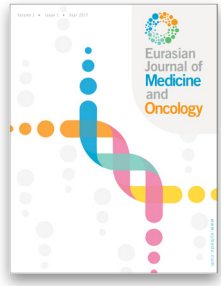
EJMO

DOI: 10.14744/ejmo.2017.03522
EJMO 2018;2(1):27-31



Research Article

Influence of Hospitalization-Requiring Gastroenteritis in Pregnancy on Perinatal Outcome

Zeynep Ozturk Inal,[1] Hakan Timur,[2] Hasan Ali Inal,[1] Burak Ersak,[2] Burcu Timur,[3] Dilek Uygur[2]

[1]Department of Obstetric and Gynecology, Konya Training and Research Hospital, Konya, Turkey
[2]Departmant of Perinatology, Zekai Tahir Burak Women's Health Training and Research Hospital, Ankara, Turkey
[3]Department of Obstetric and Gynecology, Etlik Zubeyde Hanim Women's Health Training and Research Hospital, Ankara, Turkey

Abstract

Objectives: To evaluate the clinical and perinatal outcomes of pregnant women who require hospitalization due to acute gastroenteritis.

Methods: Clinical and perinatal outcomes of 108 pregnant women who required hospitalization due to acute gastroenteritis in Maternal and Fetal Medicine Unit and 110 healthy pregnant women who were admitted to Dr. Zekai Tahir Burak Woman's Health Education and Research Hospital between January 2014 and March 2015 were evaluated.

Results: No statistically significant difference was observed between the groups with respect to the age, educational status, body mass index, gravida, parity, number of abortions, smoking status, history of preterm delivery, consumption of dairy products and coffee, systolic and diastolic blood pressures, white blood cell count, type of delivery, and sex (p>0.05). Although higher fast food consumption (31.8% vs 45.4%, p=0.040), well and tap water consumption (0.9% vs 7.4% and 12.7% vs 37.0% p=0.001), preterm labor (8.2% vs 24.1%, p=0.003), preterm premature rupture of membranes (5.5% vs 14.8%, p=0.025), elevated serum C-reactive protein values (4.20+0.89 mg/L vs 11.73+9.64 mg/L, p=0.001), and admission to newborn intensive care unit (9.2% vs 24.5%, p=0.005) were observed in the gastroenteritis group, lower gestational week (38.29+1.22 vs 37.33+2.81, p=0.001) and birth weight (3475.82+320.34 g vs 3285.65+588.44 g, p=0.004) were observed.

Conclusion: Gastroenteritis, which requires hospitalization during pregnancy, may lead to preterm delivery and low birth weight. Prospective studies are needed to confirm our results.

Keywords: Gastroenteritis, perinatal outcome, pregnancy

Cite This Article: *Ozturk Inal Z, Timur H, Inal H, Ersak B, Timur B, Uygur D. Influence of Hospitalization-Requiring Gastroenteritis in Pregnancy on Perinatal Outcome. EJMO. 2018; 2(1): 27-31*

Gastrointestinal symptoms such as nausea and vomiting are quite frequent in pregnancy. Gastroenteritis has started to occur more frequently during pregnancy in recent times. Prevalence of acute infections is reported to be 3.5%–5% during pregnancy.[1] Acute infections such as gastroenteritis in pregnancy may lead to abortion, preterm labor, preterm delivery, and even stillbirth.[2, 3] Epidemiological studies have reported that infections observed during pregnancy may lead to cardiovascular diseases, myocardial infarction, preeclampsia, and even stroke.[4, 5]

Limited amounts of systemic inflammatory markers such as prostaglandin E2, tumor necrosis factor alpha, and interleukin 1 and 6 enable the intrauterine development of fetus by contributing to vascular remodeling, which is necessary for placental invasion in the early stages of pregnancy. Vascular remodeling is completed in the 20th gestational week, at which the placenta reaches its full size. Limited amount of systemic inflammatory markers begin to decrease in cir-

