Original Article

Demographic Features and Risk Factors for Congenital Anomalies in Baghdad: Single Center Experience

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Abstract

Background: Congenital anomalies (CAs) are a major cause of childhood deaths and disability worldwide. Etiology is thought to be multifactorial. Those factors may be genetic (10%–30%), environmental (5%–10%), or multifactorial (20%–35%), whereas 30%–45% are unknown. **Objectives:** This study aimed to identify socio-demographic features and possible risk factors for CAs in a sample of Iraqi children in Baghdad. **Materials and Methods:** A prospective case-control study was conducted at Fatima Al-Zahraa Pediatrics and Maternity Hospital in Baghdad. Neonates diagnosed with CAs were included and compared to age and gender-matched randomly selected healthy newborns. Neonatal and maternal information was taken for every individual in the study. **Results:** The most commonly affected system was the musculoskeletal system (29.69%). Consanguinity, smoking, and irregular visits to antenatal care (ANC) centers were more frequent among mothers of neonates with CAs than those without anomalies. In addition, a family history of CAs was reported in 40.62% and 23.43% of neonates with and without CAs with statistical significance. Folic acid supplementation was absent in 29.69% of mothers with congenital anomaly children compared with 14.06% in those without congenital anomaly children. Previous sibling anomalies were reported in 18.75% of neonates with CAs versus 3.12% of those without CAs. **Conclusion:** Several independent risk factors for CAs were identified, including preterm birth, residency in an urban area, smoking, previous sibling anomalies, positive family history of CAs, lack of folic acid supplementation and irregular visits to ANC centers.

Keywords: Congenital anomalies, prematurity, risk factors

Introduction

According to the World Health Organization (WHO), congenital anomalies (CAs) are disorders that can develop in the fetus or embryo from conception until they are born.[1] They are a major cause of childhood deaths and disability worldwide.[2] A study conducted in 2015 revealed that CAs were one of the leading causes of death among children under five years old. They were also associated with 11% of infant fatalities.[3-5] A report released by the March of Dimes stated that around 8 million babies are born each year with major CAs. These defects contribute to around 300,000 yearly infant deaths within the first four weeks following their birth.[6]

The prevalence of CAs has been estimated to vary depending on the country. For instance, in North Africa and the Middle East, the highest incidence of birth defects is reported at 82 cases per thousand live births. In France, the rate is around 39.7 per thousand. On the other hand, in developing and low-income countries, the prevalence of CAs is around 55.7, and 64.2 per thousand births, respectively.[7] However, because many CAs can be difficult to identify without advanced clinical and laboratory capacities, it is very challenging to carry out epidemiological studies of CAs outside a high-income setting.[8] Information on the prevalence of congenital malformation in our locality is deficient, with significant underestimation of CAs in developing countries due to

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non-presentation at health facilities, poor awareness, under-reporting, and lack of proper diagnostic capacity.[9]

The etiology of CAs remains unclear but multiple factors are thought to be responsible. These factors may be genetic (10%–30%), environmental (5%–10%), or due to multifactorial inheritance (20%–35%), whereas 30%–45% are unknown.[10] Factors that may aggravate the risk of CAs involve nutritional factors such as maternal overweight and obesity, demographic and socioeconomic factors, infections, and taking certain medications during pregnancy, chemicals, air pollution, and ionizing radiation.[11-13]

According to a study by Al-Sabbak *et al*.[14] in Iraq, which was carried out at the Fallujah General Hospital, newborns with birth defects had lead levels that were three times higher and mercury levels that were six times higher than those of average Iranian children, whose traces of lead and mercury were slightly greater than those in European nations. Children with birth deformities in the Iraqi village of Hawija had levels of magnesium and titanium about twice as high as those of their Iranian counterparts on average, whereas samples taken from children with symptoms mimicking cerebral palsy had extremely high levels of cadmium and arsenic. These metals can contribute to the underdevelopment of vital tissues and a wide range of neurological disorders in developing fetuses by depleting folate levels.[15]

