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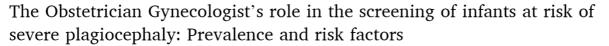
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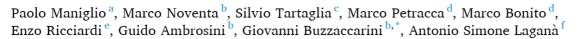
European Journal of Obstetrics & Gynecology and Reproductive Biology

journal homepage: www.journals.elsevier.com/european-journal-of-obstetrics-and-gynecology-andreproductive-biology



Full length article





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ARTICLE INFO

Keywords: Positional plagiocephaly Deformational plagiocephaly Maternal risk factors Fetal risk factors Plagiocephaly screening

ABSTRACT

This study was conducted to determine the prevalence, maternal and/or neonatal risk factors for severe plagiocephaly in order to early detect and refer infants at risk. A prospective observational study was conducted, involving 4337 infants who visited the Perinatology Center at San Pietro Fatebenefratelli Hospital in Rome, evaluated following the Plagiocephaly Severity Scale of Atlanta. ©The plagiocephaly prevalence resulted 1.89%, considering moderate to severe forms. Maternal risk factors include primiparity, older age, gestational diabetes, and uterine fibromatosis. Neonatal risk factors are early term gestational age, low weight, twin pregnancy, and prolonged labor with an emergency cesarean section. Screening for severe plagiocephaly should begin antenatally. Although the low prevalence, identifying infants at risk can prevent potential permanent sequelae. We suggest a multidisciplinary approach for the management of plagiocephaly, involving the figure of the Obstetrician Gynecologist, who can highlight the risk factors ranging from obstetric and birth conditions.

Introduction

Positional Plagiocephaly (PPC) is one of the most common form of morphological anomaly of the skull observed in the absence of an early cranial suture synostosis. It is identified as a deformation of the skull bones that produces a characteristic asymmetry of the head. These deformities do not present pathological elements of malformation but are the product of a dynamic distortion of the skull secondary to the application of pre and/or post-natal external forces [1]. The term "plagiocephaly" is derived from the Greek "plagios" meaning oblique and "kephale" meaning head. This skull deformity results from repeated external pressure to a newborn's skull due to the head being in one position for extended periods. The most characteristic presentation is a parallelogram-shaped deformity, when the infant head is observed from above, and the forehead on the affected side is typically prominent. Initially, PPC was considered a purely cosmetic disorder for decades [1]. Nevertheless, PPC patients were found to have no significant difference in social functioning [2], and recently it was highlighted that schoolaged children who had moderate to severe PPC as infants scored lower on measures of cognition and academic achievement than unaffected children [3]. However, studies are very few and some did not find similar development impacts [4].

The real prevalence of this condition is unknown. The epidemiological studies show different diagnostic criteria and no differences among the different grade of severity. Several times, the condition is underestimated for a poor counselling of medical personnel or overestimated in case of lack of skills in differencing the various type and outcome of the clinical forms. Many types of cranial deformation have been studied, identifying an incidence of localized cranial flattening at the birth of 13% [5] but having a correct incidence of PPC based on severity is a difficult determination. PPC seems to be related to infant age, primarily manifesting in the first months of life: the prevalence at 6–7 weeks is estimated to be 16–22.1% [6] and by 2 years of age as low as 3.3% [7]. However, differences in the prevalence between mild and severe form were not reported.

In 2004, Argenta formulated a 5-grade classification of PPC based on



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the severity of the clinical presentation, although no data were reported about possible treatments [8] (Fig. 1). A recent classification, the Plagiocephaly Severity Scale, has been published from the Children's Healthcare of Atlanta in 2015 (Table 1). It is divided in 5 levels of severity and suggest different managements according to the level of severity: observation for the 1st level, manual repositioning for the 2nd and 3rd level, the possibility of using an orthosis for cranial remolding, in addiction of repositioning, in the 4th and 5th levels. This classification identifies the moderate form as level 2–3 and severe form as level 4–5 [9].

Treatments vary depending on the nature and severity of the deformity. Many mild cases do not require any treatment and PPC will remit spontaneously when the infant begins to sit [1]. However, more severe deformities may need treatment. In these cases, early referral is advised (between 1 and 6 months of age) as the majority of cranial growth is achieved during the first 12 months of life, and the greatest amount of correction will be achieved during this time. The most common treatments are counter positioning and helmet therapy [10,11].

Counter positioning involves consistently repositioning of the

Table 1
Plagiocephaly Severity Scale. From: Children's Healthcare of Atlanta (2015).

