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A CLINICAL STUDY OF MATERNAL AND PERINATAL OUTCOME IN OLIGOHYDRAMNIOS

Gynecology

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ABSTRACT

BACKGROUND Oligohydramnios is a major cause of maternal and perinatal mortality.This study was aimed to evaluate maternal and perinatal outcome in oligohydramnios.

AIMS -To find out the incidence of oligohydramnios by ultrasonographic evaluation of amniotic fluid index(AFI)

- To find out risk factors associated with oligohydramnios
- To find out maternal outcome in form of mode of delivery
- To find out congenital anomalies associated with oligohydramnios
- To find out NICU admission rate and stillbirth rate associated with oligohydramnios

MATERIALS AND METHODS We took history of patients after taking their consent.Obtetric history,medical history,surgical history,past history,family history ,menstrual history,personal history and negative history were taken.AFI was measured by Phelan's four quadrant ultrasound technique.

RESULTS -Incidence of oligohydramnios was 3%.

- 78.5% cases were associated with obstetric complications.
- LSCS was done in 47% cases.

36% neonates werr admittef to NICU.

Perinatal mortality was 15%.

Congenital anomalies were detected in 9% cases.

CONCLUSION Hence it can be concluded that regular antenatal and intranatal monitoring should be done to improve maternal and perinatal outcome.

KEYWORDS

Maternal,Fetal,Oligohydramnios,AFI

INTRODUCTION

The aquatic environment of the fetus has long remained enigma for patients and obstetricians.Early in the developmental life the fetus becomes enclosed by amnion and is surrounded by amniotic fluid which initially is similar to extracellular fluid.Importance of amniotic fluid volume as indicator of fetal status has made assessment of amniotic fluid volume an important part of antenatal fetal surveillance.

Incidence of oligohydramnios is 1 to 5 % of total pregnancies.

Oligohydramnios is associated with post datism,premature rupture of membranes,abruptio placentae,preeclampsia,uteroplacental insufficiency,intrauterine growth restriction,etc.It is also associated with congenital fetal anomalies like Potter's syndrome,club foot,club hand,pulmonary hypoplasia,hip dislocation,etc.

Major mechanical function of amniotic fluid is to provide cushion for umbilical cord.Without this cushion there is compression of cord leading to fetal distress.This required operative vaginal delivery or cesarean section.

MATERIALS AND METHODS

This study included 200 cases of oligohydramnios(AFI<5).

Inclusion criteria

- Sonographically proven cases of oligohydramnios
- Gestational age>28 week
- Singleton pregnancies

Exclusion criteria

- AFI > 5 cm
- Multiple gestations
- Presence of respiratory,cardiovascular or other abdominal diseases

METHODOLOGY

Plan of activity was formulated after taking verbal consent from patients and their relatives.Patients sociodemographic data were obtained including maternal age,maternal risk factors like PIH,etc,at the time of admission.History was obtained.General,systemic and obstetric examination was done.Hematologic profile was done.AFI was measured by Phelan's four quadrant ultrasound technique.In this method,uterus is devided arbitrarily in 4 quadrants by umbilicus transversally and linea nigra vertically.The largest vertical pocket free of fetal parts and umbilical cord loops is measured in each quadrant and sum of all these measurements gives AFI in cm.Narmal value of AFI is 5 to 24 cm.USG included fetal biometry,colour doppler and amniotic fluid volume.Outcome was noted in form of mode of delivery,fetal outcome,postnatal complications,NICU admission,etc.

RESULTS AND DISCUSSION

TABLE-1 Incidence Of Oligohydramnios

Total admissions	**12854**
Total deliveries	**10362**
Cases of oligohydramnios	**386**
Incidence of oligohydramnios	**3%**

TABLE-2 Maternal Risk Factors Associated With Oligohydramnios

Maternal risk factors	Number of cases
Postterm pregnancy	1
Prolonged pregnancy	33
Preeclampsia	15
Gestational hypertension	26
Eclampsia	1
PROM	17
Breech	21
Chirioamnionitis	1
Malnutrition+Anemia	110

TABLE-3 Congenital Anomalies Associated With Oligohydramnios

Congenital anomaly	Number of cases
Hydronephrosis	9
Polycystic kidney disease	1
Renal agenesis	1
Renal ectasia	1
Hydrocephalus	2
CTEV	1

TABLE-4 Mode Of Delivery In Cases Of Oligohydramnios

Mode of delivery	Number of cases
Vaginal	106
Cesarean	94

TABLE-5 Perinatal Outcome In Cases Of Oligohydramnios

Perinatal outcome	Number of cases
Healthy	118
NICU admissions	72
Stillbirths	10

Clinical features of oligohydramnios are decreased symphysio-fundal height,undue prominence of fetal parts,fetal malpresentations,etc.

Complications are cord compression,musculoskeletal abnormalities like facial distortion and club foot,intrauterine growth restriction, pulmonary hypoplasia,amnion nodosum,etc.Amnion nodosum is presence of nodules on fetal surface of amnion.

