Barakaldo, España: Asociación Española de Pediatría; 2008.
- Fehlmann E, Tapia JL, Fernández R, Bancalari A, Fabres JD, Apremont I, et al. Impacto del síndrome de dificultad respiratoria en recién nacidos de muy bajo peso de nacimiento: estudio multicéntrico sudamericano. Archivos argentinos de pediatría. 2010;108(5):393-400.
- Kliegman RM, Behrman RF, Jenson HB, Stanton B. Tratado de Pediatría. 18 ed. Barcelona: Elsevier; 2008. pp. 731-41.
- López-Candiani C, Santamaría-Arza C, Macías-Avilés HA, Cruz-Galicia J. Dificultad respiratoria por deficiencia de surfactante en un hospital público de tercer nivel sin maternidad. Factores asociados a mortalidad. Boletín médico del Hospital Infantil de México. 2010;67(2):98-107.
- De Nobrega-Correa H, Reyna-Villasmil E, Santos-Bolívar J, Mejía-Montilla J, Reyna-Villasmil N, Torres-Cepeda D, et al. Enfermedad de membrana hialina en recién nacidos de pacientes preeclámpticas. Revista de Obstetricia y Ginecología de Venezuela. 2012;72(2):77-82.
- Ashford KB, Hahn E, Hall L, Rayens MK, Noland M, Ferguson JE. The effects of prenatal secondhand smoke exposure on preterm birth and neonatal outcomes. J Obstet Gynecol Neonatal Nurs. 2010;39(5):525-35.
- Andriani H, Kuo HW. Adverse effects of parental smoking during pregnancy in urban and rural areas. BMC Pregnancy Childbirth. 2014; 14:414.