Journal of The Cardiovascular Thoracic Anaesthesia and Intensive Care Society

GKDA Derg 2025;31(2):95-101
Doi: 10.14744/GKDAD.2025.92300

RESEARCH ARTICLE

Thyroid Hormones, Cortisol, and Prolactin: Are They Associated with Respiratory Distress Syndrome in Premature Infants in the Neonatal Intensive Care Unit?

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ABSTRACT

Objectives: In infants hospitalized in neonatal intensive care units due to prematurity, respiratory distress syndrome (RDS) and its complications represent the leading causes of morbidity and mortality. Although surfactant deficiency is the primary cause of RDS, various endogenous hormones—such as prolactin, cortisol, and thyroid hormones—may also influence fetal lung maturation. This study aimed to investigate the risk factors for RDS and to evaluate the relationship between blood levels of prolactin, cortisol, and thyroid hormones and RDS in premature infants.

Methods: The study included 117 premature infants, 56 with RDS and 61 without RDS.

Results: Infants with RDS had significantly lower gestational ages and birth weights. A fifth-minute APGAR score of ≤7 was associated with a higher incidence of RDS. Neonates delivered via cesarean section had a lower risk of RDS than those born vaginally. A significant association was observed between preterm premature rupture of membranes and RDS. Total T4, cortisol, and prolactin levels were significantly lower in infants with RDS. Both TSH and prolactin levels showed a decreasing trend with lowering gestational age in neonates with RDS. Notably, hospital stays were longer and mortality rates were higher in the RDS group.

Conclusion: The inverse correlation between cortisol levels and RDS supports the hormone's protective role in pulmonary development. Furthermore, serum prolactin levels decreased proportionally with gestational age in infants with RDS, suggesting a potential role of prolactin in lung maturation. The findings also highlight the contribution of thyroid hormones in promoting surfactant synthesis and pulmonary function in preterm neonates.

Keywords: Cortisol, prematurity, prolactin, respiratory distress syndrome, thyroid hormones

Please cite this article as: "Hekim Yılmaz E, Akın Y, Yılmaz B. Thyroid Hormones, Cortisol, and Prolactin: Are They Associated with Respiratory Distress Syndrome in Premature Infants in the Neonatal Intensive Care Unit?. GKDA Derg 2025;31(2):95-101".

Introduction

Respiratory distress syndrome (RDS) is one of the most common diseases in premature newborns. The accepted definition of preterm birth is one at less than 37-week completed gestation. RDS is caused by structural immaturity of the lungs and lack of surfactant. Most pulmonary surfactant is synthesized usually after 30–32 weeks of gestation.[1,2] Surfactant enables optimal gas exchange by reducing the surface tension within the alveoli, enhancing alveolar expansion, and preventing alveolar collapse during expiration. Insufficiency and immaturity of the surfactant lead to progressive atelectasis and dysfunction of the lungs. Clinical manifestations of RDS begin shortly after birth with tachypnea, dyspnea, intercostal retractions, grunting, nasal flaring, and cyanosis. Impaired gas exchange leads to hypoxia, impaired CO_2 excretion, and respiratory acidosis. Persistent hypoxia results in metabolic acidosis, decreased cardiac

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Submitted: January 01, 2023 **Revised:** February 01, 2023 **Accepted:** February 01, 2023 **Available Online:** June 30, 2025

The Cardiovascular Thoracic Anaesthesia and Intensive Care - Available online at www.gkdaybd.org

output, and hypotension. Despite advances in neonatal intensive care and treatment modalities, RDS is still a major cause of morbidity and mortality in premature infants.[2]