Oligohydramnios is also aaaociated with intrauterine infections, triploidy,use of drugs like indomethacin and ACE inhibitors,etc.

Diagnosis is made from following features:
- Uterine size is much smaller than period of amenorrhoea
- Fewer fetal movements
- Malpresentations like breech
- Undue prominence of fetal parts
- USG shows largest liquid pool of < 2 cm
- Evidences of IUGR

Potter's syndrome is atypical physical appearance of the baby due to oligohydramnios.It includes clubbed feet,pulmonary hypoplasia and cranial anomalies.It is also called Potter's sequence or oligohy dramnios sequence.It is mainly caused by bilateral renal agenesis.It is also characterised by bowing of legs,sirenomelia or Mermaid syndrome,parrot beak nose,redundant skin and skin fold extending from medial canthus across the cheek(most characteristic). Ears are slightly low and pressed against head making them appear large.In males vas deferens and seminal vesicles may be absent and in females uterus and upper vagina may be absent.Other abnormalities include esophageal atresia,duodenal atresia,anal atresia,absence of rectum and sigmoid colon and a single umbilical artery.Diaphragmatic hernia is also seen.

Bilateral renal agenesis has been estimated to occur at a frequency of 1:4000 to 1:8000 fetuses and neonates.However recent analysis has estimated that the condition may occur at much greater frequency.It occurs more commonly in males than in females.It has predominantly genetic etiology with incomplete penetrance and variable expressivity. There are several genetic pathways that could result in this condition.In 2017 researchers identified heritable autosomal dominant mutations in the gene GREB1L in two unrelated families as being the cause of both bilateral renal agenesis and unilateral renal agenesis utilising exome sequencing and direct sequence analysis.This is the first reported genetic lesion implicated in activation of retinoic acid receptor targets that has been associated with renal agenesis.The majority of other possible candidate genetic pathways are autosomal recessive in nature and do not coincide with frequency or penetrance at which bilateral renal agenesis occurs in population.Potter sequence is classified into classic form,type 1,type 2,type 3,type 4 and other forms.

Classic form-This term is traditionally used in cases of bilateral renal agenesis(BRA) i.e,kidneys do not develop due to malformation of ureteric bud.True BRA is also associated with bilateral agenesis of ureters.

Type 1-It occurs due to autosomal recessive polycystic kidney disease which occurs at a frequency of 1 in 16000 infants.Kidneys are enlarged.Kidneys cannot produce adequate fetal urine.Mutiple amalk cysts are present in kidneys which are filled with fluid.Liver and pancreas show fibrosis and/or cystic change.

Type 2-It is usually due to renal agenesis which also fall under category known as hereditary urogenital adysplasia or hereditary renal adysplasia.It is characterised by agenesis or absence of one kidney.The other kidney is small and malformed.

Type 3-It is associated with autosomal polycystic kidney disease.It is caused by mutations in PKD1 and PKD2 genes.It usually affects adults but it is also found in neonates in rare cases.Splenomegaly and hepatic cysts may be present.Increased prevalence of vascular disease is also found in these cases.

Type 4-It is caused by longstanding obstruction of kidney or ureter.It leads to cysic kidneys and hydronephrosis.

Other forms if Potter sequence are known as multicystic renal dysplasia.

Pathogenesis-Development of mature kidneys begins between 5 and 7 week of gestation.Fetal urine production begins in early gestation and it comprises majority of amniotic fluid in second and third trimester of pregnancy.The fetus continuosly swallows amniotic fluid which is absorbed by gastrointestinal tract and then reintroduced in the ambiotuc cavity via urination.The fetal urine is critical to the development of lungs by aiding in the expansion of airways-alveoli by means of hydrodynamic pressure and by also supplying prolin which is critical amino acid for development of lungs.

The outcome of Potter sequence is poor.Survivors suffer from chronic kidney disease.Some develop end stage renal disease.Some show growth impairement and delay in cognitive and motor development.

Mode of delivery-LSCS was done in 47% cases of oligohydramnios. Rates of LSCS and induction of labour were increased. Vaginal delivery was conducted in 53% cases.

PERINATAL OUTCOME-Apgar score was decreased < 7 at 5 minutes in 17.5% newborns in this study.It was measured at 1 minute and 5 minute.

