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Risk factors and morbidity associated with neonatal polycythemia in the last 7 years

Abstract

Introduction: Neonatal polycythemia is defined as a venous hematocrit higher than 65%, and it's a common problem in newborns. An increased incidence is seen in infants born postterm or small for gestational age. There are some Pregnancy-related conditions associated with chronic fetal hypoxia like pregnancy-induced hypertension, maternal smoking fetal hyperthyroidism, diabetic mothers, recipient twins in twin-to-twin transfusion syndrome, and those who have genetic disorders such as chromosomal abnormalities like trisomy 13, trisomy 18, trisomy 21, and Beckwith-Wiedemann syndrome are at higher risk for developing polycythemia. Although the cause of polycythemia is often multifactorial, we can classify the cause of polycythemia in the infants as an active which is an increased fetal erythropoiesis including placental insufficiency due to preeclampsia, maternal chronic hypertension, maternal congenital heart disease, maternal smoking or passive that refers to an erythrocyte transfusion which includes placental-fetal transfusion with delayed cord clamping, relative positioning of the delivered infant in relation to the maternal introitus, asphyxia, and Twin-to-twin transfusion syndrome. As the venous hematocrit rises above 65%, the thickness or viscosity of whole blood also increases and polycythemia can impair microcirculatory flow in the organs and can present with neurologic, cardiopulmonary, gastrointestinal, and metabolic symptoms.

It's important to know which infants have associated factors, because they have to be monitored closely. Asymptomatic infants can be monitored closely and be hydrated with a significant incrementation with enteral intake or administration of intravenous fluids. The hematocrit, bilirubin and glucose should be analyzed in the next 12 to 24 hours. Partial exchange transfusion has been used to treat both symptomatic and asymptomatic patients and the potential benefit in symptomatic infants depends on the symptoms.

Objective: determine the associated risk factors in neonatal polycythemia and their impact in the infants of the service of neonatology at the National Institute of Perinatology in the last 7 years.

Material and methods: This is a retrospective, observational descriptive study based on universal screening of neonatal polycythemia and associated risk factors and morbidity. Polycythemia was defined as venous hematocrit 65%.

Results: The study population consisted of 110 consecutive term polycythemic infants with a mean gestational age of 36 ±3.1 weeks of gestation, with a minimum of 26 and a maximum of 40.5 weeks, In which 36.4% of the patients were female and 63.4% male. The most frequently associated maternal comorbidity was preeclampsia in a total of 39 patients, with a higher frequency of preeclampsia with severity data. The most frequent comorbidity followed was diabetes mellitus, followed in frequency by maternal obesity. The most frequent placental pathology was chorangiosis in 29.1%, followed by decidual infarcts in 19.1%. The most frequent way of delivery was cesarean section in 80.9% of cases. The initial measurement of hemoglobin had a mean of 22.14 ± 1.48 and hematocrit of 68.86 ± 3.29. The most frequent clinical data was hyperbilirubinemia in 90% of the cases, followed by ruddiness in 84.5%. Regarding treatment, 83.6% required phototherapy, and Partial exchange transfusion was documented in 12.7% of the study population. Gestational age had a statistically significant association with saline exchange (Krustal wallis p=0.006). Advanced reanimation was statistically significantly associated with Partial exchange transfusion (chi-square p= 0.043). Other factors associated in a statistically significant way with saline exchange. Redness had a statistically significant association with hematocrit (mean 69.1% in patients with ruddiness vs 67.3% in patients without it (Krustal wallis p=0.017).

Conclusions: The controversy and the need for continued research envelope the issues of which infants are at risk and need to be treated. This study shows the associated risk factors and morbidity of neonatal polycythemia.