Although the main cause of RDS is surfactant deficiency, various hormones, including prolactin, cortisol, testosterone, estradiol, estrone, and thyroid hormones, are known to influence the production and secretion of surfactant. In the amniotic fluid, a rise in cortisol levels is recorded before an increase in the lecithin/sphingomyelin ratio, which is an indicator of surfactant maturation. Also, lower blood cortisol level is associated with higher severity of RDS in preterm infants.[3,4] These findings implicate cortisol as one of the key factors in fetal surfactant maturation, and glucocorticoids have been used to accelerate surfactant maturation and reduce the incidence of RDS.[5] Thyroid hormones are essential for adaptation to extrauterine life, including lung development and fluid control, adaptive thermogenesis, normal growth, central nervous system maturation, and a diverse range of metabolic processes.[6] They have a synergistic effect with glucocorticoids and influence surfactant synthesis. Hypothyroxinemia of prematurity is characterized by low levels of circulating thyroid hormones, particularly serum T4 levels, in the first 1–2 weeks of life despite normal thyroid-stimulating hormone (TSH) levels.[6] Hypothalamic-pituitary-thyroid immaturity and smaller mass of the thyroid gland itself contribute to hypothyroxinemia of prematurity. Complications of prematurity such as RDS contribute to nonthyroidal illness-like changes and hypothyroxinemia.[7] In the vast majority of preterm infants, hypothyroxinemia is transient; however, permanent hypothyroidism due to thyroid dysgenesis or enzyme defects may occur concurrently. Therefore, careful monitoring of thyroid function and long-term follow-up is mandatory. Human fetal prolactin levels rise prior to the increase in surfactant synthesis. The possibility of a significant role for prolactin as a trigger of lung surfactant synthesis was suggested by the rapid increase in lung phospholipid levels in rabbit fetuses injected with prolactin.[8]

In the light of all these data, this study was designed to investigate the risk factors for development of RDS and to delineate the association of blood prolactin, cortisol, and thyroid hormone levels and RDS in premature neonates hospitalized in the neonatal intensive care unit.

Methods

The subjects of this study were premature infants admitted to a tertiary neonatal intensive care unit (NICU) over a 7-month period. Gestational age was determined based on at least two of the following criteria:

1. Clinical assessment using the New Ballard Scoring System within the first 48 hours of life;[9]
2. Date of the mother's last menstrual period;
3. Findings from fetal ultrasonography.

Infants with a gestational age of less than 37 completed weeks were classified as premature newborns. Neonates with clinical or laboratory findings suggestive of infection, major congenital anomalies, intracranial hemorrhage; pneumothorax, a history of complications such as necrotizing enterocolitis, maternal endocrine disorders, or those who had received blood transfusions or exchange transfusions were excluded from the study.

The fifth-minute Apgar score was recorded and utilized to evaluate the clinical status of the newborn. A score of 7 or more indicates that the baby is in good condition.

Participants were categorized into two groups based on the presence or absence of RDS. The diagnosis of RDS was established based on the presence of at least two of the following criteria:

1. Requirement for oxygen therapy and ventilatory support within the first 24 hours of life;
2. Diffuse reticulogranular pattern observed on chest radiography;
3. Clinical signs such as tachypnea, dyspnea, intercostal retractions, grunting, nasal flaring, and cyanosis.

It was approved by the local ethics committee (15.09.2008, 28001928-501.07.01-24) and conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from the parents of all participants.

Blood samples were obtained either from cord blood or from venous samples collected within the first 24 hours of life for other clinical indications. Serum cortisol, prolactin (PRL), TSH, free T4 (fT4), and total T4 (tT4) levels were measured using a chemiluminescent immunoassay method in the biochemistry laboratory of our center. Demographic data, presence of RDS, need for mechanical ventilation, mode of ventilatory support, serum hormone levels (cortisol, prolactin, TSH, fT4, tT4), duration of hospitalization, and patient survival were recorded. Risk factors associated with the development of RDS, as well as the relationship between hormone levels and RDS, were evaluated.

Statistical Analysis

The data obtained from the study were transferred to an electronic platform and analyzed using the Statistical Package for the Social Sciences (SPSS) version 20.0 (SPSS Inc., Chicago, IL, USA). Categorical variables were expressed as numbers and percentages, while continuous variables such as T3, fT4, TSH, birth weight, and gestational age were