0 1 7	
Level	Clinical Presentation
1	All asymmetry within normal limits
2	 Minimal asymmetry in one posterior quadrant
	No secondary changes
3	Two quadrant involvement
	Moderate to severe posterior quadrant flattening
	Minimal ear shift and/or anterior involvement
4	 Two or three quadrant involvement
	Severe posterior quadrant flattening
	Moderate ear shift
	Anterior involvement including noticeable orbit asymmetry
5	 Three or four quadrant involvement
	Severe posterior quadrant flattening
	Severe ear shift
	Anterior involvement including orbit and cheek asymmetry

infant's head so that they do not rest on the flat spot. Increased tummy time and side lying play when the infant is awake is also encouraged. This treatment is recommended when a helmet is not warranted and is

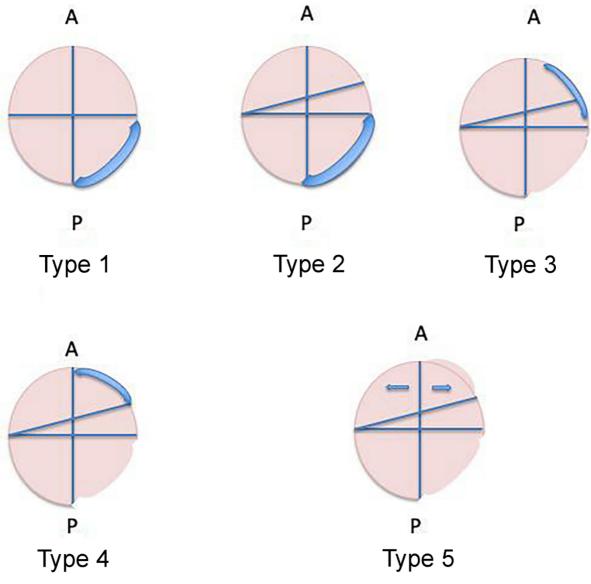


Fig. 1.

most effective between birth and 4 months of age. This counter positioning techniques can be taught by health professionals, including osteopath, maternal and child health nurses and pediatric physiotherapists.

The cranial remodeling helmet assists the skull molding process by removing the pressure over the flat area, allowing the skull to grow into the space provided. This treatment is preferred in severe cases of deformational PPC, and/or where a trial of counter positioning has failed. Helmets are most effective between 4 and 8 months of age. The average duration of helmet therapy is usually 2–6 months, depending on the age of the infant and the severity of the PPC. In rare cases, there is the need of neurosurgical intervention to correct the severe and pejorative conditions.

The incidence of the PPC has increased in recent decades due to strategies adopted to prevent sudden and unexpected infant death (SIDS) [12]. Indeed, the incidence of SIDS has been severely reduced thanks to the "Back to sleep" campaign started in USA in 1994 that has allowed to sensitize the medical staff and the families to the risks of using a prone position of the newborn. This campaign has led to an increase in the use of the supine position and an exponential decrease in the mortality rate [12]. This campaign was therefore very successful but resulted in a drastic increase in PPC incidence [12].

The head asymmetry can be caused by a number of factors, some of which are interrelated. Besides the well-known risk factors in the neonatal age, principally the persistent supine position of the newborn, several studies have also analyzed obstetric and fetal-neonatal factors such as twin pregnancies, male sex, intra-uterine position of the fetus, growth restriction and many others [13-15]. Bialocerkowski and colleagues in 2008 made a review of literature highlighting, among other factors, a higher risk of PPC for first-born and for operative vaginal deliveries [16]. In order to understand and evaluate possible antenatal and pregnancy-related factors for moderate and severe PPC, we performed this observational study including only infants with a PPC of level 2-5 according to the Atlanta's classification. The aim of the study was to understand how obstetrics features may be associated to PPC in order to create a multidisciplinary team that includes Gynecologists and Obstetrics as a figure of reference in the screening of infants at risk of developing severe deformation.

Materials and methods

Study design and inclusion/exclusion criteria

We conducted an observational prospective cohort study on infants born at the San Pietro Fatebenefratelli Hospital in Rome (Italy) during the period from January 2017 to September 2018.

Infants were evaluated at the first weeks of life in our center for the evaluation and the management of infant cranial asymmetry. We included only infants with diagnosis of moderate to severe PPC respecting the criteria of level 2–5 of Plagiocephaly Severity Scale, from the Children's Healthcare of Atlanta [9]. Exclusion criteria were the following: infants with congenital deformations, infants born before 24 weeks of gestation and infants who needed long intensive care treatments.