Several studies have identified old maternal age, multiparity, pregnancy status (singleton or multiple), and chronic maternal diseases as the main risk factors for CAs.[16-19]

Protective measures that may decrease the incidence of some CAs include ensuring having a healthy lifestyle by maintaining a healthy weight and adequate intake of minerals and vitamins, especially folic acid, avoiding smoking, and reducing environmental exposure to harmful substances such as pesticides, heavy metals, and radiation exposure.[17] This study aimed to identify socio-demographic features and possible risk factors for CAs in a sample of Iraqi children in Baghdad City.

Materials and Methods

A prospective case-control study was conducted in the neonatal care unit at Fatima Al-Zahraa Pediatrics and Maternity Hospital, which is a tertiary center in Baghdad, Iraq from August 21, 2022 to March 31, 2023. During the period of study, all live-birth neonates delivered with CAs were included in the study, with the exclusion of those delivered before completing 24 weeks of gestation. Included cases (64 cases) were compared to 64 age and gender-matched randomly selected healthy newborns who have no CAs as they visited the outpatient clinic for checkups.

Sample size

The sample size was calculated according to the following formula: $n = Z^2P(1 - P)/d^2$, where n is the sample size, Z is the statistic corresponding to the level of confidence, P is expected prevalence, and d is precision (corresponding to effect size).[20] According to previous studies, the prevalence of CAs among Iraqi children was found to be about 2.4%, the sample size was calculated as follows:

$$N = (1.96)^2 \times (0.024) \times (1 - 0.024)/(0.05)^2 = 36$$

Questionnaire

Detailed history and clinical examination were done on every individual in the study according to a standardized questionnaire. Neonatal information includes gender, gestational age (GA; term vs preterm), birth weight, mode of delivery (cesarean section vs vaginal delivery), birth status (single, twin, triple), and types of CAs according to the International Statistical Classification of Diseases and Related Health Problems, as congenital malformations of the nervous system, circulatory system, respiratory system, digestive system, genitourinary system, musculoskeletal system, and chromosomal abnormalities, not elsewhere classified.[21]

Maternal information includes age, residency (urban vs rural), consanguinity, previous abortion, smoking, folic acid supplementation during pregnancy, chronic drug use in 1st trimester, previous siblings' anomalies, family history of CAs, chronic maternal diseases (diabetes mellitus whether type I, II, or gestational diabetes, and hypertension), maternal infections during pregnancy (urinary tract infections and/or vaginal infections), and regular antenatal visits.

Statistical analysis

Statistical analysis was performed by using SPSS software version 25.0 (SPSS, Chicago). Continuous data were presented as mean and standard deviation and analyzed with a Student's t test. Categorical variables were expressed as numbers and percentages and analyzed with the Chi-square test. Independent risk factors for CAs had been found by a logistic regression test. From this test, the odds ratio (OR) and its corresponding 95% confidence interval (CI) were calculated. A P-value less than 0.05 was considered to be statistically significant.

Ethical approval

The study was conducted in accordance with the ethical principles outlined in the Helsinki Declaration. A local Ethics Committee examined and approved the study protocol, subject information, and permission form by document number 6 (including the number and the date in August 1, 2022) to receive this approval. The Mustansiriyah Medical College Ethical Committee gave their approval to the study protocol. Informed consent was obtained from the parents for recruitment in the study.