Table 1. Demographic characteristics of the patients and risk factors for RDS

Characteristics	All patients		RDS (+)		RDS (-)		p
	n	%	n	%	n	%	
Gender (male)	52	44.4	56	50	24	39.4	0.246
Birth weight (gram)							<0.0001
<1000	17	14.5	16	28.6	1	1.6	
1000-1500	34	29.1	24	42.9	10	16.4	
>1500 g	66	56.4	16	28.6	50	82	
Gestational age (week)*							<0.0001
<28	6	5.1	6	10.7	0	0	
28-30	15	12.8	11	19.6	4	6.6	
31-33	39	33.3	24	42.9	15	24.6	
34-37	57	48.7	15	26.8	42	68.9	
Appropriateness of BW for GA							0.337
AGA	92	78.6	42	75	50	82	
SGA	25	21.4	14	25	11	18	
5th min apgar score**							<0.0001
≤7	60	51.3	43	76.8	17	27.9	
>7	57	48.7	13	23.2	44	72.1	
Mode of delivery							0.001
Vaginal delivery	25	21.4	19	33.9	6	9.8	
C/S	92	78.6	37	66.1	55	90.2	
pPROM	13	11.1	11	19.6	2	3.3	0.007
LOHS (days), mean±SD	17.2±16.4		22.95±19.81		11.90±9.90		<0.0001
Mortality	28	23.9	22	39.3	6	9.8	<0.0001
Total	117	100	56	47.9	61	52.1	

*: Gestational age according to new Ballard score; **: Apgar scoring system is detailed in Table 4. RDS: Respiratory distress syndrome; BW: Birth weight, GA: Gestational age; AGA: Appropriate for gestational age; SGA: Small for gestational age; min: Minute; C/S: Cesarean section; pPROM: Preterm premature rupture of membranes; LOHS: Length of hospital stay; SD: standard deviation.

presented as mean±standard deviation (SD). The data were tested for normality, and since they were found to follow a normal distribution, parametric tests were applied. Chi-square tests were used for the analysis of frequency distributions and prevalence rates, while independent samples t-test and one-way analysis of variance (ANOVA) were employed for comparisons between groups. A p-value of <0.05 was accepted as statistically significant.

Results

The study included 117 patients who were hospitalized for prematurity over a 7-month period in a tertiary neonatal intensive care unit. Gestational ages ranged from 26 to 36 weeks, and 56 infants (47.9%) developed RDS. The demographic characteristics of the neonates enrolled in the study are presented in Table 1.

Fifty percent of the patients who developed RDS and 39.4% of those who did not develop RDS were male. There was no significant association between gender and the risk of RDS (p=0.246). Birth weights of infants in the RDS-positive group were significantly lower than those in the RDS-negative group (p<0.0001). No statistically significant difference was found regarding RDS risk between infants classified as appropriate for gestational age (AGA) or small for gestational age (SGA) (p>0.05). A fifth-minute Apgar score of ≤7 was identified as a significant risk factor for RDS (p<0.0001). Neonates born via cesarean section were less likely to develop RDS compared to those delivered by spontaneous vaginal delivery (p=0.001). A significant association was observed between preterm premature rupture of membranes and the occurrence of RDS (p=0.007). The duration of hospital stay was significantly longer in infants with RDS, with a mean of 22.95±19.81 days compared to 11.9±9.9 days in those without RDS (p<0.0001). The mortality rate was 9.8% and 39.3% for the RDS (–) and RDS (+) groups, with a significantly higher rate in the RDS (+) subjects (p<0.0001) (Table 1).

When TSH, tT4, fT4, cortisol, and prolactin levels were compared between RDS-negative and RDS-positive infants, tT4, cortisol, and prolactin levels were found to be significantly lower in infants with RDS (all p-values <0.05; Table 2).

Table 2. Comparison of TSH, tT4, fT4, Cortisol, and Prolactin levels between infants with and without RDS

Hormones (mean±SD)	RDS (+)	RDS (-)	p
TSH (IU/mL)	19.47±22.83	15.77±11.59	0.278
tT4 (nmol/l)	93.23±34.62	118.44±37.72	<0.0001
fT4 (pmol/l)	15.80±7.51	16.87±6.06	0.396
Cortisol (mcg/dl)	11.68±9.87	35.10±39.15	<0.0001
Prolactin (ng/mL)	207.84±151.00	274.24±142.05	0.016

TSH: Thyroid stimulating hormone; tT4: Total T4; fT4: Free T4; RDS: Respiratory distress syndrome; SD: Standard deviation.

When infants with RDS were categorized according to gestational age, TSH and prolactin levels decreased significantly with decreasing gestational age (both p-values <0.05; Table 3). From the 34th week of gestation onwards, cortisol levels also showed a decreasing trend in parallel with gestational age; however, this association did not reach statistical significance.