Keywords: neonatal polycythemia, hematocrit, hyperviscosity

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Introduction

Background

Polycythemia in newborns is first mentioned in the Bible in Genesis 25:25 with the birth of Isaac and Rebekah's twins, where the first baby is described as having pale skin and the second as having a ruddy complexion.[1,2]

In the 1970s, some case reports and small series of newborns were published with signs and symptoms that at that time were thought to be secondary to elevated hematocrit and blood viscosity.[1]

In 1980, research began into the possible relationship between polycythemia and hyperviscosity, and how their effects, if left untreated, can lead to complications in various organs due to a significant increase in hematocrit, viscosity, and arterial oxygen content.[1]

Neonatal polycythemia is defined as a venous hematocrit above 65% or a hemoglobin level above 20 g/dL in capillary blood samples; the upper limit for HCT is 75% and for HGB is 23.7 g/dL.[3]

It will be characterized by a venous hematocrit that exceeds the normal values for the gestational age of the newborn and is greater than two standard deviations from the normal values for gestational and postnatal age.[3]

Henry and Christensen, in 2015 They generate the definition of polycythemia proposing that it is fulfilled when the hematocrit, the blood concentration ¬of hemoglobin or the red blood cell count or all three are above the upper reference interval of the 95th percentile for gestational and postnatal age.[3–7]

This value is taken into account for both sexes in the neonatal population. Although some studies suggested that male neonates had a higher hematocrit than female neonates, there are some more extensive studies of both sexes in which no significant differences were found in hematocrit or hemoglobin concentration.[3]

The etiology of polycythemia is related to intrauterine hypoxia or hypoxia secondary to fetal transfusion. Infants who experience chronic or acute fetal hypoxia have a higher incidence.[1] Increased blood viscosity is associated with symptoms of hypoperfusion.[3,4]

Its occurrence has been observed in approximately 3% of all newborns at sea level, with a prevalence slightly exceeding 5% in newborns at higher altitudes.[3]

The incidence increases in neonates who are small for gestational age and in neonates who are large for gestational age.[1,4]

Several articles report that the prevalence is approximately 1 to 5% of live births, influenced by gestational age, birth weight, and birth altitude.[1] Thus, it occurs in 2% to 4% of term newborns for gestational age, 10% to 15% of small for gestational age newborns, and 6% to 8% of large for gestational age newborns.[5,6] It is rare in premature newborns less than 34 weeks of gestational age because hematocrit increases progressively with gestational age.[1,5,6,7]

However, most newborns with polycythemia are of appropriate size or weight for their gestational age. At sea level, the incidence of polycythemia and hyperviscosity is 1–2%, while above 430 meters it has been found to be as high as 5%. There is a higher incidence in neonates who have suffered chronic or acute fetal hypoxia.[4–6]

The incidence of hyperviscosity in infants born to mothers with diabetes is reported in some articles as 10 to 30%, while in others it is reported as over 40%, and in infants born to mothers with gestational diabetes, the incidence is greater than 30%. Hyperviscosity occurs in 6.7% of neonates.[1,2,4]

Most patients do not present with clinical manifestations and the condition is asymptomatic. Clinical manifestations result from hyperviscosity secondary to the increase in red blood cell mass.[7]

When mentioning the term polycythemia, it is important to clarify that it is not synonymous with hyperviscosity, since the latter constitutes an increase in resistance to blood flow, which is generated by multiple factors.[8]

Hematocrit is the main determinant of blood viscosity, but not the only one.[8,9] Blood pH is also a determining factor because if it is less than 7, it will cause red blood cells to deform, increasing their effect on blood viscosity.[9] Hyperviscosity is caused by an increase in the internal friction of the blood required to achieve flow. Polycythemia, on the other hand, is caused by an abnormal increase in the number of erythrocytes.[1,9]

However, polycythemia should be distinguished from hyperviscosity, which is defined as a blood viscosity >12 cP measured at a shear rate of 11.5 per second; or >6 centipoises, measured at a shear rate of 106 per second.[8,9]

The relationship between hematocrit and viscosity is almost always linear with a hematocrit up to 65% and exponential above this value.[8]

The terms neonatal polycythemia and hyperviscosity have often been used interchangeably; however, this is incorrect, as polycythemia can occur with or without hyperviscosity in many cases, and vice versa.[9,10]

A minority of infants with polycythemia have hyperviscosity. Conversely, some infants with hyperviscosity are not polycythemic. Forty-seven percent of patients with polycythemia have hyperviscosity, and only 24% of patients with hyperviscosity are diagnosed with polycythemia.[9–11]

The viscosity of whole blood is affected by multiple factors, including red blood cell mass, plasma components, proteins, pH, and the interaction of cellular components with the blood vessel wall.[10,11]

