Taiwanese Journal of Obstetrics & Gynecology 57 (2018) 546–550

Contents lists available at ScienceDirect

Taiwanese Journal of Obstetrics & Gynecology

journal homepage: www.tjog-online.com

Original Article

Maternal pregnancy-induced hypertension increases the subsequent risk of transient tachypnea of the newborn: A nationwide population-based cohort study

Wei-Shan Chang [a], Li-Te Lin [a, b, c], Li-Chuan Hsu [a], Pei-Ling Tang [d, e, f], Kuan-Hao Tsui [a, b, g, *, 1], Peng-Hui Wang [b, h, i, **, 1]

[a] Department of Obstetrics and Gynecology, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan
[b] Department of Obstetrics and Gynecology, National Yang-Ming University School of Medicine, Taipei, Taiwan
[c] Department of Nursing, Shu-Zen Junior College of Medicine and Management, Kaohsiung, Taiwan
[d] Research Center of Medical Informatics, Kaohsiung Veterans General Hospital, Kaohsiung, Taiwan
[e] Department of Nursing, Meiho University, Ping-Tung, Taiwan
[f] College of Nursing, Kaohsiung Medical University, Kaohsiung, Taiwan
[g] Department of Pharmacy and Graduate Institute of Pharmaceutical Technology, Tajen University, Pingtung County, Taiwan
[h] Department of Obstetrics and Gynecology, Taipei Veterans General Hospital, Taipei, Taiwan
[i] Department of Medical Research, China Medical University Hospital, Taichung, Taiwan

ARTICLE INFO

Article history:
Accepted 15 December 2017

Keywords:
Pregnancy-induced hypertension
Hypertension in pregnancy
Gestational hypertension
Preeclampsia
Transient tachypnea of the newborn

ABSTRACT

Objective: To determine the association between pregnancy-induced hypertension (PIH) and transient tachypnea of the newborn (TTN) and to identify the predictive risk factors.

Materials and Methods: Pregnant women with a newly diagnosed PIH (between 2000 and 2013) from the Taiwan National Health Insurance Research Database (NHIRD) were compared with a matched (with respect to age and year of delivery) cohort of pregnant women without PIH. The occurrence of TTN was evaluated in both cohorts.

Results: Among the 23.3 million individuals registered in the NHIRD, 29,013 patients with PIH and 116,052 matched controls were identified. According to a multivariate analysis, PIH (odds ratio [OR] = 1.85, 95% confidence interval [CI] = 1.69–2.03, $p < 0.0001$), age ≥ 30 years (OR = 1.38, 95% CI = 1.26–1.51, $p < 0.0001$), primiparity (OR = 1.37, 95% CI = 1.24–1.5, $p < 0.0001$), preterm birth (OR = 3.4, 95% CI = 3.09–3.75, $p < 0.0001$), multiple births (OR = 2.54, 95% CI = 2.24–2.89, $p < 0.0001$), and cesarean section (OR = 1.71, 95% CI = 1.56–1.88, $p < 0.0001$) were independent risk factors for the development of TTN.

Conclusion: Women with PIH have an increased risk of having infants who develop TTN compared with those without PIH. Additionally, age ≥30 years, primiparity, preterm birth, multiple births, and cesarean section were independent risk factors for the development of TTN.

Introduction

Pregnancy-induced hypertension (PIH), including gestational hypertension, preeclampsia, or eclampsia, is a major cause of maternal morbidity and mortality [1–5]. Preeclampsia is a complication in approximately 3–5% of pregnancies [6,7] and is associated with a higher risk of neonatal death [8–10]. It is characterized by the de novo development of hypertension and proteinuria that arise after 20 weeks of gestation [11–14]. Although the exact pathogenesis of preeclampsia has not been fully elucidated, the main hypothesis is that abnormal cytotrophoblast invasion of