When RDS-negative patients were grouped according to gestational age, no statistically significant differences were observed between cortisol, TSH, tT4, fT4, prolactin levels, and gestational age (all p-values >0.05; Table 3).

Discussion

More than a third of newborn deaths are related to preterm birth and its related complications. RDS is one of the commonest health problems of preterm neonates. Structural immaturity of the lungs and surfactant deficiency are the main causes of RDS. It is still one of the significant predictors of neonatal morbidity and mortality in premature newborns despite improvements in treatment and prevention.[10,11] In previous studies, gestational age, male gender, cesarean delivery, perinatal asphyxia, perinatal infection, infants of diabetic mothers, vitamin D status, and abnormal placental implantation were reported as risk factors for RDS.[12] In accordance with previous reports, our study showed that small gestational age and low birth weight are the ultimate threats for RDS. In previous reports, higher predominance of RDS in male infants was attributed to the variances in hormonal regulation involved in the development of lung and delay in surfactant synthesis due to action of androgens.[1] However, in our study, although the majority of children without RDS are girls, we didn't prove any relation with gender and RDS.

Apgar scoring system is used to assess the clinical status of the newborn at birth (Table 4). The scoring system was

Table 3. Comparison of TSH, tT4, fT4, cortisol and prolactin levels in infants with and without RDS according to gestational age

Hormones	Gestational age (week)	RDS (+)		RDS (-)	
		Mean±SD	p	Mean±SD	p
TSH (IU/mL)	<28	5.53±6.51	0.038	-	0.231
	28-30	12.83±12.49		7.60±6.48	
	31-33	17.88±24.09		18.69±15.57	
	34-37	32.48±25.77		15.51±10.05	
tT4 (nmol/l)	<28	86.58±39.82	0.518	-	0.137
	28-30	99.16±44.09		83.48±53.92	
	31-33	86.74±29.63		116.30±39.16	
	34-37	101.90±33.37		122.54±34.76	
fT4 (pmol/l)	<28	14.49±7.08	0.978	-	0.254
	28-30	15.89±5.19		19.35±7.22	
	31-33	15.99±9.87		18.62±7.88	
	34-37	15.95±4.83		16.01±5.12	
Cortisol (mcg/dl)	<28	8.37±5.70	0.670	-	0.038
	28-30	11.75±6.57		83.38±93.36	
	31-33	13.31±12.85		29.72±28.59	
	34-37	10.35±7.59		32.42±33.62	
Prolactin (ng/mL)	<28	90.71±51.20	0.023	-	0.345
	28-30	131.55±142.29		196.23±184.63	
	31-33	244.27±160.41		309.09±111.47	
	34-37	252.37±130.60		269.22±147.40	

TSH: Thyroid stimulating hormone; tT4: Total T4; fT4: Free T4; RDS: Respiratory distress syndrome; SD: Standard deviation.

Table 4. Apgar scoring system

Sign	0	1	2
Heart rate	Absent	Below 100/minute	Above 100/ minute
Respiratory effort	Absent	Slow, irregular	Good, crying
Muscle tone	Flaccid	Some flexion of extremities	Active motion
Reflex irritability	No response	Grimace	Vigorous cry
Color	Pale	Cyanotic	Completely pink

developed in 1952 by Dr. Virginia Apgar as an objective tool to measure signs of physiologic adaptation and gained universal acceptance after it was shown that it predicts survival particularly at 5th minute.[13] A score of 7 or more indicates that the baby is in good condition. Aspects such as hypoxia, hypothermia, and acidosis diminish the synthesis and secretion of surfactant, and further exacerbate RDS.[1] Our analysis revealed that 5th minute Apgar score ≤7 is strongly associated with the risk of RDS.