Hyperviscosity will contribute to the signs and symptoms observed in approximately half of the patients with polycythemia.[11]

Only 47% of patients with polycythemia have hyperviscosity, and only 24% of patients with hyperviscosity will have a confirmed diagnosis of polycythemia.[11]

As hematocrit increases, viscosity also increases, resulting in abnormalities in blood flow kinetics. This increase in red blood cell mass is a fetal response to increased hemoglobin production in a relatively hypoxic intrauterine environment, which manifests as impaired flow and intravascular agglutination, predisposing to microthromb formation and generating a significant decrease in tissue oxygenation, giving rise to the clinical manifestations in infants.[11]

Although blood viscosity could be a useful guide for deciding on appropriate management strategies in affected patients, viscosity measurement is not widely available in many clinical settings, and therefore clinical decisions are based on hematocrit measurement.[12]

Perinatal asphyxia and acute fetal hypoxia remain important causes of polycythemia.[1]

Pregnancy-related conditions associated with chronic fetal hypoxia considered risk factors for developing neonatal polycythemia include maternal diabetes, systemic or pregnancy-induced hypertension,

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hyperthyroidism, maternal smoking, high altitude, intrauterine growth restriction, advanced maternal age, use of antihypertensive drugs such as propranolol , heart disease, and kidney disease.[13–15] Placental factors that can influence fetal hematocrit include placental infarction, placenta previa, and viral infections, especially respiratory infections.[16]

All of these factors have been associated with an increased risk of neonatal polycythemia. However, not all infants exposed to adverse intrauterine conditions will develop polycythemia.[1,16]

Not all cases of polycythemia develop due to hypoxia or prolonged oxygen deprivation. Many may have acute or chronic placental transfusions due to delayed clamping of the umbilical cord or arteriovenous malformations. Still others may have fetal conditions that contribute to the development of polycythemia. In the vast majority of infants, there is no identifiable cause.[16]

Neonatal polycythemia is usually due to one of two possible situations: increased intrauterine erythropoiesis or fetal hypertransfusion. Other causes observed in older children, such as arterial hypoxemia in patients with cyanotic heart disease or chronic lung disease, abnormal hemoglobins, or hypersecretion of erythropoietin by tumors, are rare, and polycythemia vera is practically nonexistent in neonates.[3]

The increase in hematocrit is due to 3 mechanisms:

1. Passive, secondary to transfusion of red blood cells from other vascular beds;

2. Active, due to intrinsic red blood cell production; and

3. Hemoconcentration as a consequence of volume depletion.[8,17,18,21]

(i) Passive red blood cell transfusion via placentofetal transfusion can be associated with delayed umbilical cord clamping. A meta-analysis published in 2007 by Hutton et al. demonstrated an increase in mean hematocrit in term births where umbilical cord clamping occurs after 2 minutes of life compared to those where it is performed in less than 10 seconds.[18] However, although delayed clamping is associated with a moderate increase in polycythemia and blood hyperviscosity rates, there is no evidence of significant clinical effects. In term neonates, a decreased risk of anemia and iron deficiency is observed in both the short and medium term, and in preterm neonates, a decreased need for transfusions due to anemia or hypotension and a lower incidence of intraventricular hemorrhage.[17,18] The risks of delayed umbilical cord clamping could be exacerbated; however, it is important that obstetrics and gynecology staff position the newborn at an appropriate height when clamping the umbilical cord due to the risk of causing neonatal anemia or polycythemia.[18,21]

Regarding fetoplacental transfer, Phil et al. demonstrated that placental volume decreases in situations of acute hypoxia.[16] The explanation for this phenomenon is based on a vasoconstriction mechanism of the placental vascular bed, resulting in a transfer of blood volume from the placental vessels into the fetal bloodstream.[16–18]

Secondly, twin-to-twin transfusion syndrome can occur in monochorionic twins, in which arteriovenous or arterial communications can allow the transfer of a larger volume of blood to one twin, causing anemia in the other.[19]