* Corresponding author. Department of Obstetrics and Gynecology, Kaohsiung Veterans General Hospital, No. 386, Dazhong 1st Rd., Zuoying Dist., Kaohsiung City, 81362 Taiwan. Fax: +886 7 3468189.
** Corresponding author. Department of Obstetrics and Gynecology, Taipei Veterans General Hospital, 201, Secion 2, Shihpai Road, Taipei 112, Taiwan. Fax: +886 2 5570 2788.
E-mail addresses: khtsui60@gmail.com (K.-H. Tsui), phwang@vghtpe.gov.tw, pongpongwang@gmail.com (P.-H. Wang).
[1] Kuan-Hao Tsui and Peng-Hui Wang contributed equally to this work.

https://doi.org/10.1016/j.tjog.2018.06.013

spiral arterioles results in reduced uteroplacental perfusion and causes placental ischemia, followed by the release of several anti-angiogenic factors, reactive oxygen species (ROS), and inflammatory cytokines, which then lead to the onset of the clinical symptoms of preeclampsia [15–18].

Transient tachypnea of the newborn (TTN) is a respiratory disorder characterized by tachypnea that develops immediately after birth but resolves within 2–5 days [19]. Delayed reabsorption of the fetal lung fluid has been reported to be a critical mechanism underlying the development of TTN [20]. Amiloride-sensitive sodium (Na+) channels play an important role in fetal pulmonary fluid clearance [21], and dysfunction of those channels may result in TTN [22].

B-type natriuretic peptides (BNP), which play a role in the maintenance of extracellular fluid volume, seem to be able to reduce amiloride-sensitive Na + transport [23,24]. N-terminal pro-B-type natriuretic peptide (NT-proBNP) has been shown to be significantly higher in neonates with TTN [25]. In addition, patients with preeclampsia have been shown to have higher levels of BNP and NT-proBNP [26,27]. Therefore, it is reasonable to hypothesize that BNP and NT-proBNP, induced by PIH, may be released into the fetal circulatory system and may be involved in the development of TTN. To test this hypothesis, we designed a nationwide population-based matched cohort study to assess the relationship between PIH and TTN.

Materials and Methods

Data sources

The National Health Insurance program has covered almost 98% of the population (23 million residents of Taiwan) since 1995 [28–31]. We obtained data for the current study from the National Health Insurance research database (NHIRD), which was established by The National Health Research Institute (NHRI). The NHRID protects the privacy of individuals and provides data to researchers who have the necessary ethical approvals. We obtained anonymous data from the NHRID that did not include information regarding individuals' identities.

Study design and participants

PIH patients between 20 and 50 years of age were assessed based on International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) codes 642.3–642.6 for the following conditions: gestational hypertension (ICD-9-CM codes 642.30, 642.31, 642.32, 642.33, and 642.34), mild preeclampsia (ICD-9-CM codes 642.40, 642.41, 642.42, 642.43, and 642.44), severe preeclampsia (ICD-9-CM codes 642.50, 642.51, 642.52, 642.53, and 642.54), and eclampsia (ICD-9-CM codes 642.60, 642.61, 642.62, 642.63, and 642.64). Only patients with a diagnosis of PIH and who had experienced an inpatient hospitalization were selected for the study to ensure diagnostic validity and to avoid any potential misclassifications.

The data for this study were obtained from January 1, 2000, to December 31, 2013. A total of 29,013 PIH patients were assessed. For each patient with PIH, four patients who were matched with respect to age and year of delivery and who did not have a history of PIH were randomly selected from the NHIRD and were included in the comparison cohort. The index date for the patients in the PIH cohort was the date of their initial PIH diagnosis. The study endpoints were defined as the date of a TTN diagnosis (ICD-9-CM: 770.6), death within 28 days after birth, or the date of the end of the study period. Pregnancy characteristics of the patients were obtained, including age, parity, gestational age, gestational number, whether they had a cesarean section, and any comorbidities. The comorbidities in our study were as follows: diabetes mellitus (DM) (ICD-9-CM: 250), hypertension (HTN) (ICD-9-DM: 401–405), coronary artery disease (CAD) (ICD-9-CM: 410–414), dyslipidemia (ICD-9-CM: 272), chronic kidney disease (CKD) (ICD-9-CM: 585 and 403), chronic obstructive pulmonary disease (COPD) (ICD-9-CM: 491.2, 493.2, and 496), and cerebrovascular disease (ICD: 430–437).