Address for correspondence: Zeynep Ozturk Inal, MD. Konya Egitim ve Arastirma Hastanesi, Kadin Hastaliklari ve Dogum Anabilim Dali, Konya, Turkey
Phone: +90 332 323 67 09 - 51 06 **E-mail:** zeynephafiza@gmail.com
Submitted Date: June 24, 2017 **Accepted Date:** October 26, 2017 **Available Online Date:** December 05, 2017

culation.[6-8] The amount of these markers increases again during labor, and this increase aids cervical maturation and uterine contractility.[9, 10] Elevated inflammatory marker amounts due to infections, except during the delivery process, may lead to some negative perinatal outcomes.[11, 12]

Low birth weight babies may be encountered as the result of malabsorption, similar to that observed in cases of inflammatory bowel diseases (such as Crohn's disease), if gastroenteritis in pregnancy becomes chronic.

Although acute bacterial gastroenteritis is most commonly caused by Escherichia coli, followed by Shigella sp., Salmonella sp., Campylobacter sp., Enterohemorrhagic E. coli (EHEC), Vibrio sp., and Listeria monocytogenes, acute viral gastroenteritis is most commonly caused by rotavirus and adenovirus in children below 2 years of age. Giardia intestinalis, Cryptosporidium sp., Entamoeba histolytica, and Dientamoeba fragilis are the common causes of acute parasitic gastroenteritis.[13]

In this study, we aimed to retrospectively evaluate the clinical and perinatal outcomes of the patients who were admitted to the Maternal and Fetal Medicine Unit of our hospital due to pregnancy and acute gastroenteritis.

Methods

Clinical and perinatal outcomes of 108 pregnant women who required hospitalization due to acute gastroenteritis in Maternal and Fetal Medicine Unit and 110 healthy pregnant women who did not have a history of gastroenteritis and risky pregnancy (i.e., without preeclampsia, gestational diabetes, preterm labor, etc.) were admitted to Dr. Zekai Tahir Burak Woman's Health Education and Research Hospital, Ankara, Turkey between January 2014 and March 2015 were evaluated. Patients with inflammatory bowel diseases such as ulcerative colitis and Crohn's disease, celiac disease, lactose intolerance, or cow's milk allergy were excluded from the study. Stool cultures were obtained from pregnant women with gastroenteritis. Clinical and perinatal outcomes of the patients were evaluated after obtaining the approval of the Ethics Committee of our hospital. The groups were compared with respect to the age, educational status, body mass index (BMI), gravida, parity, number of abortions, smoking status, history of preterm delivery, milk and dairy product consumption, coffee consumption, prepared food consumption, drinking water source, systolic and diastolic blood pressures, serum white blood cell count, delivery type, sex of the baby, serum C-reactive protein (CRP) value, admission to newborn intensive care unit, gestational week, and birth weight.

Statistical analysis was performed using the Statistical Package for Social Sciences 15 (SPSS Inc.) software and Excel program. Kolmogorov–Smirnov test was performed for the detection of continuous variables normally and not

Table 1. Demographic characteristics of the participants

	Control (110) (%)	Gastroenteritis (108) (%)	p
Age (years)	30.33+3.96	29.93+5.84	0.593
BMI (kg/m^2)	25.95+3.75	26.07+3.23	0.864
Number of pregnancies	2 (1-6)	1 (1-6)	0.224
Number of live births	0 (0-3)	0 (0-3)	0.306
Number of spontaneous abortion	0 (0-3)	0 (0-4)	0.379
Smoking rate	10 (9.1)	7 (6.5)	0.615
History of a previous preterm delivery	7 (6.4)	14 (13)	0.096
Prepared food consumption	35 (31.8)	49 (45.4)	0.040*
Milk and dairy product consumption	94 (87)	99 (90)	0.530
Educational status			
Illiterate	4 (3.6)	4 (3.7)	0.765
Primary-Secondary school	87 (79.1)	84 (77.8)	
High school	16 (14.5)	16 (14.8)	
University	3 (2.7)	4 (3.7)	
Drinking water supply			
Well water	1 (0.9)	8 (7.4)	0.001*
Tap water	14 (12.7)	40 (37.0)	
Purified water	45 (40.9)	25 (23.1)	
Natural spring water	50 (45.5)	35 (32.4)	

BMI: body mass index; *Statistically significant.

normally distributed. Results were given as mean±standard deviation using independent samples tests for normally distributed data; results were given as median (min–max) using Mann–Whitney U test for not normally distributed data. Pearson chi-square or Fisher Exact test was used for categorical variables. A p value of <0.05 was accepted as statistically significant.