In normal, full-term newborns, a delay in clamping the umbilical cord of 3 minutes or more can occasionally lead to the transfer of a sufficient amount of fetal blood to cause polycythemia. Placental insufficiency and chronic intrauterine hypoxia, as typically seen in small-for-gestational-age newborns, can induce an increase in ¬Epo-mediated erythropoiesis.[3]

(ii) Increased erythropoiesis is the active mechanism for increasing hematocrit. Physiologically, this is more pronounced during the fetal period in response to a lower partial pressure of oxygen. However, all diseases that cause uteroplacental insufficiency will result in intrauterine growth restriction because they sustainably affect fetal oxygenation, stimulating erythropoietin production in the fetus.[14–16]

Multifactorial gestational diabetes has also been associated with an increased incidence of polycythemia, ranging from 10 to 15%.[19,20]

Maternal diabetes is associated with an increased risk of intrauterine hypoxia.[20] These newborns have a high prevalence of polycythemia, elevated erythropoietin levels, and reduced iron and ferritin levels. Hod et al., showed a higher prevalence of polycythemia in infants of diabetic mothers (13.3%) compared to controls (4.9%).[19,21] However, hypoxia and polycythemia can be prevented with good maternal glycemic control.[20] Fetal hyperthyroidism is associated with an increased fetal metabolic rate and produces chronic hypoxia and polycythemia in the newborn.[14–16] Another factor related to hypoxia is maternal smoking during pregnancy.[14,15]

In these cases, tissue hypoxemia occurs due to an increase in carbon monoxide content, which competes with oxygen for binding to hemoglobin.[16]

(iii) Decreased plasma volume, primarily related to low feeding intake, increases hematocrit due to hemoconcentration. This third mechanism acts beyond the immediate neonatal period, unlike the previous ones, and it is important to differentiate it from neonatal polycythemia, which requires a different approach and management. Both require timely management and increased fluid intake, either enterally or via IV.[21]

Fetal hypoxia

Perinatal asphyxia and acute fetal hypoxia remain important causes of polycythemia.[1,16] Acute intrauterine hypoxia results in a shift of blood from the placental compartment to the fetus.[22] Felipe et al., examined residual placental volumes and neonatal outcomes.[23] Fetal distress and low Apgar scores were associated with low residual placental volumes.[1] There is a correlation between the duration of hypoxia and the volume of blood shifted into the fetal compartment. The data also suggest that fetal vasodilation associated with acute fetal hypoxia is responsible for this change in blood volume.[1,22,23]

Intrauterine growth restriction

Environmental factors such as maternal hypertension, diabetes, and smoking suggest that fetal hypoxia may play a key role in the development of intrauterine growth restriction. Kramer et al.,[14] studied a large cohort of 8719 unique infants who had no evidence of congenital infection, chromosomal abnormalities, or other major malformations, while controlling for the severity of intrauterine growth restriction. They found fetal polycythemia in 7.5% of patients without IUGR compared to 41.5% of newborns with severe IUGR. Fetal polycythemia, defined as a peak capillary hemoglobin level of -21 g/dL, was observed in 7.5% of newborns without IUGR, compared to newborns with severe IUGR.[14,16]

Cord clamping and cord milking

Saigal and Usher first raised concerns about delayed cord clamping contributing to polycythemia in 1977.[25]

More recent randomized studies have confirmed higher hematocrit levels in both preterm and term newborns with delayed cord clamping compared to early cord clamping, described as at thirty seconds.[16,25]

Delayed cord clamping allows for a greater volume of blood to be delivered to the baby.[1,16] Clamping the umbilical cord towards the newborn leads to significant placental transfusion, resulting in an increase in blood volume and red blood cell mass, especially if the newborn is kept below the level of the placenta.[16,25]

Gravity, due to the newborn's position relative to the maternal introitus, and the release of oxytocin may also be contributing factors that increase the volume of blood transfused into the newborn's circulation.[16]

It has been observed that delaying cord clamping for more than 3 minutes increases blood volume by approximately 30%, resulting in polycythemia and hyperbilirubinemia.[16,25]