Results

Seven out of 11 socio-demographic characteristics were significantly associated with CAs. Twenty neonates (31.25%) with CAs were preterm compared to nine neonates (14.06%) without CAs. The vast majority of neonates with CAs (52 cases, 81.25%) were urban residents compared to 37 cases (57.81%) without CAs. Consanguinity was more frequent among parents of neonates with CAs than those without CAs (62.5% vs. 45.31%). Furthermore, non-exposure to smoking (whether passive or active) were more common among mother of neonates without CAs (68.75%) than those with CAs (39.06%). The mean maternal age of neonates with CAs was 30.28 ± 6.6 years which was higher than that of maternal neonates without CAs (26.89 ± 6.97 years). In addition, 40.62% of mothers of neonates with CAs had an irregular visit for antenatal care (ANC) compared with 21.87% of mothers of neonates without CAs who had such visits. Finally, a family history of CAs was reported in 26 (40.62%) and 15 (23.43%) neonates with and without CAs with a significant difference [Table 1].

Three maternal clinical factors were significantly associated with CAs. Folic acid supplementation was absent in 19 (29.69%) mothers with CA children compared to 9 (14.06%) mothers without CA children. Previous sibling anomalies were reported in 12 (18.75%) neonates with CAs versus two (3.12%) neonates without CAs. Diabetes mellitus was more frequent whereas hypertension was less frequent (18.75% and 3.12%, respectively) among mothers whose neonates had CAs than those with intact neonates (1.65% and 6.25%, respectively) as shown in Table 2.

Table 1: Neonatal and maternal socio-demographic factors

Variables	Congenital anomaly		Total (n = 128)	P-value
	Yes (n = 64)	No (n = 64)		
Birth weight, kg				0.267
Mean ± SD	2.69 ± 0.54	2.58 ± 0.6	2.67 ± 0.57	
Range	1.0-3.5	1.2-4.0	1.0-4.0	
Gender				1.00
Male	42 (65.63%)	42 (65.63%)	84 (65.63%)	
Female	22 (34.37%)	22 (34.37%)	44 (34.37%)	
Gestational age				0.020
Term	44 (68.75%)	55 (85.94%)	99 (77.34%)	
Preterm	20 (31.25%)	9 (14.06%)	29 (22.66%)	
Mode of delivery				0.269
CS	44 (68.75%)	38 (59.38%)	82 (64.06%)	
Vaginal	20 (31.25%)	26 (40.62%)	46 (35.94%)	
Pregnancy type				0.062
Singleton	63 (98.44%)	57 (89.06%)	120 (93.75%)	
Multiple	1 (1.56%)	7 (10.94%)	8 (6.25%)	
Residence				0.004
Urban	52 (81.25%)	37 (57.81%)	89 (69.53%)	
Rural	12 (18.75%)	27 (42.19%)	39 (30.47%)	
Consanguinity				0.050
No	24 (37.5%)	35 (54.69%)	59 (46.09%)	
Yes	40 (62.5%)	29 (45.31%)	69 (53.91%)	
Smoking habit				0.020
Never	25 (39.06%)	44 (68.75%)	69 (53.91%)	
Passive	36 (56.25%)	19 (29.69%)	56 (43.75%)	
Active	3 (4.69%)	1 (1.56%)	3 (2.34%)	
Mother's age, years				0.006
Mean ± SD	30.28 ± 6.6	26.89 ± 6.97	28.59 ± 6.97	
Range	19-45	14-45	14-45	
Family history of CA				0.037
None	38 (59.38%)	49 (76.56%)	87 (67.97%)	
Present	26 (40.62%)	15 (23.44%)	41 (32.03%)	
Regular ANC visit				0.022
Regular	38 (59.38%)	50 (78.13%)	88 (68.75%)	
Irregular	26 (40.62%)	14 (21.87%)	40 (31.25%)	