In our study, although cesarean deliveries were approximately four times more frequent than vaginal deliveries, neonates born via spontaneous vaginal delivery demonstrated a higher likelihood of developing RDS compared to those delivered by cesarean section. Whereas previous studies reported cesarean section as a risk factor for RDS.[1] This contradiction with the literature may be due to the following reason. Since our hospital is located in a low-income region of the city, the vast majority of these babies born prematurely through spontaneous vaginal delivery probably might not have proper antenatal care during pregnancy and were admitted to the hospital imminent to birth. It is known that lack of antenatal care increases risk of death or severe morbidity.[5]

Premature rupture of the fetal membranes is a major cause of preterm birth and its associated infant morbidity and mortality. Our study showed significant association between early membrane rupture and RDS. RDS developed in 84.6% of the examined newborns from pregnancies complicated by premature rupture of the fetal membranes, a higher rate than reported in the literature.[12]

Thyroid hormones act on the lungs of the fetus, increase surfactant production, and facilitate fetal lung maturation. Immediately after birth, significant alterations occur in the thyroid hormone axis. In response to environmental temperature changes, TSH secretion from the pituitary gland increases up to 70 mIU/L within the first 30 minutes of life. This physiological response, known as the "TSH surge," stimulates the thyroid gland to secrete thyroid hormones. Consequently, serum fT4 levels reach their peak concentrations observed during the lifetime and subsequently begin to decrease gradually after the first week of life.[6,14] The pattern of the TSH surge in newborns can be influenced by various factors including lower gestational age, lower body weight, and complications during the fetal period.[14]

In our study, a significant inverse relationship between TSH levels and gestational age was observed in infants with RDS. Previous studies have reported that TSH surge levels in preterm infants were lower in those with RDS. They showed that lower TSH levels immediately after birth were associated with an increased risk of developing RDS. Transient hypothyroxinemia is the most common thyroid dysfunction in preterm infants and is characterized by a temporary postnatal reduction in serum levels of T4 and free T4 but with normal TSH levels.[14] Although the etiology of transient hypothyroxinemia is not clear, withdrawal of maternal–placental T4 transfer, hypothalamic–pituitary–thyroid immaturity, iodine deficiency, and nonthyroidal illness may contribute to this state. In nonthyroidal illnesses, with increasing severity of illness, serum T4 levels decrease.[15] RDS is one of the most common critical illnesses in premature babies over the first week of life. Therefore, whether RDS alters serum thyroid hormone levels or lower hormone levels contribute to increased disease severity has been frequently studied. It has been reported that postnatal T3 and T4 increase in infants with RDS is decreased or may not occur in the first 24 hours. T3 and T4 increases gradually occur in the following weeks.[16]

In our study, we found that tT4 levels were significantly lower in children with RDS. This condition might represent hypothyroxinemia of prematurity, be a consequence of RDS, or reflect a hyperthyroxinemic state that exacerbates the clinical presentation of RDS. Previous studies have shown that tT4 is more affected than fT4 due to low concentrations of binding proteins in these infants, which is attributed to immature liver function.[6]

Thyroid hormones have a crucial role in optimal brain development during the first 2 years of life. Therefore, it is important to determine whether hypothyroxinemia associated with severe illness in preterm infants causes neurodevelopmental deficits or represents merely an

epiphenomenon of the underlying illness. It is also known that in nonthyroidal illnesses, a rise in serum TSH levels, followed by an increase in serum T4 and T3 levels, should occur in parallel with clinical recovery.[16] Correcting the hypothyroxinemia of premature babies with T4 supplements made no difference to long-term neurodevelopmental outcome. Therefore, there must be other factors associated with acute illness in preterm infants, which have significant negative impacts on subsequent neurodevelopmental outcome.[17] Despite available guidelines, timing of screening and optimal treatment of thyroid dysfunction in premature infants remains controversial.[6]

Numerous studies have shown that cord cortisol shows a significant correlation with gestational age and birth weight.[5,12,18,19] Blood cortisol levels tend to be low in premature infants, particularly in those who develop RDS.[3,4] Moreover, the stress response during acute illness in preterm infants may be attenuated due to the limited secretory capacity of the adrenal cortex. Antenatal corticosteroids, at levels mimicking physiological stress, accelerate fetal lung maturation by enhancing the activity of enzymes involved in surfactant biosynthesis. Based on these data, numerous studies have supported the widespread use of antenatal corticosteroids in cases of impending preterm delivery to prevent RDS and its associated complications.[5,18,19] Consistent with previous studies, cortisol levels in the blood samples of our study group were significantly lower in infants with RDS. Although previous studies have reported an association between lower blood cortisol levels and the severity of RDS in preterm infants, in our RDS group, no significant association was found between gestational age and cortisol levels.[3,4] When the relationship between gestational age and blood cortisol levels was examined in non-RDS infants, cortisol levels were significantly higher at 28–30 weeks of gestation compared to other gestational age groups. This may suggest a potential protective role of elevated cortisol levels in this high-risk group against the development of RDS.

