A Study on Neonatal Polycythaemia- Incidence and Clinical Profile

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ABSTRACT

BACKGROUND

Raised haematocrit results in increased blood viscosity which in turn, according to Poiseuille's law, reduces flow of blood causing sludging of red blood cells within the microcirculations causing ischemic damage to different organs. A good number of neonates suffer from polycythaemia. Data on the extent & severity of this problem is scarce in this part of our country.

METHODS

Capillary haematocrit was estimated by heel puncture from prewarmed heel in a cohort of 400 consecutively born intramural neonates. Peripheral venous haematocrit was estimated from blood obtained from antecubital vein in neonates with capillary haematocrit ≥65%. Neonates with peripheral venous haematocrit ≥65% were considered to be suffering from polycythaemia. Polycythaemic neonates were clinically screened thoroughly & critically evaluated for signs & symptoms and selective biochemical parameters (bilirubin, glucose calcium etc). Polycythaemic neonates who manifested with two or more signs & symptoms were considered as symptomatic polycythaemia.

RESULTS

Of the 400 neonates, 196 were boys and 204 were girls. Birth weight ranged from 955 Gms to 4830 Gms with an average of 2744 ± 487 Gms. Gestational age of the neonates ranged from 31 weeks to 42 weeks with an average of 38 ± 1.6 wks. Capillary haematocrit (cap. Hct) ranged from 49% to 89% with an average of (66.5 ± 8)%. Out of the 400 neonates, cap. Hct of 243 neonates was 65% and above. 27 neonates were found to have peripheral venous haematocrit in the range of polycythaemia (≥65%) with an incidence of 6.7%. Of the 27 polycythaemic neonates, 15 (55.5%) were boys and 12 (44.5%) were girls. 19 (70.4%) were with AGA, 7 (25.9%) were SGA and one (3.7%) was LGA. 24 (88.8%) polycythaemic neonates were term and 3 (11.12%) were preterm. 22.2% polycythaemic neonates were symptomatic. Common signs & symptoms were peripheral cyanosis (18.5%), tachycardia (14.8%), systolic murmur (7.4%), tachypnoea (14.8%), jitteriness (11.1%), irritability (7.4%), lethargy (11.1%), seizure (11.1%) and vomiting with feeding difficulty (14.8%).

CONCLUSIONS

Polycythaemia is an important neonatal health problem, affecting an impressive number (6.7%) of neonates who require special care. So, all the neonates, born out of normal or high-risk pregnancy should be screened for polycythaemia. SGA & LGA are more prone to develop polycythaemia. Premature neonates are also equally likely to develop polycythaemia, like that of term neonates contrary to usual belief.

KEYWORDS

Polycythaemia, Capillary, Venous, Haematocrit, Neonate

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BACKGROUND

In Biblical references, "to shed" blood was a term used in the sense of "to kill"[1]. But excess blood is also detrimental to health. Raised haematocrit results in increased blood viscosity which in turn, according to Poiseuille's law, reduces flow of blood & sludging of red blood cells within the microcirculations causing ischemic damage of different organs. A good number of neonates suffer from polycythaemia. Data on the extent & severity of this problem is scarce in this part of our country.

Aims and Objectives

1. To find out the incidence of polycythaemia in a cohort of neonates taking peripheral venous haematocrit ≥ 65% as the criteria for diagnosis[2].
2. To find out the profile of clinical signs & symptoms and abnormalities in selective biochemical parameters of blood (glucose, calcium, bilirubin etc.) in polycythaemic neonates of the same cohort.

METHODS

The proposed study was undertaken in the sick new-born care unit of Department of Paediatrics of Bankura Sammilani Medical College. 400 consecutive live inborn neonates constituted the study cohort. Detailed maternal & perinatal history was noted in each neonate. In each neonate capillary haematocrit was estimated by heel puncture from prewarmed heel. haematocrit was estimated by taking blood in a heparinised capillary tube & spun for five minutes at a rate of 12000 rpm in a microhematocrit centrifuge machine. haematocrit was estimated between 2 to 12 hours of postnatal age. Peripheral venous haematocrit was estimated by the same method from blood obtained from antecubital vein in neonates with capillary haematocrit ≥65%.

Neonates with peripheral venous haematocrit ≥65% were considered to be suffering from polycythaemia.[2] Polycythaemic neonates were clinically screened thoroughly & critically evaluated for signs & symptoms and selective biochemical parameters (bilirubin, glucose calcium etc). Routine blood examination including platelet count was done in every polycythaemic neonate. As sings & symptoms of polycythaemia are non-specific and many other conditions can manifest similar signs & symptoms, necessary investigations were done to rule out other etiologist. Polycythaemic neonates manifested with two or more signs & symptoms were considered as symptomatic polycythaemia.[2] Necessary permission & consent was taken to conduct the study.