Several studies have examined the incidence of polycythemia as a potential complication of delayed umbilical cord clamping. One study by Rincon D, Foguet A, Rojas M, et al., involving 242 newborns whose cords were clamped in less than 60 seconds, between 1 and slightly less than 2 minutes, or between 2 and 3 minutes after birth, reported hematocrit levels at 48 hours postpartum of 53%, 58%, and 59%, respectively.[27] Ferritin and hemoglobin levels also increased in relation to delayed cord clamping. Furthermore, the number of infants with polycythemia was significantly higher in the group clamped at 2–3 minutes; however, the authors noted that none of the patients who presented with symptoms related to polycythemia and hyperviscosity required additional treatment.[26]

A more recent study of 73 infants showed that delayed cord clamping up to 5 minutes after birth did not lead to an increased incidence of polycythemia compared with early cord clamping.[27] Another study comparing early cord clamping within 10 seconds of birth with delayed clamping at 3 minutes or later found no difference in the incidence of polycythemia at 4 months of age.[28] Therefore, although delayed cord clamping increases hematocrit levels, the currently available evidence indicates that there is a minimal risk of symptomatic polycythemia requiring treatment.

However, there is concern that cord clamping may be delayed for more than a few minutes or that the baby may have other risk factors that could lead to polycythemia and require more invasive treatment.[16,27,28]

There is a case report in the literature of severe symptomatic polycythemia following a water birth in which the cord was clamped after 40 minutes.[16] Furthermore, Linderkamp et al.,[32] reported a marked increase in viscosity in infants whose cords were clamped late. Milking the umbilical cord is not recommended because the volume of blood that may pass to the newborn is unknown, potentially leading to the development of symptomatic polycythemia.

Twin-to-twin transfusion syndrome

Twin-to-twin transfusion syndrome due to a vascular communication occurs in approximately 10% of monozygotic twin pregnancies. In intrapartum asphyxia, blood volume shifts from the placenta to the fetus.[31–34]

Monochorionic twin pregnancies Diamniotic twins with amniotic fluid discordance appear to almost double the risk of developing twin anemia-polycythemia sequence.[31] They have a higher risk of perinatal mortality and morbidity than dichorionic twin pregnancies, primarily due to twin-to-twin transfusion syndrome, in which they will have selective fetal growth restriction and anemia-polycythemia sequence.[31,32,34]

Intertwin transfusion without amniotic fluid volume disparity between the donor and recipient twins.[31]

While significantly discordant hemoglobin levels and reticulocyte counts between twins comprise the postnatal diagnostic criteria for anemia-polycythemia sequence, prenatal diagnosis is based on the presence of abnormalities on Doppler ultrasound without signs of polyhydramnios in the recipient twin or oligohydramnios in the donor twin.[33,34] The peak systolic velocity of the fetal middle cerebral artery is increased in the donor twin, suggesting fetal anemia, while it is decreased in the recipient twin, consistent with polycythemia.[31–34]

Monochorionic twin pregnancies and,[31–34] due to residual anastomoses, after laser surgery in 2 to 16%.

High blood pressure in the mother

Preeclampsia and maternal hypertension represent a significant risk of Neonatal polycythemia.[13,16]

Neonatal polycythemia is a frequent finding in pregnancies complicated by diabetes and maternal hypertension with intrauterine growth restriction.[16] It is still unclear whether the association of polycythemia with hypertension is a result of intrauterine growth restriction or whether hypertension alone is involved. To establish the incidence of neonatal polycythemia in at-risk populations, a study was conducted by Kurlat and Sola.[16,17]

Of 1592 neonates born consecutively at the Hospital de Clínicas in Buenos Aires,[17] it was reported that the risk of polycythemia was 12.6 times higher in babies of hypertensive mothers who were of appropriate gestational age compared to non-hypertensive mothers.[16,17]

These data show that maternal hypertension presents a significant risk of polycythemia, independent of fetal growth. This study suggests routine blood counts in infants of mothers with hypertension to prevent possible sequelae and to allow for timely diagnosis and treatment.[16–18]

Another factor related to hypoxia is the use of beta-blockers, since these medications significantly reduce adequate placental perfusion. It has been suggested that beta-blockers without intrinsic sympathomimetic activity cause selective vasoconstriction of placental blood vessels.[16,17] However, there is still insufficient evidence.