In our study group, prolactin levels were significantly lower in infants with RDS. Furthermore, we demonstrated that serum prolactin levels decreased significantly with decreasing gestational age in these patients. Previous studies have reported that serum prolactin levels increase in proportion to gestational age and birth weight. Additionally, compared to infants with normal lung function at the same gestational age, lower serum prolactin levels have been observed in those with RDS.[3,8,20,21] Based on these data, it can be suggested that prolactin may play a role in surfactant synthesis and lung maturation. However, the presence of healthy infants with relatively low PRL levels indicates that prolactin is not the sole factor required for lung maturation. Furthermore, the occurrence of RDS despite elevated serum prolactin levels in two infants born to diabetic mothers highlights that high prolactin levels alone do not provide protection against RDS.[21]

Limitations

Hormone levels were measured from blood samples obtained within the first 24 hours, using either cord blood or postnatal venous samples. This time range may have influenced hormone levels. Repeat measurements were performed only in cases of suspected hypothyroidism; therefore, follow-up data were not available for all patients. Consequently, we were unable to evaluate whether RDS has a persistent effect on serum thyroid hormone levels.

Conclusion

Despite advances in neonatal care, RDS remains a leading cause of morbidity and mortality in premature newborns. The incidence of RDS increases with decreasing gestational age. Therefore, the most effective strategy for reducing the risk of RDS is the prevention of preterm birth. However, several additional factors should be considered, including appropriate antenatal care, transferring mothers at high risk of preterm birth to perinatal centers with experience in the management of RDS, and prevention of perinatal asphyxia. Given that cortisol, tT4, and prolactin levels were significantly lower in infants with RDS compared to those without RDS, these hormones may play a role in surfactant synthesis and fetal lung maturation.

The lower cortisol levels observed in patients with RDS support an inverse relationship between cortisol concentration and the occurrence of RDS. The administration of prenatal corticosteroids to promote lung maturation has become standard practice for preventing RDS in premature infants. In preterm neonates, the expected postnatal rise in TSH and thyroid hormones is often absent, particularly in those with concomitant conditions such as RDS. This phenomenon may be attributed to immaturity of the hypothalamic–pituitary–thyroid axis or the disease state associated with RDS. Although the benefit of routine thyroid hormone replacement therapy has not been demonstrated in these infants, careful monitoring for hypothyroidism is recommended. Serum prolactin levels decreased proportionally with gestational age in all infants, with this decline reaching statistical significance in those who developed RDS, highlighting the potential role of prolactin in lung maturation, as supported by existing literature. Intrauterine therapy involving these hormones, or pharmacological interventions aimed at increasing their levels, remains an open area for future research.

Disclosures

Ethics Committee Approval: The study was approved by the Dr. Siyami Ersek Thoracic Cardiovascular Surgery Training and Research Hospital Ethics Committee (no: 28001928-501.07.01-24, date: 15/09/2008).

Informed Consent: Informed consent was obtained from all participants.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study received no financial support.

Use of AI for Writing Assistance: No AI technologies utilized.

Authorship Contributions: Concept – E.H.Y., Y.A.; Design – Y.A., E.H.Y.; Supervision – Y.A., E.H.Y.; Funding – Y.A., E.H.Y.; Materials – E.H.Y., B.Y.; Data collection and/or processing – E.H.Y., B.Y.; Data analysis and/or interpretation – E.H.Y., B.Y.; Literature search – E.H.Y.; Writing – E.H.Y., B.Y.; Critical review – Y.A., B.Y., E.H.Y.

Peer-review: Externally peer-reviewed.