RESULTS

Table 1 shows the patient's profile of this study. Of the 400 neonates, 196 were boys and 204 were girls. Birth weight ranged from 955 Gms to 4830 Gms with average (2744±487) Gms. Gestational age of the neonates ranged from 31 weeks to 42 weeks with average (38±1.6) wks. 45 neonates were preterm, 353 neonates were term and 02 were post term. 336 were AGA, 57 were SGA and 07 neonates were LGA. Post-natal age at the time of estimation of capillary haematocrit ranged from 2 hours to 12 hours with an average of (6.9 ± 4.8) hours.

Characteristics	Total	AGA	SGA	LGA
No. of patients	400	336	57	07
Boys	196	165	27	04
Girls	204	171	30	03
Birth weight	2744 ± 487	2839 ± 383	2095 ± 307	3386 ± 456
Gestational age	38 ± 1.6	38 ± 2.2	38 ± 1.8	38 ± 1.5
Preterm	45	41	04	00
Term	353	294	52	07
Post term	02	01	01	00
Age at estimation of haematocrit (hours)	6.9 ± 4.8	7 ± 2.9	6.5 ± 2.7	7.4 ± 2.1

Table 1. Patients' Profile

Capillary haematocrit (cap. Hct) ranged from 49% to 89% with an average of (66.5 ± 8)%. Highest haematocrit was noted in LGA neonates (74.2 ± 5)% followed by in SGA neonates (70.2±7.4)% and then AGA neonates (65.7 ± 7.9)%. The raised capillary haematocrit values in LGA & SGA neonates in comparison to AGA neonates were statistically significant with Z values 4.23 & 4.4 respectively. Out of the 400 neonates, cap. Hct of 243 neonates were 65% and above. In these neonates, peripheral venous haematocrit (PV Hct.) and haemoglobin level were estimated.

Characteristics	Total	AGA	SGA	LGA
No. of polycythaemic neonates	27	19	07	01
Boys	15	10	04	01
Girls	12	09	03	00
Birth weight	2964±575	3099±352	2333±113	4830±0
Gestational age	38±1.54	38±1.73	39±0.78	39±0
Term	24	16	07	01
Pre-term	03	03	00	00
Post term	00	00	00	00
Age at estimation	6.6±1.9	6.4±2.2	7.3±1.2	6.5±0
CAP. HCT	79.9±4.6	79±4.5	82.2±5	81±0
PV haematocrit	67.9±3.3	66.8±1.9	70±5	73±0
Haemoglobin	22.5±1	22.1±0.7	23.1±1.5	23.8±0

Table 2. Profile of Polycythaemic Neonates

Of the 400 neonates of our study cohort, 27 were found to have peripheral venous haematocrit in the range of polycythaemia (≥65%). Table 2 shows the profile of polycythaemic neonates. Of the 27 polycythaemic neonates 15 (55.5%) were boys and 12 (44.5%) were girls. 19 (70.4%) were with AGA, 7 (25.9%) were SGA and one (3.7%) was LGA. 24 (88.8%) polycythaemic neonates were term and 3 (11.12%) were preterm. Of the two post term neonates in this study series on one was with PV Hct in the range of polycythaemia. The birth weight of polycythaemic neonates ranged from 2188 Gms to 4830 Gms with mean (±SD) of (2964 ± 575) Gms. The gestational age ranged from 34 wks. to 40 wks., with mean (±SD) of (38±1.5) wks. The average age of estimation of haematocrit was (6.6 ± 1.9) hrs with a range of 4 hrs to 12 hrs. The Cap. HCT in polycythaemic neonates ranged from 71% to 89% with mean (±SD) of (79.9 ± 4.6)%. The corresponding PV Hct ranged from 65% to 81% with mean (±SD) was (67.9 ±

3.3)%. The corresponding haemoglobin level ranged from 21.3 gm% to 26.6 gm%.

Overall incidence of polycythaemia in our study was 6.75% (27/400). In AGA, SGA and LGA neonates the incidences were 5.6% (19/336), 12.2% (7/57) and 14.2% (1/7). This difference in incidence in different groups was not significant (Z values were 1.46 & 0.64 in SGA vs AGA and LGA vs AGA respectively). In term & preterm neonates the incidences were 6.7% (24/353) and 6.6% (03/45). Highest incidence was noted in term-LGA neonates (14.2%), followed by term-SGA neonates (13.46%). Lowest incidence was in term-AGA neonates (5.44%). In preterm AGA neonates the incidence was 7.3%.