Statistical analysis

The study groups were compared using the chi-square test for categorical variables and independent *t*-tests for continuous variables. The Cox proportional hazards model [32–35] was used to identify risk factors for TTN. Control variables, such as PIH, age, parity, gestational age, gestational number, whether patients had a cesarean section, and common comorbidities, including DM, HTN, CAD, dyslipidemia, COPD, CKD, and cerebrovascular disease, were included as covariates in the univariate and multivariate model. Statistical Analysis Software (SAS) version 9.4 (SAS Institute, Inc., Cary, NC, USA) was used for the data analysis. Comparisons with a p value of less than 0.05 were considered statistically significant.

Results

Participant characteristics

A total of 29,013 patients with PIH and a matched cohort of 116,052 subjects were identified and included in this study. Table 1 shows the demographics and comorbidities of the PIH patients and the matched subjects. The mean patient ages were 30.96 and 30.83 years in the PIH and matched cohort groups, respectively. The majority of patients in both the PIH (56.29%) and matched cohort (56.29%) groups were older than 30 years of age. Patients with PIH had lower parity but higher preterm birth, higher multiple births, and higher cesarean section rates than patients in the comparison

Table 1
Baseline characteristics of patients with pregnancy-induced hypertension and matched cohort.

Parameters	PIH (n = 29,013)		Matched cohort (n = 116,052)		*p* value
	n	%	n	%	
Age, years, mean	30.96		30.83		
< 30	12,681	43.71	50,724	43.71	1
≥ 30	16,332	56.29	65,328	56.29	
Parity, n					
1	17,819	61.42	67,437	58.11	<.0001
≥ 2	11,194	38.58	48,615	41.89	
Gestational age					
Term	22,553	77.73	110,597	95.30	<.0001
Preterm	6,460	22.27	5,455	4.70	
Gestational number					
Singleton	27,316	94.15	113,949	98.19	<.0001
Multiple	1,697	5.85	2,103	1.81	
Cesarean section					
Yes	21,574	74.36	42,288	36.44	<.0001
No	7,439	25.64	73,764	63.56	
Comorbidities					
Diabetes mellitus	112	0.39	69	0.06	<.0001
Hypertension	266	0.92	85	0.07	<.0001
Dyslipidemia	99	0.34	90	0.08	<.0001
Coronary artery disease	26	0.09	71	0.06	0.0938
Chronic obstructive pulmonary disease	36	0.12	66	0.06	0.0001
Chronic kidney disease	187	0.64	158	0.14	<.0001
Cerebrovascular disease	54	0.19	87	0.07	<.0001

PIH, pregnancy-induced hypertension.

cohort. Furthermore, patients with PIH had higher prevalence of DM, HTN, dyslipidemia, COPD, CKD, and cerebrovascular disease.

Incidence of and risk factors for transient tachypnea of the newborn

The incidence of TTN was higher in the PIH group (3.84%) than in the matched cohort group (1.25%). As demonstrated in the univariate and multivariate analyses (Table 2), independent risk factors for the development of TTN included PIH (OR = 1.85, 95% CI = 1.69–2.03, $p < 0.0001$), age ≥ 30 years (OR = 1.38, 95% CI = 1.26–1.51, $p < 0.0001$), single parity (OR = 1.37, 95% CI = 1.24–1.5, $p < 0.0001$), preterm birth (OR = 3.40, 95% CI = 3.09–3.75, $p < 0.0001$), multiple births (OR = 2.54, 95% CI = 2.24–2.89, $p < 0.0001$), and cesarean section (OR = 1.71, 95% CI = 1.56–1.88, $p < 0.0001$).

Discussion

This was a nationwide population-based retrospective cohort study performed to determine whether women who developed PIH had an increased risk of having infants who developed subsequent TTN. In the current study, we assessed individuals who were diagnosed with PIH and a matched control cohort over a follow-up period of 28 days after they gave birth. A higher incidence of subsequent TTN was observed among patients with PIH compared with the control group. The results from the multivariate analysis showed that PIH was an independent risk factor for the development of TTN. Furthermore, age ≥30, single parity, preterm birth, multiple births, and cesarean section were also independent risk factors for developing TTN.