References

1. Monica NF, Pamela S, Juan QL, Li J. Recent understanding of pathophysiology, risk factors and treatments of neonatal respiratory distress syndrome: A review. Sci Lett 2017;5:70–8.
2. Carnielli VP, Cogo PE. Surfactant metabolism in neonatal lung diseases. In: Neonatology: A Practical Approach to Neonatal Diseases. Milano: Springer Milan; 2012. p. 433–40.
3. Turhan NÖ, Danışman N, Beysanoğlu M, Doğu N, Gökmen O. Umblikal kord prolaktin ve kortisol düzeyleri: Respiratuar distress sendromu ve gebelik haftası ile korelasyonu. Anatolian J Gynecol Obst 1993;3:203–6.
4. Gunes T, Koklu E, Ozturk MA, Koklu S, Cetin N. Evaluation of serum cortisol levels in a relatively large and mature group of ventilated and nonventilated preterm infants with respiratory distress syndrome. Am J Perinatol 2006;23:335–9.
5. Sweet DG, Carnielli V, Greisen G, Hallman M, Ozek E, Te Pas A, et al. European consensus guidelines on the management of respiratory distress syndrome - 2019 update. Neonatology 2019;115:432–50.
6. Zdraveska N, Kocova M. Thyroid function and dysfunction in preterm infants-Challenges in evaluation, diagnosis and therapy. Clin Endocrinol (Oxf) 2021;95:556–70.
7. LaFranchi S. Thyroid function in the preterm infant. Thyroid 1999;9:71–8.
8. Hamosh M, Hamosh P. The effect of prolactin on the lecithin content of fetal rabbit lung. J Clin Invest 1977;59:1002–5.
9. Ballard JL, Khoury JC, Wedig K, Wang L, Eilers-Walsman BL, Lipp R. New Ballard Score, expanded to include extremely premature infants. J Pediatr 1991;119:417–23.
10. Avery ME, Mead J. Surface properties in relation to atelectasis and hyaline membrane disease. AMA J Dis Child 1959;97:517–23.
11. Minuye Birihane B, Alebachew Bayih W, Yeshambel Alemu A, Belay DM, Demis A. The burden of hyaline membrane disease, mortality and its determinant factors among preterm neonates admitted at Debre Tabor General Hospital, North Central Ethiopia: A retrospective follow up study. PLoS One 2021;16:e0249365.
12. Niesłuchowska-Hoxha A, Cnota W, Czuba B, Ruci A, Ciaciura-Jarno M, Jagielska A, et al. A retrospective study on the risk of respiratory distress syndrome in singleton pregnancies with preterm premature rupture of membranes between 24+0 and 36+6 weeks, using regression analysis for various factors. Biomed Res Int 2018;2018:7162478.
13. Apgar V. A proposal for a new method of evaluation of the newborn infant. Originally published in July 1953, volume 32, pages 250-259. Anesth Analg 2015;120:1056–9.
14. LaFranchi SH. Thyroid function in preterm/low birth weight infants: Impact on diagnosis and management of thyroid dysfunction. Front Endocrinol (Lausanne) 2021;12:666207.
15. Simpson J, Williams FL, Delahunty C, van Toor H, Wu SY, Ogston SA, et al. Serum thyroid hormones in preterm infants and relationships to indices of severity of intercurrent illness. J Clin Endocrinol Metab 2005;90:1271–9.
16. Kim Y, Kim Y, Chang M, Lee B. Association between thyroid function and respiratory distress syndrome in preterm infants. Pediatr Rep 2022;14:497–504.
17. Van Wassenaer AG, Kok JH, de Vijlder JJ, Briët JM, Smit BJ, Tamminga P, et al. Effects of thyroxine supplementation on neurologic development in infants born at less than 30 weeks' gestation. N Engl J Med 1997;336:21–6.
18. McPherson C, Wambach JA. Prevention and treatment of respiratory distress syndrome in preterm neonates. Neonatal Netw 2018;37:169–77.
19. Ballard PL, Ballard RA. Scientific basis and therapeutic regimens for use of antenatal glucocorticoids. Am J Obstet Gynecol 1995;173:254–62.
20. Parker CR Jr, MacDonald PC, Guzick DS, Porter JC, Rosenfeld CR, Hauth JC. Prolactin levels in umbilical cord blood of human infants: Relation to gestational age, maternal complications, and neonatal lung function. Am J Obstet Gynecol 1989;161:795–802.
21. Smith YF, Mullon DK, Hamosh M, Scanlon JW, Hamosh P. Serum prolactin and respiratory distress syndrome in the newborn. Pediatr Res 1980;14:93–5.