Data collection

We collected the subsequent data of each newborn: length, birth weight, cranial circumference, Apgar score at 5 min, type of delivery, type of anesthesia, gestational age, episiotomy and sex. We collected data of the mother concerning: maternal age, parity and maternity obstetric conditions.

The physical examination of infants for evaluating the presence of PPC was carried out at 6, 8, and 12 weeks after birth. The anthropometric measurements were detected using an anthropometer to evaluate cranial or facial asymmetry, head shape and anomalies of the ears. All

measurements were taken by the same operator and with the same device.

Statistical analysis

Statistical analysis was performed using SPSS version 22.0 (spss Inc., Chicago, IL, USA). Continuous variables were presented as mean \pm standard deviation (SD), whereas qualitative variables were presented as absolute frequencies and percentages. Comparisons between categorical variables were tested using contingency tables and chi-square test or Fisher's test when necessary. Comparisons between normally distributed continuous variables were performed by using Student's t-test. A p-value <0.05 was considered statistically significant.

Ethical approval

Parents signed a document approved by our institution before the procedure for the consent to the anonymous use of the clinical data for scientific purposes according to the European privacy law. The Institutional Review Board of the study Center approved the study. However, we do not provide a protocol code since this is not applicable in our institution for observational studies.

Results

A total of 4475 infants were evaluated during the study period. We ruled out a total of 138 patients because of the lack of obstetric data, 5 because of intrauterine death, and 35 for severe fetus malformation. Among the remaining 4297 infants, we enrolled in the study group 82 patients with diagnosis of moderate to severe PPC and 4215 patients in the control group. The prevalence of moderate to severe PPC, according to the classification adopted, in the analyzed population resulted 1.9% (82/4297).

Maternal and pregnancy-related factors

Concerning maternal/pregnancy-related factors and PPC we found a significant different distribution, in the two groups, for maternal age, gestational age at delivery, parity, previous uterine surgery, presence of uterine fibroids and twin pregnancy. In particular, mothers of PPC infants resulted significantly older than mothers of non-PPC infants (36.2 $\,$ \pm 4.7 vs 34.0 \pm 5.7, p < 0.01). For the nulliparous, the risk of having PPC children is significantly greater (p = 0.01). Gestational diabetes (GD) showed a higher frequency in the PPC group versus non-PPC group (8.5% versus 3.2%, p < 0.01). Also, the frequency of twin pregnancy resulted significantly higher in PPC group (9.8% versus 4.4%, p = 0.02). Moreover, the presence of uterine myomas, diagnosed during pregnancy, provides a statistically significant risk for the development of PPC (p = 0.03). We found no significant association between PPC and premature rupture of membrane (PROM), oligohydramnios, previous cesarean section, placenta previa and maternal hypertension (p > 0.05). Table 2 and Table 3 summarizes all these findings.

Fetal and delivery factors

Concerning fetal and delivery risk factors, we found a significant different distribution in the two groups, for weight at birth, length of newborn, unusual cephalic presentation, prolonged labor, and emergency caesarean section. The weight and the length of newborn at birth resulted significantly lower in PPC group versus non-PPC group (2990.6 \pm 607.4 vs 3165.8 \pm 522.8 cm, p < 0.01; 48.2 \pm 2.68 vs 48.9 \pm 6.5 cm, p = 0.02). Differences for cranial circumference at birth and sex resulted not significant between groups. In PPC group, we found a significant higher rate of urgent cesarean section performed for fetal condition compared to non-PPC group (29.2% vs 18.6% respectively, p = 0.03) as well as a higher rate of PPC in urgent cesarean section compared to

Table 2
Maternal and pregnancy-related factors for PPC.

Maternal Factors	PPC (n = 82)	no PPC (n = 4215)	p value	coefficient	OR
Maternal Age	36.2 ± 4.7	34.0 ± 5.7	<0.01	-	-
Gestational Diabetes	7 (8.5%)	137 (3.2%)	< 0.01	1.19	3.28
Hypertensive disorders	1 (1.2%)	78 (1.8%)	0.6	0.3	1.35
Hepatic disorders	0 (0%)	23 (0.5%)	_	_	-
History of uterine fibroids	2 (2.4%)	25 (0.6%)	0.03	1.77	5.9
Parity					
0	62 (75.6%)	2619 (62.1%)	0.01	0.63	1.89
>0	20 (24.3%)	1596 (37.9%)		-0.63	0.53
Pregnancy-related factors	PPC (n = 82)	no PPC (n =	p value	coefficient	OR
Oligohydramnios	1 (1.2%)	4215) 199 (4.7%)	0.13	-2.67	0.06
Polyhydramnios	0 (0%)	57 (1.35%)	-	-	-
Premature Rupture of Membrane (PROM) >24 h	15 (18.3%)	604 (14.3%)	0.3	1.42	4.14
Placental abnormalities	2 (2.4%)	33 (0.8%)	0.09	1.5	4.52
Number of foetuses					
Singleton	74 (90.2%)	4029 (95.6%)	0.02	-0.84	0.43
Twins	8 (9.8%)	186 (4.4%)		1.56	4.80