TTN, a common cause of respiratory distress in newborns, often spontaneously resolves within days but may occasionally contribute to fetal morbidity because of hypoxemia, pulmonary air leakage, and persistent pulmonary hypertension [19]. The established diagnostic criteria for TTN were as follows: (1) tachypnea (respiratory rate > 60/min) that developed during the first 6 h following delivery; (2) tachypnea that persisted for at least 12 h; (3) chest radiography findings that were consistent with TTN (e.g., hyperaeration, mild cardiomegaly, distinction of pulmonary vascular structures, or pleural or interstitial fluid); and (4) absence of hyaline membrane disease, meconium aspiration syndrome, congenital pulmonary abnormalities, and congenital heart disease [36]. The etiology of TTN has been suggested to be the delayed resorption of fetal lung fluid, which may originate from defective amiloride-sensitive Na + transport because this plays a major role in clearing the excess lung fluid during the fetal period [37].

BNP plays a key role in the regulation of cardiovascular homeostasis, blood pressure maintenance, and extracellular fluid volume [38]. The main storage form of BNP is pro-BNP, which is cleaved into active BNP, and an inert amino-terminal fragment of pro-BNP (NT-proBNP) [39]. Atrial natriuretic peptide (ANP) has been shown to reduce amiloride-sensitive Na + transport by acting on natriuretic peptide receptor-A (NPR-A) and NPR-B [23]. BNP is known to act on these receptors [24] and may be implicated in neonatal lung fluid clearance. A prospective controlled study by Aydemir and colleagues revealed that neonates with TTN had increased levels of plasma NT-proBNP [25]. Several studies have shown that having hypertensive disorders of pregnancy, especially early-onset or severe preeclampsia, was associated with elevated levels of NT-proBNP [26,40,41] and BNP [27,42]. Additionally, Janus et al. found pro-BNP and NT-proBNP protein in maternal spiral arteries and in syncytiotrophoblasts in all placental samples, and they also found that women with preeclampsia had higher levels of NT-proBNP than controls. Thus, these authors suggested that the placentas in patients with PIH might produce proBNP and NT-proBNP and release them into the maternal circulation [43]. It is reasonable to theorize that if proBNP and NT-proBNP are produced by the placentas of patients who have PIH, these proteins may also be released into the fetal circulatory system, which could result in TTN.

TTN is related to surfactant deficiency [44]. Preeclampsia is associated with oxidative stress (OS) in the maternal circulatory system [16]. By sampling umbilical cord blood during delivery, Negi and colleagues demonstrated that increased OS was observed in the fetal circulatory systems in mothers who experienced preeclampsia or eclampsia [45]. Moreover, OS and ROS can inactivate surfactant by altering the structure and function of surfactant proteins [46,47]. As a result, it is reasonable to postulate that OS and ROS are generated from the placentas of patients with PIH and may also be released into the fetal circulatory system, which would lead to TTN.

Based on the above findings, we hypothesized that maternal PIH may lead to an increased risk of subsequent TTN. A prospective study conducted by Badran et al. showed that maternal hypertension was an independent risk factor for TTN and respiratory distress syndrome (RDS) [48]. In our study, the incidence rate of TTN was 1.85-fold higher in patients who experienced PIH (95% CI = 1.69–2.03, $p < 0.0001$) compared with those in the matched control cohort. Therefore, it is reasonable to believe that abnormal placental products, such as pro-BNP, OS, and ROS, which are

Table 2
Analyses of risk factors for transient tachypnea of newborn among the patients with pregnancy-induced hypertension and comparison cohort.

