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Risk factors of placenta previa: a population based study and the review of the literature

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Abstract
The aim of our study is to provide a population-based risk factor profile for placenta previa and thereby enable early diagnosis plus the necessary management for this risky group. Methods This research was a retrospective case control study including 25105 pregnancies in the period January 2000- December 2010. The data of 25105 pregnancies were examined on the hospital database. A total of 139 single pregnancies complicated by placenta previa were compared with a randomly selected control group of 1200 single pregnancies. Evaluation was made of risk factors for placenta previa. Results Of the 25105 pregnancies presenting at our clinic during the 10-year period, placenta previa was determined in 139, giving an incidence rate of 0.55%. In the comparison of the two groups; advanced maternal age, multiparity, a history of caesarean section, a history of abortion or uterine curettage, cigarette smoking and male gender were found to be statistically significant as a risk factor for placenta previa. Conclusions The knowledge of the predisposing obstetric factors in respect of the development of placenta previa on a population basis provides the possibility of a careful approach on an individual patient basis.

Keywords: Antepartum bleeding, placental pathology, placenta previa, risk factors

Introduction

Placenta previa is an obstetric complication in which the placenta is inserted partially or wholly in the lower uterine segment. It is a major cause of antepartum haemorrhage [1]. Placenta previa is known to cause complications such as abnormal presentation, intrauterine growth restriction, prematurity and fetal death. Increased rates of peripartum bleeding, disseminated intravascular coagulation, hypovolemic shock, caesarean section operation, hysterectomy and maternal mortality have been reported in placenta previa [2-4].

The incidence of placenta previa and complicated pregnancy varies according to the population and has been reported as approximately 0.3%-0.8% [5-7]. While the etiology of placenta previa has not been fully clarified, related risk factors are known to be a history of caesarean section, advanced maternal age, multiparity, history of abortion, cigarette smoking and male gender fetus [5, 6].

Several studies have researched the risk factors for placenta previa and therefore the aim of our study was to provide a population-based risk factor profile and thereby enable early diagnosis and the necessary management for this risky group.

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Material and Methods

This research was a retrospective case control study including 25105 pregnancies in the period January 2000-December 2010. The data of 25105 pregnancies were examined on the hospital database and placenta previa was determined in 144 patients. Gestational age was determined from the first day of the last menstrual period and was confirmed by ultrasonography applied at 8-12 weeks. The diagnosis of placenta previa was defined as placental tissue partially or fully covering the cervical os ultrasonographically and this was confirmed during caesarean section operation. A control group was randomly selected to include 1255 pregnancies with vaginal or caesarean delivery. The study protocol was subject to local ethics committee approval.

A total of 5 pregnancies were excluded from the placenta previa group due to multiple gestation. 55 patients from the control group were excluded because of multiple gestation and incomplete data.

A total of 139 single pregnancies complicated by placenta previa were compared with a randomly selected control group of 1200 single pregnancies. Evaluation was made of risk factors for placenta previa of maternal age, parity, cigarette smoking in pregnancy, working in pregnancy, a history of caesarean delivery, a history of abortion or uterine curettage, fetal gender, gestational diabetes, diabetes mellitus or gestational hypertensive disease.

Data analyses were performed using SPSS v.21.0 statistical software (SPSS Inc. IBM, Chicago, IL, USA). The Pearson Chi-square test was applied with the Exact method. To determine significant risk factors in the categorical data of the groups, the odds ratio was used. Categorical data were stated as number (n) and percentage (%). Data were examined at a 95% confidence interval and a value of $p<0.05$ was accepted as statistically significant.

Results

Of the 25105 pregnancies presenting at our clinic during the 10-year period, placenta previa was determined in 139, giving an incidence rate of 0.55%. Placenta previa totalis was determined in 53 (38.1%) cases, placenta previa marginalis in 37 (26.6%), and placenta previa partialis in 49 (35.2%). In 13 (9.3%) cases placenta accreata and in 10 (7.1%) cases, placenta increata were determined concomitant to placenta previa.

The pregnancies of the placenta previa cases were all delivered by caesarean section and in the control group, the rate of caesarean deliveries was 20% as 240 cases. The gestational week at birth was determined as 38+1.5 weeks (range, 34-40 weeks) in the control group and 34.8+ 4 weeks (range, 22-40 weeks)in the study group. Deliveries before the 30th gestational week were 10 in the placenta previa group and 0 in the control group, and between weeks 30-34 were 30 (21.5%) in the placenta previa group and 102 (8.5%) in the control group.