ANC: antenatal care

Table 2: Association of maternal clinical factors with congenital anomalies

Variables	Congenital anomaly		Total (n = 128)	P-value
	Yes (n = 64)	No (n = 64)		
History of abortion				0.108
Absent	32 (50%)	41 (64.06%)	73 (57.03%)	
Present	32 (50%)	23 (35.94%)	55 (42.97%)	
Folic acid supplementation				0.033
Absent	19 (29.69%)	9 (14.06%)	28 (21.88%)	
Present	45 (70.31%)	55 (85.94%)	100 (78.12%)	
Drug use in the 1st trimester				0.144
Absent	62 (96.88%)	58 (90.63%)	120 (93.75%)	
Present	2 (3.12%)	6 (9.37%)	8 (6.25%)	
Previous sibling anomalies				0.005
Absent	52 (81.25%)	62 (96.88%)	114 (89.06%)	
Present	12 (18.75%)	2 (3.12%)	14 (10.94%)	
Chronic maternal disease				0.011
None	48 (75%)	55 (85.94%)	103 (80.47%)	
Diabetes mellitus	12 (18.75%)	1 (1.56%)	13 (10.15%)	
Hypertension	2 (3.125%)	4 (6.25%)	6 (4.69%)	
Urinary tract/vaginal infections	2 (3.125%)	4 (6.25%)	6 (4.69%)	

In order to find independent risk factors for CAs, a logistic regression test was used. For this test, all factors with P-value ≤ 0.100 were entered into the model. Continuous variables were categorized into categorical variables using appropriate cut-off values. The results are shown in Table 3. Each of preterm birth (OR = 2.9, 95% CI = 1.09-7.69, P = 0.033), urban residence (OR = 3.36, 95% CI = 1.4-8.8, P = 0.007), never smoking (OR = 0.32, 95% CI = 0.15-0.67, P = 0.002), previous sibling's anomalies (OR = 4.75, 95% CI= 1.04-45.15, P = 0.041), family history of CA (OR = 3.11, 95% CI = 1.11-13.22, P = 0.038, absence of folic acid supplementation (OR = 2.81, 95% CI = 1.01-9.87, P = 0.041), and an irregular visit to ANC (OR = 2.47, 95% CI = 1.04-5.88, P = 0.041) are independent factors associated with CAs.

Classification of CAs according to the system affected shows that the most commonly affected system is the musculoskeletal (29.69%) followed by the genitourinary system (14.06%), gastrointestinal system (12.5%), whereas multiple systems were affected in 18.75% of patients as shown in Table 4.

Discussion

Few studies in Iraq have focused on risk factors for CAs. The current study shows that prematurity is a significant risk factor for CAs which is consistent with previous studies by Al Dewik *et al*. in Qatar,[6] Egbe *et al.* in the USA,[22] and Thakor *et al*. in India.[23] The majority of CAs have unknown causes, and it is also uncertain how or why these birth problems could induce preterm delivery. There are at least two potential explanations for the relationship between preterm delivery and birth defects. In some instances, similar risk factors could act independently to induce both birth defects and preterm birth. In other situations, a specific risk factor (such as inadequate folic acid supplementation) might result in a birth defect (such as spina bifida), and the presence of that birth defect may then be a contributing factor to preterm delivery.[24]

On the other hand, results by Abdou *et al.* in Egypt,[17] and Ajao *et al.* in Nigeria,[10] showed a non-significant correlation between gestational age and CAs. This variation could be explained by the lower percentages of premature neonates included in those studies in comparison to the present study (10% and 19.4%, respectively, vs 31.25% in the current study).

This study also found that living in an urban area, passive and active smoking were significantly associated with CAs, which is supported by Ameen *et al* in Erbil,[18] Abebe *et al*. and Mekonnen *et al*. in Ethiopia.[25,26] Because of the high concentration of air pollutants that urban residents are exposed to, there is a greatly increased risk of having babies with CAs.[27] These problems may be mostly caused by exposure to carbon monoxide and/or nicotine from smoking. The effect of carbon monoxide can be due to diminishing the oxygen supply to the body's tissues. Nicotine may work by increasing the hormones that constrict blood vessels and decreasing the blood flow to the uterus and placenta. These factors increase the likelihood of CAs by reducing the amount of oxygen and nutrients that are delivered to the fetus.[18]