Parameters	Univariate analysis		Multivariate analysis[a]	
	OR (95% CI)	*p* value	OR (95% CI)	*p* value
PIH				
Yes vs. No	3.29 (3.03–3.56)	<.0001	1.85 (1.69–2.03)	<.0001
Age, years				
≥ 30 vs. < 30	1.69 (1.56–1.84)	<.0001	1.38 (1.26–1.51)	<.0001
Parity				
1 vs. ≥ 2	1.85 (1.70–2.02)	<.0001	1.37 (1.24–1.50)	<.0001
Gestational age				
Preterm vs. Term	6.15 (5.65–6.70)	<.0001	3.40 (3.09–3.75)	<.0001
Gestational number				
Multiple vs. Singleton	7.12 (6.35–7.99)	<.0001	2.54 (2.24–2.89)	<.0001
Cesarean section				
Yes vs. No	2.92 (2.68–3.18)	<.0001	1.71 (1.56–1.88)	<.0001

PIH, pregnancy-induced hypertension; OR, odds ratio; CI, confidence interval.
[a] OR is adjusted for group differences in age, parity, gestational age, gestational number, caesarean section, diabetes mellitus, hypertension, dyslipidemia, coronary artery disease, chronic obstructive pulmonary disease, chronic kidney disease, and cerebrovascular disease.

induced by PIH, could exist in the fetal circulatory system and may be involved in the pathogenesis of TTN. However, more studies are needed to confirm these results.

The current study showed that preterm birth, cesarean section, and multiple births were independent risk factors for the development of TTN. Previous epidemiological studies have indicated that low gestational age, low birth weight, and cesarean section are well-established risk factors for TTN [19,36,49,50]. It has been suggested that fetal lung fluid clearance does not occur in prematurely delivered infants or in infants who are delivered via cesarean section before the onset of labor [51,52]. Immature epithelial Na + channel expression, which causes defective Na + transport, may be a key reason for the development of TTN in these two types of infants [37,53,54]. An Italian study by Dani et al. showed that twin pregnancy was also a risk factor for TTN [36]. In most multiple births, infants are delivered by cesarean section at a preterm gestational age. It therefore makes sense that mothers who give birth to multiples have infants who are at an increased risk of developing TTN.

This study had a longitudinal, large population-based design. Nevertheless, several limitations inherent to the use of insurance claims databases must be taken into account. One limitation is that the diagnosis of PIH in the NHIRD was based on the ICD-9 codes. Data on patients' blood pressure, proteinuria, and symptoms were not available in the database. Another limitation is that many demographic variables were not available in the database, such as body mass index, smoking status, lifestyle, socioeconomic status, and family medical history. These factors would have been valuable for assessing other factors that may be associated with PIH and TTN. A final limitation is that the diagnostic criteria for PIH have changed over time, which could result in heterogeneous patient populations across studies and may restrict comparisons. Regardless of these limitations, our study was based on a nationwide, population-based database that included nearly all of Taiwan's residents. The large sample size in our study contributed to its substantial statistical power and revealed an obvious association between PIH and TTN with minimal selection biases.

In conclusion, the current study demonstrated that maternal PIH increased the subsequent risk for TTN. In addition, age $\geq$30 years, primiparity, preterm birth, multiple births, and cesarean section were shown to be independent risk factors for TTN.

Ethics approval and consent to participate

The Kaohsiung Veterans General Hospital (VGHKS15-EM4–01) Institutional Review Board approved this study.

Conflict of interest

The authors declare that they have no conflict of interest.

Acknowledgements

We thank the Economy Company Ltd. for their technical and English editing services. We are grateful for the use of the data from the National Health Insurance Research Database that was provided by the Statistics Center of the Department of Health and Welfare. The interpretations and conclusions contained herein do not represent those of the Bureau of Health Promotion, Taiwan. This study is partly supported by grants from the Ministry of Science and Technology, Executive Yuan (MOST 101-2314-B-010-038-MY3, and MOST 106-2314-B-075-061-MY3), and Taipei Veterans General Hospital (V104C-095; V105C-096; V106C-129, V106D23-001-MY2-1, and V106A-012).