In the evaluation of advanced maternal age, both groups were subdivided into 2 groups as 15-34 years, and ≥ 35 years. The mean age of the placenta previa group was determined as 28.7+ 5.7 years (range, 16-42 years) and as 26.2+ 4.7 years (range, 15-38 years) in the control group. In the comparison of the two groups, advanced maternal age was found to be statistically significant as a risk factor for placenta previa. In cases aged ≥35 years, the risk of placenta previa was determined to be increased 2.67-fold ($p<0.001$, OR:2.67 [1.7-4.2]).

Multiparity, a history of caesarean section, a history of abortion or uterine curettage were found to be statistically significant as a risk factor ($p<0.001$, OR:3.32 [2.1-5.2], p:0.038, OR: 1.5 [1.04-2.22], $p<0.001$, OR:2.2 [1.5-3.1]; respectively).

Table 1. The results of the comparison between the study and control group

		Placenta previa(139)	Control(1200)	P Value	OR (%95 Cl)
		n(%)	n(%)		
Age	≥35	29 (20,9%)	108 (9,0%)	<0,001	2,67 (1,7-4,2)
	[15-34]	110 (79,1%)	1092 (91,0%)		
Parity	Multipar	113 (81,3%)	680 (56,7%)	<0,001	3,32 (2,1-5,2)
	Primipar	26 (18,7%)	520 (43,3%)		
Cigarette	Smoker	51 (36,7%)	104 (8,7%)	<0,001	6,1 (4,1-9,1)
	Non-smoker	88 (63,3%)	1096 (91,3%)		
Employment status	No	135 (97,1%)	1158 (96,5%)	0,812	1,2 (0,4-3,5)
	Yes	4 (2,9%)	42 (3,5%)		
Fetal gender	Male	85 (61,2%)	536 (44,7%)	<0,001	2 (1,4-2,8)
	Female	54 (38,8%)	664 (55,3%)		
Hypertensive diseases of pregnancy	Yes	9 (6,5%)	77 (6,4%)	1	1,01 (0,5-2,1)
	No	130 (93,5%)	1123 (93,6%)		
Abortion/ uterine curettage	0	81 (58,3%)	904 (75,3%)	<0,001	
	1	13 (9,4%)	132 (11,0%)		1,1 (0,6-2,03)
	2	18 (12,9%)	160 (13,3%)		1,3 (0,7-2,2)
	3	27 (19,4%)	4 (0,3%)		75,3 (25,7-220,6)
Abortion/uterine curettage	Yes	58 (41,7%)	296 (24,7%)	<0,001	2,2 (1,5-3,1)
	No	81 (58,3%)	904 (75,3%)		
Number of birth	0	26 (18,7%)	520 (43,3%)	<0,001	
	1	45 (32,4%)	391 (32,6%)		2,3 (1,4-3,8)
	2	36 (25,9%)	198 (16,5%)		3,6 (2,1-6,2)
	3	32 (23,0%)	91 (7,6%)		7 (4-12,4)
Parity	+	113 (81,3%)	680 (56,7%)	<0,001	3,3 (2,1-5,2)
	-	26 (18,7%)	520 (43,3%)		
Number of caesarean section	0	94 (67,6%)	912 (76,0%)	0,033	
	1	27 (19,4%)	192 (16,0%)		1,4 (0,9-2,2)
	2	10 (7,2%)	70 (5,8%)		1,4 (0,7-2,8)
	3	8 (5,8%)	26 (2,2%)		3 (1,3-6,8)
Caesarean section	Yes	45 (32,4%)	288 (24,0%)	0,038	1,5 (1,04-2,22)
	No	94 (67,6%)	912 (76,0%)		
Gestational diabetes/diabetes mellitus	Yes	8 (5,8%)	78 (6,5%)	0,856	0,9 (0,4-1,9)
	No	131 (94,2%)	1122 (93,5%)		

Pearson Chi-Square Test(Exact) - OR, Odds Ratio; Cl, confidence interval

The effect of cigarette smoking on placenta previa was investigated. In the study group, 51 cases (36.7%) had a history of cigarette smoking and in the control group, 104 (8.7%). When the two groups were compared, cigarette smoking was determined as a risk factor for placenta previa ($p<0.001$, OR:6.1 [4.1-9.1]).

Fetal gender distribution was investigated and male gender of the fetus was determined to be statistically significant as a risk factor for placenta previa ($p<0.001$, OR:2 [1.4-2.8]).

No statistically significant relationship was determined between the development of placenta previa and hypertensive diseases of pregnancy, gestational diabetes/diabetes mellitus or employment status.