Results

Overall, 108 patients were hospitalized due to pregnancy and gastroenteritis in the course of this retrospective study. A statistically significant difference was not detected between the control (110 patients) and gastroenteritis groups with respect to the age, educational status, BMI, parity, gravida, number of abortions, smoking status, history of a previous preterm delivery, milk and dairy product consumption, and coffee consumption. Prepared food consumption (31.8% vs 45.4%, p=0.040) and well and tap water consumption (0.9% vs 7.4% and 12.7% vs 37.0% p=0.001) were detected to be higher in the gastroenteritis group (Table 1). Systolic and diastolic blood pressures, serum white blood cell count, delivery type, and sex were similar between the groups.

The daily stool discharge (1.13±0.33 vs 9.04±2.71, p=0.001), serum CRP value (4.20±0.89 vs 11.73±9.64, p=0.001), preterm labor rate (8.2% vs 24.1%, p=0.003), preterm premature rupture of membrane rate (5.5% vs 14.8%, p=0.025), and admission to newborn intensive care unit rate (9.2% vs 24.5%, p=0.005) were statistically significantly higher in the acute gastroenteritis group. However, the gestational week (38.29±1.22 vs 37.33±2.81, p=0.001) and birth weight (3475.82±320.34 g vs 3285.65±588.44 g, p=0.004) were statistically significantly lower in the gastroenteritis group. There was no difference in the preeclampsia rate (p=0.330). Although the stool culture was positive in 6 (5.5%) patients in the gastroenteritis group (E. histolytica in 5 patients and S. enterica in 1 patient), occult blood test was positive in 4 (3.7%) patients (Table 2).

Discussion

In this retrospective study, we found that preterm labor was higher in the gastroenteritis group consistent with previous literatures. Gastroenteritis in pregnancy could lead to preterm delivery. In a study evaluating approximately 10.000 deliveries retrospectively, it was detected that gastroenteritis could be encountered in one of each three women during pregnancy and could be seen equally in every trimester.[14] In that study, gastroenteritis was detected more frequently in younger pregnant women, women who met negative life events at least once during their life,

Table 2. Clinic and laboratory outcomes of the groups

	Control (110) (%)	Gastroenteritis (108) (%)	p
Daily stool discharge	1.13+0.33	9.04+2.71	0.001*
Systolic blood pressure (mmHg)	103.23+11.66	104.03+11.01	0.603
Diastolic blood pressure (mmHg)	65.77+6.84	64.69+6.60	0.432
Body temperature (ºC)	36.90+0.63	36.93+0.59	0.714
WBC (mm^3)	11.815.73+3.238.61	12.056.39+3.143.71	0.578
CRP (mg/L)	4.20+0.89	11.73+9.64	0.001*
Gestational age at delivery	38.29+1.22	37.33+2.81	0.001*
Preterm labor (%)	9 (8.2)	26 (24.1)	0.003*
Preterm premature rupture of membranes (%)	6 (5.5)	16 (14.8)	0.025*
Preeclampsia (%)	3 (2.7)	6 (5.6)	0.330
Birth weight (g)	3.475.82+320.34	3.285.65+588.44	0.004*
Delivery type			
Normal spontaneous delivery rate	64 (58.2)	46 (41.8)	0.196
C/S rate	72 (66.7)	36 (33.3)	
Sex			
Female	54 (49.1)	50 (46.3)	0.680
Male	56 (50.9)	58 (53.7)	
NICU	10 (9.2)	26 (24.5)	0.005*
Stool culture positive	-	6 (5.5)	0.014*
Fecal occult blood positive	-	4 (3.7)	0.017*