References

[1] Say L, Chou D, Gemmill A, Tuncalp O, Moller AB, Daniels J, et al. Global causes of maternal death: a WHO systematic analysis. Lancet Global Health 2014;2:e323–33.
[2] Lin LT, Wang PH, Tsui KH, Cheng JT, Cheng JS, Huang WC, et al. Increased risk of systemic lupus erythematosus in pregnancy-induced hypertension: a nationwide population-based retrospective cohort study. Medicine (Baltimore) 2016;95:e4407.
[3] Muto H, Yamamoto R, Ishii K, Kakubari R, Takaoka S, Mabuchi A, et al. Risk assessment of hypertensive disorders in pregnancy with maternal characteristics in early gestation: a single-center cohort study. Taiwan J Obstet Gynecol 2016;55:341–5.
[4] Pan ML, Chen LR, Tsao HM, Chen KH. Risk of gestational hypertension-preeclampsia in women with preceding endometriosis: a nationwide population-based study. PLoS One 2017;12:e0181261.
[5] Zhu L, Baczyk D, Lye SJ, Zhang Z. Preeclampsia is associated with low placental transthyretin levels. Taiwan J Obstet Gynecol 2016;55:385–9.
[6] Lin LT, Hu LY, Tang PL, Tsui KH, Cheng JT, Huang WC, et al. Do racial differences exist in the association between pregnancy-induced hypertension and breast cancer risk? Hypertens Pregnancy 2017;36:138–44.
[7] Lisonkova S, Joseph KS. Incidence of preeclampsia: risk factors and outcomes associated with early- versus late-onset disease. Am J Obstet Gynecol 2013;209. 544 e1–e12.
[8] Basso O, Rasmussen S, Weinberg CR, Wilcox AJ, Irgens LM, Skjaerven R. Trends in fetal and infant survival following preeclampsia. J Am Med Assoc 2006;296:1357–62.
[9] Wang PH, Yang MJ, Chen CY, Chao HT. Endothelial cell dysfunction and pre-eclampsia. J Chin Med Assoc 2015;78:321–2.
[10] Hung TH, Hsieh TT, Chen SF. Risk of abnormal fetal growth in women with early- and late-onselt preeclampsia. Pregnancy Hypertens 2018;12:201–6.
[11] Steegers EA, von Dadelszen P, Duvekot JJ, Pijnenborg R. Pre-eclampsia. Lancet 2010;376:631–44.
[12] Magee LA, Pels A, Helewa M, Rey E, von Dadelszen P. Canadian hypertensive disorders of pregnancy working G. diagnosis, evaluation, and management of the hypertensive disorders of pregnancy. Pregnancy Hypertens 2014;4:105–45.
[13] Wang PH, Yeh CC, Chen YJ, Chen CP. Maternal serum markers and pre-eclampsia. Taiwan J Obstet Gynecol 2015;54:339–40.
[14] Chen KH, Seow KM, Chen LR. Progression of gestational hypertension to pre-eclampsia: a cohort study of 20,103 pregnancies. Pregnancy Hypertens 2017;10:230–7.