The results of the comparison between the study and control group are shown in Table 1.

Discussion

In literature, the prevalence of placenta previa has been reported as 0.3%-0.8% [5-7]. In recent years, several studies have reported an increase in the prevalence of placenta previa. This increase has been thought to be related to the enhanced trend for caesarean deliveries and the possibilities for easier diagnosis of placenta previa by improved imaging modalities [7-9].

Advanced maternal age, which leads to several fetomaternal problems such as premature labour, pre-eclampsia, gestational diabetes mellitus and congenital anomalies, also causes an increase in the prevalence of placenta previa [10]. The mechanism by which advanced maternal age impairs the development of normal placenta is not clearly understood. In the pathophysiology, it has been suggested that as a result of atherosclerotic changes occurring in the uterine blood vessels at older ages, the blood supply is impaired and compensatory placental hypertrophy develops secondary to this impaired vascularisation [8,11]. In some studies, the increased likelihood of placenta previa has been associated with increased parity at an older maternal age and therefore an increase in previous uterine interventions [8,12]. In 3 cohort studies, advanced maternal age has been reported as the cause of a 1.2-2.7-fold increase in the incidence of placenta previa [13-15]. In the current study, advanced maternal age increased the likelihood of placenta previa 2.67-fold.

When parity is examined as a risk factor, atrophic endometrium changes after frequent births, short intervals between births and multiparity have been reported to have an effect on the development of placenta previa [5,8,11]. Tuzovic et al [9] stated the risk of placenta previa to be related to a history of 3 or more previous births and Abu Heija et al [11] stated it as 5 or more. In the current study, the risk of placenta previa was determined to be increased 3.3-fold in those with a history of previous births compared to those with no previous births and as the number of births increased, this was related to an increase in the risk of placenta previa. In multiparity, just as in the age risk, the underlying pathophysiology may be infarct and atherosclerotic changes in the uterus.

A previous caesarean section scar is known to cause pathological changes in endometrial and myometrial tissue. It is thought that this scar tissue prevents the migration of the placenta to the uterine fundus [5, 9, 11, 15]. In a study by Taylor et al [16], the placenta previa risk of women with a history of caesarean delivery was found to be increased 1.48-fold. In another study by Getahun et al [17], it was shown that after a first and second pregnancy had been concluded with a caesarean delivery, the risk of placenta previa in the third pregnancy increased by 50% . In the current study, the placenta previa risk was determined to increase 1.5-fold in those with a history of caesarean delivery, which was consistent with literature.

Uterine curettage applied after spontaneous or induced abortion forms a basis for the development of placenta previa by damaging the uterine cavity [18-20]. In a study by Zhang et al [12], the placenta previa risk was shown to be increased 1.6-fold in those with a history of 1 abortion, 2.3-fold in those with a history of 2, and 3.7-fold where there was a history of 3 or more abortions. In the current study, the risk of placenta previa was determined to increase 2.2-fold in those with a history of abortion or uterine curettage, which was consistent with the reports in literature.

Some studies have reported a 2.4-fold increased risk of placenta previa in patients who smoke cigarettes [14,21-23]. The placental growth in this situation can be explained by the vasoactive properties of nicotine found in cigarettes and chronic hypoxia associated with carbon monoxide [22,23]. In the current study, a strong correlation was found between cigarette smoking and the development of placenta previa.

The relationship between male gender fetus and placenta previa has been investigated in previous studies. Although the underlying causes have not been clarified, it has been suggested that there may be a relationship with maternal hormones or prematurity [24-26]. The findings of the current study are consistent with those in literature.

In employed women with placenta previa, there was determined to be a 2-fold increased risk of presentation with bleeding. This could be related having less time to rest. Although there is insufficient data related to socio-economic factors in respect of increased risk of placenta previa, in a study in which an increased risk of placenta previa was determined in working women, it was suggested that this could be related to an increased rate of dilatation and curettage in working women [27].

In conclusion, knowledge of the predisposing obstetric factors in respect of the development of placenta previa on a population basis, provides the possibility of a careful approach on an individual patient basis. Careful ultrasonographic examination for the identification of the placental location in 2nd trimester of high risk patients allows early diagnosis of placenta previa and thereby a reduction in maternal morbidity and mortality. The limitations of our study were the selection of patients from a single center and small sample size. In this study, the risk factors affecting the Turkish population were found to be correlated with other ethnic-associated studies but in respect of additional factors, there is a need for further multi-center, population-based studies with a larger patient group.

Dısclosure

We have no financial or commercial conflicts of interest.

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