Maternal diabetes

The prevalence of polycythemia in children of diabetic mothers has been found to range from 5% to 40%. Fetal hyperglycemia will generate significant catabolism, requiring increased oxygen consumption and leading to a reduction in oxygen tension. During pregnancy, the fetus will remain in a state of relative hypoxemia, which will stimulate erythropoiesis, thus producing polycythemia.[3,16]

Some small studies have indicated an association between maternal diabetes control, depending on HbA1c levels per trimester and neonatal hematocrit.[3]

In a large prospective longitudinal study by Hod et al.,[21] the prevalence of polycythemia in children of diabetic mothers was 13.3%, significantly higher than that of controls (only 4.9%).[16,20]

An increase in erythropoietin concentration in the amniotic fluid of mothers with diabetes has also been reported, and this increase has also been observed at birth.[3]

Fetal risk factors

Fetal risk factors associated with polycythemia include chromosomal abnormalities such as trisomy 13, 18 and 21, congenital adrenal hyperplasia, hypo- and hyperthyroidism, Beckwith-Wiedemann syndrome, hemoglobin disorders, erythropoietin receptor defects, cyanotic congenital heart disease and perinatal asphyxia (Table 1).

Table 1 Risk factors associated with the development of neonatal polycythemia

Advanced maternal age[1,3,4,16]	IUCN[1,3,4,16]	Oligohydramnios[1,3,4,16]
Smoking[1,3,4,16]	Substance abuse[1,3,4,16]	Diabetes mellitus[1,3,4,16]
Preeclampsia[1,3,4,16]	Kidney diseases[1,3,4,16]	Maternal cyanogenous heart disease[1,3,4,16]
Use of propranolol[1,3,4,16]	Placental infarction[1,3,4,16]	TORCH viral infections[1,3,4,16]
Trisomy[13,18,21,1,3,4,16]	Hyperthyroidism[1,3,4,16]	Hypothyroidism[1,3,4,16]
Beckwith wiedemann syndrome[1,3,4,16]	Perinatal asphyxia[1,3,4,16]	Delayed pinching[1,3,4,16]
Newborn below the maternal introitus[1,3,4,16]	Transfusion fetus fetus[1,3,4,16]	Systemic arterial hypertension[1,3,4,16]

Clinical presentation and complications

Infants with polycythemia often exhibit increased blood viscosity. As the hematocrit rises above 65%, there may be a greater tendency toward decreased blood flow due to changes in red blood cell mass, particularly in the cerebral, hepatic, renal, and mesenteric microcirculation. Clinical symptoms may include lethargy, cyanosis, respiratory distress, nervousness, hypotonia, feeding intolerance, hypoglycemia, and hyperbilirubinemia.[1,16]

Polycythemia has a wide range of complications, including numerous organ systems, and 50% of newborns with polycythemia develop one or more symptoms.[1,16] Furthermore, most of these symptoms are nonspecific and can be attributed to the underlying conditions.[1,2,3,4,16] However, any newborn with a component suggestive of polycythemia or who has identifiable risk factors should be screened for polycythemia because newborns with polycythemia may be at increased risk of complications resulting from hyperviscosity; these include ischemia and infarction, both in the kidneys and the central nervous system. These complications are attributable to hyperviscosity (Table 2).[1,2,3,4,16]

Table 2 Clinical presentation Neonatal Polycythemia

CNS[1,3,4,16]	Lethargy
	Apnea
	Seizures
	Tremors
	Irritability
	Poor suction
Hematological[1,3,4,16]	Thrombocytopenia
	Increased erythroblasts
	CID
	increased reticulocytes
Cardiac[1,3,4,16]	Tachycardia
	Plethora
	Congestive heart failure
	Cardiomegaly
Respiratory[1,3,4,16]	Polypnea
	Apnea
	Difficulty breathing
	Desaturation
	Increased need for supplemental oxygen
	Increased pulmonary vascular resistance
Metabolic[1,3,4,16]	Hypoglycemia
	Hyperbilirubinemia
	Hypocalcemia
	Vitamin D deficiency
Gastronutritional[1,3,4,16]	Enterocolitis
	Food intolerance
	vomit
Dermatological[1,3,4,16]	Ruddiness
	Jaundice
	Plethora
	Delay in capillary refill
Renal[1,3,4,16]	Oliguria
	Proteinuria
	Hematuria
	Renal vein thrombosis
	Acute renal failure