vaginal delivery (p = 0.17). We did not confirm a correlation with operative vaginal delivery and between vaginal delivery and elective caesarean, regardless of the indication. We found no significant differences in the two groups according to other factors such as episiotomy, Apgar score at birth, modality of labour induction. Table 3 summarizes all these findings.

Discussion

This study defines the prevalence of moderate to severe PPC in a large cohort of Italian women and correlated obstetrics and neonatal findings to PPC incidence. In literature, numerous studies try to identify the prevalence of PPC in infants, unfortunately without agreement between the definition of plagiocephaly, method of measurement and age of diagnosis. First of all, it is interesting noticing that the studies that used the Argenta score [16,17] observed a significantly higher PPC prevalence than the current study, mostly because we considered only moderate to severe PPC infants who need treatment due to the risk of permanent deformations [8,18-21]. The choice to consider only moderate to severe PPC cases derives from the importance of early diagnosis, and the need to initiate the proper treatment immediately to improve the prognosis avoiding permanent sequalae. For this reason, we focused on neonatal and maternal factors that potentially increase the risk of developing moderate to severe PPC that should be taken into account by the gynecologist during pregnancy and at the time of childbirth, in order to define a class of infants at risk.

We found an interesting correlation between PPC and some antenatal condition like gestational diabetes (GD), the presence of uterine myomas and previous uterine surgery. Moreover, in accordance with literature, we confirm other features associated with PPC like maternal age and nulliparity. [6,10,16,22].

Table 3
Fetal and delivery factors.

	PCP (n = 82)	No PCP (n = 4215)	p value	coefficient	OR
Fetal presentation					
Cephalic	77	4072	0.18	-0.61	0.54
	(93.9%)	(96.6%)			
Breech	5 (6.1%)	143 (3.4%)		0.52	1.68
Type of Delivery:					
Vaginal (n 2667)	0.5	0070	0.17	0.00	1.0
Spontaneous Birth	35 (42.7%)	2272 (53.9%)	0.17	0.28	1.32
Operative Vaginal	9 (11.0%)	351 (8.3%)		0.89	2.4
Delivery	7 (11.0%)	331 (0.370)		0.09	2.4
Cesarean (n 1630)					
Elective Cesarean	14	809	0.03	1.12	3.0
	(17.1%)	(19.2%)			
Emergency Cesarean	24	783		1.36	3.9
	(29.2%)	(18.6%)			
Induction of labor					
Pharmacological	4 (4.9%)	147 (3.5%)	0.49	-	1.4
Induction	70	4068			0.7
Spontaneous labor	78 (95.1%)				0.7
	(93.170)	(96.5%)			
Epidural anesthesia*					
None	3 (7.0%)	477	0.06	-	0.3
n	40	(18.2%)			
Epidural	40 (93.0%)	2139 (81.8%)			2.9
	(93.070)	(61.670)			
Episiotomy*	0.7	1707	0.77		
No	27	1707	0.7	_	0.9
Yes	(62.8%) 16	(65.2%) 909			1.1
ies	(37.2%)	(34.8%)			1.1
	(37.270)	(34.670)			
Gestational age at deli	very 37.7 ± 2.4	38.6 ± 2.0	< 0.01		
	37.7 ± 2.4	36.0 ± 2.0	<0.01	_	_
APGAR at 5 min	9.5 ± 0.8	9.6 ± 1.1	0.55		
	9.5 ± 0.6	9.0 ± 1.1	0.55	_	_
Sex	25	2021	0.22		0.0
Female	(42.7%)	2031	0.32	_	0.8
Male	(42.7%) 47	(48.2%) 2184			1.2
1724.16	(57.3%)	(51.8%)			1.2.
Weight (gr)					
ALCIBITE (ST)	2990.6 ±	3165.8 \pm	< 0.01	_	_
	607.4	522.8	~0.01	_	-
Height (cm)					
Height (cm)	48.2 ± 2.6	48 9 ± 6 5	0.02	_	_
	40.Z ± 2.0	48.9 ± 6.5	0.02	-	-
Cranial Circumference					
	33.8 ± 2.2	34.2 ± 1.7	0.054	_	-

^{*} Only in vaginal deliveries n 2659.