Furthermore, we found that the maternal age for neonates with CAs was significantly higher than those without CAs, which agreed with Thakor *et al*. in India,[23] and Gedamu *et al*. in Ethiopia.[28] This could be a result of the risk-increasing effect of chromosomal abnormalities,

Table 3: Logistic regression

Variables	Congenital anomaly		*P*-value	OR (95% CI)
	Yes ($n = 64$)	No ($n = 64$)		
Gestational age				
Term	44 (68.75%)	55 (85.94%)	0.033	1.0
Preterm	20 (31.25%)	9 (14.06%)		2.9 (1.09-7.69)
Pregnancy type				
Singleton	63 (98.44%)	57 (89.06%)	0.078	1.0
Multiple	1 (1.56%)	7 (10.94%)		0.14 (0.02-1.25)
Residence				
Rural	12 (18.75%)	27 (42.19%)	0.007	1.0
Urban	52 (81.25%)	37 (57.81%)		3.36 (1.4-8.8)
Consanguinity				
No	24 (37.5%)	35 (54.69%)	0.094	1.0
Yes	40 (62.5%)	29 (45.31%)		1.96 (0.89-4.33)
Smoking habit				
Active	3 (4.69%)	1 (1.56%)	0.004	1.0
Passive	36 (56.25%)	19 (29.69%)	0.113	3.72 (0.81-12.62)
Never	25 (39.06%)	44 (68.75%)	0.002	0.32 (0.15-0.67)
Previous sibling anomalies				
Absent	52 (81.25%)	62 (96.88%)	0.041	1.0
Present	12 (18.75%)	2 (3.12%)		4.75 (1.04-45.15)
Family history of CAs				
None	38 (59.38%)	49 (76.63%)	0.038	1.0
Present	26 (40.62%)	15 (23.43%)		3.11 (1.11-13.22)
Folic acid supplementation				
Present	45 (70.31%)	55 (85.94%)	0.047	1.0
Absent	19 (29.69%)	9 (14.06%)		2.81 (1.01-9.87)
Mother's age, years				
≤30	38 (59.38%)	47 (73.44%)	0.359	1.0
>30	26 (40.62%)	17 (26.56%)		1.51 (0.63-3.63)
Regular ANC visit				
Regular	38 (59.38%)	50 (78.13%)	0.041	1.0
Irregular	26 (40.62%)	14 (21.87%)		2.47 (1.04-5.88)
Chronic maternal disease				
None	48 (75%)	55 (85.94%)	0.283	1.0
Diabetes mellitus	12 (18.75%)	1 (1.56%)	0.552	3.2 (0.54-13.6)
Hypertension	2 (3.125%)	4 (6.25%)	0.124	0.42 (0.76-18.33)
Infection	2 (3.125%)	4 (6.25%)	0.983	0.97 (0.08-12.0)

Table 4: Systemic distribution of congenital anomalies ($n = 64$)

System	Number	%
Musculoskeletal system	19	29.69
Genitourinary system	9	14.06
Gastrointestinal system	8	12.5
Central nervous system	6	9.38
Cardiovascular system	4	6.25
Respiratory system	2	3.13
Multiple systems	12	18.75
Syndromes	4	6.25

which are more common in older mothers, in addition to the accumulating effects of environmental exposures over time.[29] Ajao *et al.*[10] and Ameen *et al.*[18] studies found a non-significant relationship between maternal age and neonatal CAs. The majority of mothers in those studies were younger than 35 years old, which could explain their variations with current results.