[15] Wang PH, Lee WL, Yang YH, Chen YJ, Tsai YC, Yuan CC. Alpha 2,6-sialyltransferase I expression in the placenta of patients with preeclampsia. J Chin Med Assoc 2007;70:152–8.
[16] Chaiworapongsa T, Chaemsaithong P, Yeo L, Romero R. Pre-eclampsia part 1: current understanding of its pathophysiology. Nat Rev Nephrol 2014;10:466–80.
[17] Warrington JP, George EM, Palei AC, Spradley FT, Granger JP. Recent advances in the understanding of the pathophysiology of preeclampsia. Hypertension 2013;62:666–73.
[18] Cheng MH, Wang PH. Placentation abnormalities in the pathophysiology of preeclampsia. Expert Rev Mol Diagn 2009;9:37–49.
[19] Tutdibi E, Gries K, Bucheler M, Misselwitz B, Schlosser RL, Gortner L. Impact of labor on outcomes in transient tachypnea of the newborn: population-based study. Pediatrics 2010;125:e577–83.
[20] Takaya A, Igarashi M, Nakajima M, Miyake H, Shima Y, Suzuki S. Risk factors for transient tachypnea of the newborn in infants delivered vaginally at 37 weeks or later. J Nippon Med Sch 2008;75:269–73.
[21] Bardou O, Prive A, Migneault F, Roy-Camille K, Dagenais A, Berthiaume Y, et al. K+ channels regulate ENaC expression via changes in promoter activity and control fluid clearance in alveolar epithelial cells. BBA 2012;1818:1682–90.
[22] Hummler E, Barker P, Beermann F, Gatzy J, Verdumo C, Boucher R, et al. Role of the epithelial sodium channel in lung liquid clearance. Chest 1997;111:113s.
[23] Ito Y, Marumo F, Ando K, Hayashi M, Yamashita F. The physiological and biological significances of human atrial natriuretic peptide in neonates. Acta Paediatr Scand 1990;79:26–31.
[24] Marquis M, Fenrick R, Pedro L, Bouvier M, De Lean A. Comparative binding study of rat natriuretic peptide receptor-A. Mol Cell Biochem 1999;194:23–30.
[25] Aydemir O, Aydemir C, Sarikabadayi YU, Altug N, Erdeve O, Uras N, et al. The role of plasma N-terminal pro-B-type natriuretic peptide in predicting the severity of transient tachypnea of the newborn. Early Hum Dev 2012;88:315–9.
[26] Moghbeli N, Srinivas SK, Bastek J, Lu Y, Putt ME, Cappola TP, et al. N-terminal pro-brain natriuretic Peptide as a biomarker for hypertensive disorders of pregnancy. Am J Perinatol 2010;27:313–9.
[27] Afshani N, Moustaqim-Barrette A, Biccard BM, Rodseth RN, Dyer RA. Utility of B-type natriuretic peptides in preeclampsia: a systematic review. Int J Obstet Anesth 2013;22:96–103.
[28] Lai JC, Chen HH, Weng CS, Chou YJ, Huang N, Wen SY, et al. The characterization of trachelectomy for benign and precancerous indications in Taiwan: a