WBC: White blood cell; CRP: C-reactive protein; C/S: Cesarean section; NICU: Neonatal intensive care unit; *Statistically significant.

and hospital staff. Young women were also shown to be at a higher risk for gastroenteritis in another study. This was possibly because young women are contaminated from their younger children compared with those of older women.[15]

Contamination with rotavirus and other gastrointestinal pathogens occurs through contaminated hands and food and also inter-personal contact within family.[16] Gastroenteritis was reported to be more common among crowded families.[15]

Diagnosis of gastroenteritis is mostly based on its signs and symptoms. Slutsker et al.[17] could not detect any pathogen in 91.6% of stool specimens of 30.000 patients who were admitted with diagnosis of gastroenteritis. Growing could be detected in only 5.5% of our patients, similar to that in the above study. Mc Carthy et al.[15] reported that only 2% of the gastroenteritis patients received medical care. Unfortunately, we cannot report a ratio of medical care in pregnant women.

In a study from Sweden, cause of gastroenteritis was detected to be mostly of bacterial origin (Campylobacter sp., Clostridium difficile, enterotoxigenic E. coli, and calicivirus). Viral pathogens were detected to be most frequent among pediatric age group, mainly rotavirus.[18]

Infectious gastroenteritis should be discriminated from urinary tract infections, acute appendicitis, and prostaglandin-related increased bowel movements in pregnancy. Prostaglandins may increase bowel movements through stimulating smooth muscles, and this is quite common in pregnancy.[19] Prostaglandin-related nausea and vomiting also increase in pregnancy; however, this is most commonly seen during the first trimester.[20]

Although Ludvigsson et al.[14] included all pregnant women and gastroenteritis cases in their study regardless of hospitalization, we included only the pregnant women who were hospitalized due to gastroenteritis. They reported that gastroenteritis in months 4, 5, and 7 shortens the duration of pregnancy; infections seen in the other months did not influence the duration of pregnancy or affect overall perinatal outcome. We observed that preterm delivery occurred, and more infants were admitted to the newborn intensive care unit.

Inflammatory bowel diseases such as Crohn's disease and ulcerative colitis were shown to cause preterm delivery and intrauterine growth retardation.[21] However, hospitalization-requiring gastroenteritis can also lead to preterm delivery similar to inflammatory bowel diseases.

We recommend that prepared food consumption and well and tap water consumption should be avoided for the prevention of gastroenteritis. Pregnant women with gastroenteritis should be hospitalized, and parenteral supportive therapy must be provided; they must also be warned regarding preterm birth.

In conclusion, it should be kept in mind that hospitalization-requiring gastroenteritis could lead to preterm delivery. Prospective studies are needed to confirm our results.

Disclosures

Ethics Committee Approval: The study was approved by the Local Ethics Committee.

Peer-review: Externally peer-reviewed.

Conflict of Interest: The authors have no disclosures and no conflict of interest to declare.

Authorship contributions: Concept – Z.O.I., H.T.; Design – H.A.I., B.E.; Supervision – H.T., B.T., D.U.; Materials – H.T.; Data collection &/or processing – Z.O.I., H.T.; Analysis and/or interpretation – H.A.I.; Literature search – Z.O.I, H.A.I.; Writing – H.A.I.; Critical review – Z.O.I., H.A.I.