The role of GD in the genesis of PPC was already suggested by several studies [11,23-27]. This association could be explained by the series of morphological alterations related to this pathological condition, such as congenital heart defects, bowel and nervous system dysfunction, that can include not clinically relevant alterations in the musculoskeletal tissues. Subsequently, considering this pathological background the pre-

and post-natal position can have important effects on the cranium tissue. Other possible explanation for GD impact lies on the increase of biometric parameters and amniotic fluid index that occur in many diabetic pregnant women, that can lead to abnormal positioning of the fetus in utero before delivery.

Intriguingly, nulliparity is significantly correlated with PPC. We can speculate that term pregnancy uterine muscular tissue offers a higher resistance than multiparous women, whose uterus is used to stretch out. This, along with the longer time of the stages of labor in the nulliparous [28], can cause a persistent and prolonged compression on the head by the uterine forces, before delivery.

The same consideration is congruent with the presence of uterine fibroids or previous uterine surgery, such as myomectomy. In particular, uterine fibroids may be frequently subject to significant volumetric modifications during pregnancy. This event complicates the clinical management of patients affected by fibroids [29-31]. The growth of the myomas could limit the freedom of movement of the fetus from the earliest stages of pregnancy when the musculoskeletal tissue is not yet mature. This can even affect the newborn positions in the postnatal life, leading to an increased risk of PPC deformation.

For other obstetric conditions analyzed, such as placenta previa, breech presentation, previous cesarean section, premature rupture of membranes, abnormal quantity of amniotic fluid, no significant correlation was statistically detected for moderate to severe PPC. Summarizing, intrauterine environment could theoretically be attributed to development of PPC [32]. However, stronger protocols are needed to assess this pathological correlation.

Regarding neonatal factors, twin pregnancy was found correlated to PPC. This was also observed in the work of Meckinney et al. [24].

No correlations with PPC were found for the use of episiotomy. This could suggest that the head compression occur not during the head expulsion, but during the head passage through the birth canal [31].

We observed also a correlation to PPC for child's factors such as smaller length and lower birth weight. For this reason, we can strengthen our hypothesis that child's PPC tissues are not yet ripe and more susceptible to deformation assuming wrong positions [16,32,33].

Finally, considering the modality of delivery, we found contrasting results. First, operative vaginal delivery was not related to moderate to severe PPC (p = 0.174), in contrast to what should be expected from the traction pressure exercised on the fetal head. The visible alteration on the head shape after operative delivery is temporary and not permanent, in quite all of the cases. On the contrary, the urgent cesarean section (and not the elective one) for fetal indications, like non-reassuring cardiotocography or abnormal labor, is significantly correlated with the risk of severe PPC, although likely to be related to the indication rather than the surgical procedure itself.

Our results suggest that PPC onset prevention relies on first week of life action, as proposed by various studies [19-21]. Although, as stated above, different studies fail in demonstrating a correlation between PPC and neurodevelopment disorders, it is necessary to increase the awareness of the parents about the factors involved with the etiology of these conditions, and the importance to address the infant in dedicated pathways of care assistance [34-36]. In particular, it is important to inform the parents about simple acts useful in the prevention of PPC [16] without increasing the risk of Sudden Infant Death Syndrome, like periodically changing the orientation of the infant head during activity, a daily dose of supervised prone time, and reducing time in car seat [32,37-40]. Moreover, our study highlights the existence of prenatal possible factors in the genesis of cranial deformations, which suggests that the risk assessment for these conditions should begin antenatally. In this scenario, the gynecologist is the first healthcare provide able to individuate the high-risk group of fetuses observing certain conditions affecting the mother and the child during pregnancy and childbirth.

Conclusion

In conclusion, we defined different maternal and obstetrics features that could be related to the development of severe PPC. Further studies are needed to clarify the role of PPC on infant neurodevelopment. Parallelly, it is mandatory to recognize these factors in order to activate a multidisciplinary path (involving gynecologist, pediatrician, osteopath, parents and other specialized figures) with the aim of preventing possible sequelae in the newborn.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent: Informed consent was obtained from all individual participants included in the study.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

We thank all the physicians, osteopaths, statistician, nurses, and patients who participated in this study.

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