population-based study, 1998-2013. Taiwan J Obstet Gynecol 2017;56:495–501.
[29] Lai JC, Chen HH, Chu KH, Weng CS, Chou YJ, Huang N, et al. Nationwide trends and in-hospital complications of trachelectomy for surgically resectable cervical cancer in Taiwanese women: a population-based study, 1998–2013. Taiwan J Obstet Gynecol 2017;56:449–55.
[30] Huang CC, Ho CH, Chen YC, Lin HJ, Hsu CC, Wang JJ, et al. Increased risk for diabetes mellitus in patients with carbon monoxide poisoning. Oncotarget 2017;8:63680–90.
[31] Chang WT, Leu HI, Chen HP, Lin MH, Chen TJ, Hwang SJ, et al. Temporal availability of obstetrics and gynecology clinics in Taiwan: a nationwide survey. Taiwan J Obstet Gynecol 2017;56:636–41.
[32] Wang KC, Chang WH, Lee WL, Huang N, Huang HY, Yen MS, et al. An increased risk of epithelial ovarian cancer in Taiwanese women with a new surgicopathological diagnosis of endometriosis. BMC Cancer 2014;14:831.
[33] Chang WH, Wang KC, Lee WL, Huang N, Chou YJ, Feng RC, et al. Endometriosis and the subsequent risk of epithelial ovarian cancer. Taiwan J Obstet Gynecol 2014;53:530–5.
[34] Lee WL, Chang WH, Wang KC, Guo CY, Chou YJ, Huang N, et al. The risk of epithelial ovarian cancer of women with endometriosis may be varied greatly if diagnostic criteria are different: a nationwide population-based cohort study. Medicine (Baltimore) 2015;94:e1633.
[35] Huang BS, Chang WH, Wang KC, Huang N, Guo CY, Chou YJ, et al. Endometriosis might be inversely associated with developing chronic kidney disease: a population-based cohort study in Taiwan. Int J Mol Sci 2016;17, E1079.
[36] Dani C, Reali MF, Bertini G, Wiechmann L, Spagnolo A, Tangucci M, et al. Risk factors for the development of respiratory distress syndrome and transient tachypnoea in newborn infants. Italian Group of Neonatal Pneumology. Eur Respir J 1999;14:155–9.
[37] O'Brodovich HM. Immature epithelial Na+ channel expression is one of the pathogenetic mechanisms leading to human neonatal respiratory distress syndrome. Proc Assoc Am Phys 1996;108:345–55.
[38] Woodard GE, Rosado JA. Natriuretic peptides in vascular physiology and pathology. Int Rev Cell Mol Biol 2008;268:59–93.
[39] Weber M, Hamm C. Role of B-type natriuretic peptide (BNP) and NT-proBNP in clinical routine. Heart 2006;92:843–9.
[40] Kale A, Kale E, Yalinkaya A, Akdeniz N, Canoruc N. The comparison of amino-terminal probrain natriuretic peptide levels in preeclampsia and normotensive pregnancy. J Perinat Med 2005;33:121–4.
[41] Seong WJ, Kim SC, Hong DG, Koo TB, Park IS. Amino-terminal pro-brain natriuretic peptide levels in hypertensive disorders complicating pregnancy. Hypertens Pregnancy 2011;30:287–94.
[42] Szabo G, Molvarec A, Nagy B, Rigo Jr J. Increased B-type natriuretic peptide levels in early-onset versus late-onset preeclampsia. Clin Chem Lab Med 2014;52:281–8.
[43] Junus K, Wikstrom AK, Larsson A, Olovsson M. Placental expression of proBNP/NT-proBNP and plasma levels of NT-proBNP in early- and late-onset preeclampsia. Am J Hypertens 2014;27:1225–30.
[44] Machado LU, Fiori HH, Baldisserotto M, Ramos Garcia PC, Vieira AC, Fiori RM. Surfactant deficiency in transient tachypnea of the newborn. J Pediatr 2011;159:750–4.
[45] Negi R, Pande D, Karki K, Kumar A, Khanna RS, Khanna HD. Association of oxidative DNA damage, protein oxidation and antioxidant function with oxidative stress induced cellular injury in pre-eclamptic/eclamptic mothers during fetal circulation. Chem Biol Interact 2014;208:77–83.
[46] Rodriguez-Capote K, Manzanares D, Haines T, Possmayer F. Reactive oxygen species inactivation of surfactant involves structural and functional alterations to surfactant proteins SP-B and SP-C. Biophys J 2006;90:2808–21.
[47] Andersson S, Kheiter A, Merritt TA. Oxidative inactivation of surfactants. Lung 1999;177:179–89.
[48] Badran EF, Abdalgani MM, Al-Lawama MA, Al-Ammouri IA, Basha AS, Al Kazaleh FA, et al. Effects of perinatal risk factors on common neonatal respiratory morbidities beyond 36 weeks of gestation. Saudi Med J 2012;33:1317–23.
[49] Riskin A, Abend-Weinger M, Riskin-Mashiah S, Kugelman A, Bader D. Cesarean section, gestational age, and transient tachypnea of the newborn: timing is the key. Am J Perinatol 2005;22:377–82.
[50] Visruthan NK, Agarwal P, Sriram B, Rajadurai VS. Neonatal outcome of the late preterm infant (34 to 36 weeks): the Singapore story. Ann Acad Med Singapore 2015;44:235–43.
[51] Ramachandrappa A, Jain L. Elective cesarean section: its impact on neonatal respiratory outcome. Clin Perinatol 2008;35:373–93.
[52] Lines A, Hooper SB, Harding R. Lung liquid production rates and volumes do not decrease before labor in healthy fetal sheep. J Appl Physiol 1997;82:927–32.
[53] Olver RE, Walters DV, M Wilson S. Developmental regulation of lung liquid transport. Annu Rev Physiol 2004;66:77–101.
[54] Helve O, Janer C, Pitkanen O, Andersson S. Expression of the epithelial sodium channel in airway epithelium of newborn infants depends on gestational age. Pediatrics 2007;120:1311–6.