References

1. Lain SJ, Roberts CL, Warning J, Vivian-Taylor J, Ford JB. A survey of acute self-reported infections in pregnancy. BMJ Open 2011;1:e000083. [CrossRef]
2. Brocklehurst P. Infection and preterm delivery. BMJ 1999;318:548–9. [CrossRef]
3. Goldenberg RL, Hauth JC, Andrews WW. Intrauterine infection and preterm delivery. N Engl J Med 2000;342:1500–7. [CrossRef]
4. Conde-Agudelo A, Villar J, Lindheimer M. Maternal infection and risk of preeclampsia: systematic review and metaanalysis. Am J Obstet Gynecol 2008;198:7–22. [CrossRef]
5. Smeeth L, Thomas SL, Hall AJ, Hubbard R, Farrington P, Vallance P. Risk of myocardial infarction and stroke after acute infection or vaccination. N Engl J Med 2004;351:2611–8.
6. Elenkov IJ, Wilder RL, Bakalov VK, Link AA, Dimitrov MA, Fisher S, et al. IL-12, TNF-alpha, and hormonal changes during late pregnancy and early postpartum: implications for autoimmune disease activity during these times. J Clin Endocrinol Metab 2001;86:4933–8. [CrossRef]
7. Luppi P, Haluszczak C, Betters D, Richard CA, Trucco M, DeLoia JA. Monocytes are progressively activated in the circulation of pregnant women. J Leukoc Biol 2002;72:874–84.
8. McCracken SA, Hadfield K, Rahimi Z, Gallery ED, Morris JM. NF-kappaB-regulated suppression of T-bet in T cells represses Th1 immune responses in pregnancy. Eur J Immunol 2007;37:1386–96. [CrossRef]
9. Romero R, Munoz H, Gomez R, Parra M, Polanco M, Valverde V, et al. Increase in prostaglandin bioavailability precedes the onset of human parturition. Prostaglandins Leukot Essent Fatty Acids 1996;54:187–91. [CrossRef]
10. Yuan M, Jordan F, McInnes IB, Harnett MM, Norman JE. Leukocytes are primed in peripheral blood for activation during term and preterm labour. Mol Hum Reprod 2009;15:713–24.

11. Redman CW, Sargent IL. Latest advances in understanding preeclampsia. Science 2005;308:1592–4. [CrossRef]
12. von Dadelszen P, Magee LA. Could an infectious trigger explain the differential maternal response to the shared placental pathology of preeclampsia and normotensive intrauterine growth restriction? Acta Obstet Gynecol Scand 2002;81:642–8.
13. TC Sağlık Bakanlığı. Bulaşıcı Hastalıkların İhbarı ve Bildirim Sistemi: Standart Tanı, Sürveyans ve Laboratuvar Rehberi. Ankara; 2004. Available at: https://dosyasb.saglik.gov.tr/Eklenti/1442,bhastaliktedavipdf.pdf?0. Accessed Nov 28, 2017.
14. Ludvigsson JF. Effect of gastroenteritis during pregnancy on neonatal outcome. Eur J Clin Microbiol Infect Dis 2001;20:843–9.
15. McCarthy N, de Jong B, Ziese T, Sjölund R, Hjalt CA, Giesecke J. Epidemiological explanation of an outbreak of gastro-enteritis in Sweden in the absence of detailed microbiological information. Eur J Epidemiol 1998;14:711–8. [CrossRef]
16. Dennehy PH. Transmission of rotavirus and other enteric pathogens in the home. Pediatr Infect Dis J 2000;19:S103–5.
17. Slutsker L, Ries AA, Greene KD, Wells JG, Hutwagner L, Griffin PM. Escherichia coli O157:H7 diarrhea in the United States: clinical and epidemiologic features. Ann Intern Med 1997;126:505–13. [CrossRef]
18. Johansen K, Bennet R, Bondesson K, Eriksson M, Hedlund KO, De Verdier Klingenberg K, et al. Incidence and estimates of the disease burden of rotavirus in Sweden. Acta Paediatr Suppl 1999;88:20–3. [CrossRef]
19. Bonapace ES Jr, Fisher RS. Constipation and diarrhea in pregnancy. Gastroenterol Clin North Am 1998;27:197–211. [CrossRef]
20. Broussard CN, Richter JE. Nausea and vomiting of pregnancy. Gastroenterol Clin North Am 1998;27:123–51. [CrossRef]
21. Baird DD, Narendranathan M, Sandler RS. Increased risk of preterm birth for women with inflammatory bowel disease. Gastroenterology 1990;99:987–94. [CrossRef]