Apgar score is calculated as follows:
Apgar score-It is a score for quick evaluation of neonate.It is calculated from five simple criteria on a scale from zero to two,then summing up the five values.Apgar score ranges from zero to ten.Five criteria are as below:

	0	1	2
Pulse rate	Absent	<100 bpm	>100 bpm
Reflex irritability grimace	No response to stimulation	Grimace to suction or agressive stimulation	Cry on stimulation
Skin colour	Blue or palr all over	Extrimities-blue Body-Pink (Acrocyanosis)	Whole body is pink with no cyanosis
Activity	None	Some flexion	Flexed arms and legs that resist extension
Respiratory effort	Absent	Weak,irregular,gasping	Strong robust cry

The score is calculated at 1 and 5 minutes after birth.It is repeated later if score remains low.Score of more than 7 is considerrd normal.Score of 4 to 6 is considered fairly low and score of less than 3 is considered critically low.Score of less than 3 requires immidiate resuscitation. Low score at 1 minute indicates that neonate requires medical attention but not necessarily a long term problem,particularly if score improves at 5 minute.Score of less than 3 at 10 minute may indicate long term neurologic damage leading to cerebral palsy.Term APGAR is a bacronym.Its full form is as below:

A-Appearance
P-Pulse
G-Grimace
A-Activity
R-Respiration

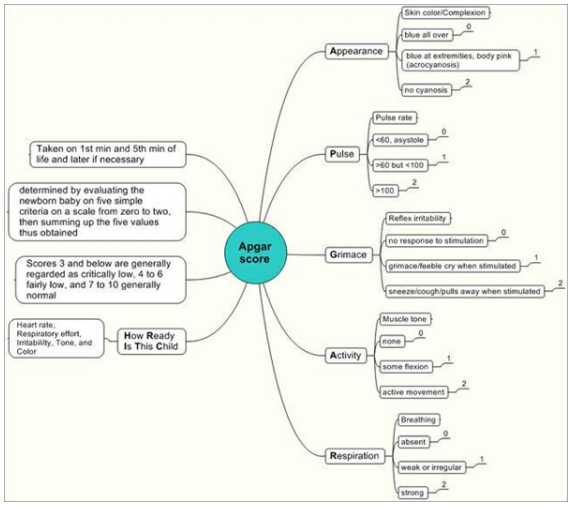

FIGURE-1 Mind map of Apgar score

A score of 10 is uncommon due to high prevalence of transient cyanosis which is common in neonates born at high altitude.It is substantially similar to score of 9.

In present study there were 95% live births and 5% stillbirths.Neonatal mortality was 10% and perinatal mortality was 15%.

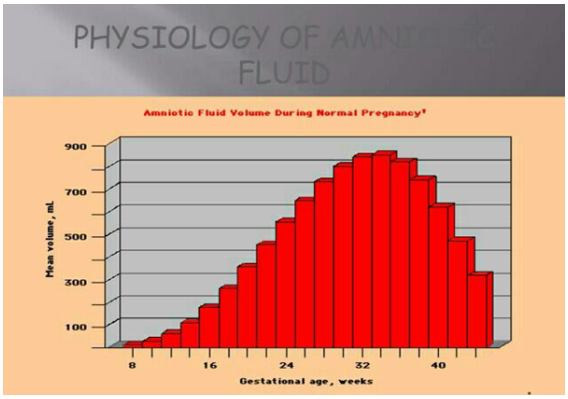

FIGURE-2 Physiology of amniotic fluid

Physiology of amniotic fluid-

Amniotic fluid is present from the development of gestational sac.Amniotic fluid is in the amniotic sac.It is generated from maternal plasma and passes through fetal mambranes by hydrostatic and osmotic forces.After development of fetal kidneys at about 16 week fetal urine also contribute to amniotic fluid.It is absorbed through fetal tissue and skin.After keratinization at 15-25 week amniotic fluid is mainly absorbed through gut.

Volume-It increases with growth of fetus.From 10 to 20 week its volume increases from 25 ml to 400 ml.At 11th week breathing and swallowing of fetus slightly decreases volume but neither swallowing nor urination significantly contribute to volume changes until the 25th week,when ketatinization of skin is complete.Then relationship between amniotic fluid and fetal growth stops.It reaches plateau of 800 ml at 28 week of gestation.Volume declines to 400 ml at 42 week of gestation.

Function-Swallowed amniotic fluid creates urine and contributes to formation of meconium.Amniotic fluid protects fetus from blows to abdomen of mother and promotes development of musculoskeletal system of fetus.It also helps in formation of gastrointestinal tract.Contrary to popular belief amniotic fluid is neither inhaled nor exhaled.

Analysis of amniotic fluid can reveal genetic health of fetus and age and viability of fetus.Amniotic fluid contains fetal cells which are used for assessing genetic defects.Amniotic fluid contains metabolic waste and compounds which are used to assess age and lung maturity.pH of amniotic fluid is 7.0 to 7.5.

CONCLUSION

In presence of oligohydramnios thorough evaluation of maternal risk factors like PROM,preeclampsia,gestational hypertension, diabetes, drig history,etc should be done. Oligohydramnios is associated with adverse maternal and fetal outcome.Colour doppler should be performed to evaluate hypoxic changes.Regular antenatal and intranatal monitoring is required to improve maternal and fetal outcome.Oligohydramnios is associated with higher rates of lower segment cesarean section and induction of labour.Oligohydramnios is associated with higher rates if NICU admissions and stillbirths. Oligohudramniis is associated with low Apgar scores and higher rates if congenital fetal anomalies.Maternal and perinatal outcome can be improved by early diagnosis and treatment.Cesarean section rates and stillbirth rates can be reduced by early diagnosis of oligohydramnios.

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