In addition; family history of CAs, previous siblings' anomalies, and positive consanguinity showed significant correlation with CAs. The results found by Abo El Ella *et al*. in Egypt,[1] Ameen *et al*.,[18] Abebe *et al*.,[25] and Sarkar *et al*.[30] were consistent with this study, which

may explain the genetic causes of CAs. It is believed that consanguineous marriages significantly increase the possibility of birth defects. Recessively inherited conditions are more likely to run in families, and the closer the relationship, the more likely it is that a child may inherit one.[18]

Regarding ANC, our study showed that lack of regular visits to ANC centers and lack of folic acid supplementation during pregnancy had significant associations with CAs. These results are supported by Abo El Ella *et al*.,[1] Ameen *et al*.,[18] and Abebe *et al*.[25] Lack of regular visits to ANC centers impairs proper gestational follow-up and prevents early detection and treatment of possible risk factors. Folic acid consumption before conception and early in pregnancy is known to decrease a woman's risk of giving birth to a child who has a neural tube defect and possibly other defects,[24] that could be explained by folic acid's significant contribution to DNA and RNA synthesis, which is necessary for cell divisions during embryonic and fetal development.[31] Ekwochi *et al*. study in Nigeria[32] found a non-significant correlation between lack of ANC visits and folic acid supplementations with the occurrence of CAs, which could be attributed to the small sample size (38 patients) taken in that study in comparison to the current study.

In addition, diabetes mellitus (whether type I, II, or gestational diabetes) in this study showed a significant correlation with CAs, which agreed with Abebe *et al*.,[25] Ameen *et al*.,[18] and Sinha *et al*.[33] Diabetes mellitus is one of the major causes with up to a nine-fold increase in birth defects, compared with the rate recorded in non-diabetic pregnancies. Despite being complex, the pathophysiology of maternal diabetes-induced birth defects is definitely related to maternal glucose levels.[34] High glucose levels during critical periods of morphogenesis appear to be the main teratogen in diabetic pregnancy. The exact mechanism is unknown, but abnormal maternal/fetal fuel metabolism, including hyperglycemia, hyperketonemia, disordered metabolism of arachidonic acid, myoinositol, and prostaglandin, as well as elevated levels of oxidative stress, are thought to be responsible for some of the changes in embryonic development.[35]

The current study found that gender, birth weight, mode of delivery, pregnancy status, and history of previous abortion has non-significant correlations with CAs, which were approximately similar to results found by Ajao *et al*.,[10] Al Dewik *et al*.,[6] Egbe *et al*.,[22] and Mekonnen *et al*.[26]

Furthermore, the current study demonstrated a non-significant correlation between maternal drugs used in the 1st trimester and CAs. However, since only 8 mothers in the current study had received medications during the 1st trimester, we cannot rely on it to say that those drugs are safe during pregnancy. Ajao *et al*.[10] study found that the practice of self-medication and the use of herbal preparations during pregnancy were associated with higher risks of CAs but this was not statistically significant.

The musculoskeletal system was the most common system affected by CAs in this study (29.69%), while multiple systems were affected in 18.75% of cases. Similar results were found by Sarkar *et al*.[30] and Bhalerao *et al*.[36] In contrast, Al Dewik *et al*.,[6] and Egbe *et al*.[22] found that the cardiovascular system was the most commonly affected (35% and 35.5% respectively), whereas Silesh *et al*.[37] and Ameen *et al*.[18] found that neural tube defects were the most common (28.1% and 37.7%, respectively). These differences could be attributed to various genetic and environmental factors in different countries.[38]

The most important limiting factor in this study was the small sample size in a single center which may not reflect the overall population.

Conclusion

Several independent risk factors for CAs were identified in this study, including preterm birth, residency in an urban area, smoking, previous sibling anomalies, positive family history of CAs, absence of folic acid supplementation and irregular visits to ANC centers. Efforts should be made seriously to minimize these risk factors, with special attention to folic acid supplementation and proper ANC to decrease premature deliveries and early detection of CAs, especially for families with a positive history of CAs. Further multi-centric studies with a larger sample size should be carried out to strengthen the study results.

Consent to participate

The consent of participation in the present study was taken from all patients from whom the samples were taken.

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Conflicts of interest

There are no conflicts of interest.

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