Aetiological Factors	No. of Polycythaemic Neonates
Maternal Diseases-	
1. Diabetes mellitus	3 (11.1%)
2. Rheumatic heart diseases	2 (7.4%)
3. Hypothyroidism (on Eltroxin therapy)	1 (3.7%)
4. Uterine fibroid	2 (7.4%)
Obstetric Factors	
1. PET/ET/PIH	5 (18.5%)
2. Oligohydramnios	4 (14.8%)
3. Placentas praevia with antepartum haemorrhage	1 (3.7%)
Foetal Factors	
1. Foetal bradycardia	2 (7.4%)
2. Birth asphyxia	2 (7.4%)
3. Congenital malformation (cleft lip & palate)	1 (3.7%)

Table 3. Aetiological Factors Associated with Neonatal Polycythaemia

Six out of 27 polycythaemic neonates were symptomatic with two or more clinical signs & symptoms or laboratory abnormalities. Peripheral cyanosis was noted in 18.5% (5/27) cases. 14.8% (4/27) neonates were plethoric. Other signs & symptoms were tachycardia (14.8%), systolic murmur (7.4%), tachypnoea (14.8%), jitteriness (11.1%), irritability (7.4%), lethargy (11.1%), seizure (11.1%) and vomiting with feeding difficulty (14.8%). Among the biochemical abnormalities, hypoglycaemia was present in 2 neonates (7.4%). Hypocalcaemia was found in 4 (14.8%) patients. Sixteen out of 27 (59.2%) developed jaundice and required phototherapy.

DISCUSSION

Early studies of hyper viscosity & polycythaemia described symptomatic neonates, who were identified because of their illness. It was not until Writh et al, screened a University hospital population of Colorado by estimation of peripheral haematocrit that incidence of neonatal polycythaemia was determined.[3] They found 4% of neonates having venous haematocrit ≥65%. But all neonates were not screened. Polycythaemia was found in 6.7% of neonates in our study. Singh S et al have reported 3.06% incidence.[4] But they also did not screen all the neonates for polycythaemia.

In SGA neonates raised Cap. HCT in comparison to AGA neonates has been reported in different studies because SGA is a common manifestation of chronic placental insufficiency and chronic foetal hypoxia leading to increased erythropoiesis in the foetus.[5,6] This has been confirmed in this study also.

Neonatal polycythaemia occurs either as a result of intrauterine hypoxia or following excessive perinatal transfusion.[7,8,9] Intrauterine hypoxia leads to increased erythropoiesis in the foetus & there by polycythaemia. Although a number of conditions have been reported to cause increased foetal erythropoiesis & polycythaemia, the most common are those associated with placental insufficiencies and chronic intrauterine hypoxia. Pre-eclamptic toxaemia, eclamptic toxaemia, pregnancy induced hypertension and Oligohydramnios, all indicate chronic hypoxia of the foetus.[10] So, all these lead to increased incidence of polycythaemia in new-borns. This has been confirmed by the findings of this study also. In this study 18.5% of polycythaemic neonates had maternal history of either pre-eclamptic toxaemia or eclamptic toxaemia or pregnancy induced hypertension & this association was statistically significant ($p<.01$). There was also association of Oligohydramnios & foetal bradycardia in this study. Congenital malformations & chromosomal anomalies have been reported to be associated with polycythaemia in different studies.[9] But in this study no chromosomal abnormality was noted in any polycythaemic neonate. Only one polycythaemic neonate was with cleft lip & palate.

The neonates of diabetic mothers are more prone to develop polycythaemia because of hyperinsulinism leading to increased oxygen consumption and chronic hypoxia resulting in increased level of foetal erythropoietin. In this study 11.1% of polycythaemic neonates were with history of maternal diabetes mellitus ($p<.01$). Other maternal diseases, those were found in this study were valvular heart disease and hypothyroidism.

The clinical symptomatology of neonatal polycythaemia is nonspecific.[11,12] Many other perinatal conditions can mask or produce similar signs & symptoms. But the presence of two or more signs & symptoms are more commonly associated with polycythaemia & hyper viscosity as opined by Ramamurthy et al.[2] Though, it does not ensure diagnosis of polycythaemia. Most reports states that 50% or more of polycythaemic neonates are asymptomatic, particularly those diagnosed during routine neonatal screening.[13,14] This has been confirmed by this study also. 87.8% of polycythaemic neonates were asymptomatic in this study also. Rest 22.2% polycythaemic neonates were symptomatic. In a study carried out by Singh M et al, 29.6% neonates were symptomatic.[13]

Case studies of polycythaemic neonates has revealed predominance of cardiorespiratory signs & symptoms.[7,9] This has been confirmed in this study also. Peripheral cyanosis, tachypnoea & tachycardia were the three most common clinical features in this study. Other clinical features in this study were lethargy, jitteriness, feeding difficulty, seizure etc. Hypoglycaemia hypocalcaemia and hyperbilirubinemia are the common laboratory abnormalities in polycythaemic neonates.[2,4,7,9] In our study commonest laboratory abnormality was hyperbilirubinemia. 80% of polycythaemic neonates developed clinical jaundice of which 25% needed phototherapy.

CONCLUSIONS

Polycythaemia is an important neonatal health problem, affecting an impressive number (6.7%) of neonates who require special care. So all neonates, born out of normal or high risk pregnancy should be screened for polycythaemia. SGA & LGA are more prone to develop polycythaemia. Premature neonates are also equally likely to develop polycythaemia, like that of term neonates contrary to the usual belief.

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