Neurological

Previously, it was believed that polycythemia and hyperviscosity of the blood caused cerebral hypoxia and ischemia due to reduced cerebral blood flow resulting from the accumulation of red blood cells within smaller blood vessels. However, a series of cases conducted between 1980 and 1995 clarified the changes in cerebral blood flow, oxygen delivery, and glucose utilization.[1] Rosenkrantz and Oh[8] used Doppler techniques to demonstrate reduced cerebral blood flow in newborns with polycythemia, which returned to normal after partial exchange transfusion.[1,8,16,34]

To understand the factors responsible for the reduction in cerebral blood flow, they used newborn lambs in which they observed that changes in cerebral blood flow resulted from an increase in CaO_2, a normal physiological response that correlated with an increase in hematocrit.[1,8,16,34]

Therefore, the decrease in cerebral blood flow in the newborn with polycythemia is a physiological response and does not cause cerebral ischemia.[1,8]

Relevant symptoms include tremors, irritability, agitation, as well as seizures and intracerebral hemorrhages. It occurs in approximately 60% of cases[16] due to a decrease in blood glucose caused by increased consumption.

Polycythemia and hyperviscosity can have lasting effects on neonatal neurodevelopment.[16] Ratrisawadi and colleagues 40 studies found a global developmental delay at 1.5 to 2 years of age in children with a history of neonatal polycythemia. They found gross motor delays, fine motor abnormalities, and speech delays in 2-year-old children with a history of neonatal polycythemia compared to age-matched controls.

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Neonatal hyperviscosity has also been linked to neurological and cognitive impairment in older children. In a study of 7-year-old children with a history of hyperviscous umbilical cord blood, Drew et al.,[33] 41 found a higher likelihood of an abnormal neurological examination and motor index as assessed by the McCarthy Scales of Infant Abilities. Delaney-Black et al.,[34] 42 found Lower performance scores in school-aged children who had a history of polycythemia as newborns compared with controls.[1,16,33,34]

Important risk factors included fetal distress, asphyxia, hypoglycemia, uncontrolled precipitous delivery, and maternal preeclampsia. Furthermore, the initial intrauterine events leading to polycythemia are likely the cause of both the polycythemia and the developmental problems.[1,16,33]

Cardiopulmonary

There is a decrease in cardiac output secondary to an increase in arterial oxygen content. Systemic oxygen transport, supply, consumption, and blood pressure are normal.[1] Pulmonary vascular resistance increases, and pulmonary blood flow decreases. This is thought to be due to changes in blood viscosity. The decrease in pulmonary blood flow may cause respiratory distress and cyanosis. It may be reversed by reducing hematocrit and blood viscosity.[1,16]

In contrast to the central nervous system, there is no evidence of subsequent cardiopulmonary complications from neonatal polycythemia. Short-term pulmonary problems may include tachypnea, respiratory distress, pulmonary vascular congestion, pleural effusions, and pulmonary hypertension.[16]

The symptoms originate from an increase in blood viscosity and an elevation of pulmonary resistance, which leads to an increased right ventricular ejection time and a decrease in the amount of blood the ventricles pump per minute due to a deficit in stroke volume. As a result, the neonate exhibits a decreased heart rate with reduced capillary refill, increased respiratory rate, cyanosis, and a plethoric appearance.[1,16,28]

Metabolic

Hypoglycemia and hypocalcemia are the most frequent metabolic abnormalities observed in infants with neonatal polycythemia and hyperviscosity. Hypoglycemia occurs in 12% to 40% of polycythemic infants.[16]

It may be mediated by contributing factors such as maternal diabetes. The pathogenesis of hypocalcemia observed in polycythemic infants is less clear. Saggese and colleagues 47 proposed that it may be related to elevated levels of calcitonin gene-related peptide.[16]

Newborns with polycythemia are at risk of hypoglycemia. It is unclear whether this is due to decreased gluconeogenesis or increased utilization. Glucose is present in the plasma fraction of the blood. Since many infants with polycythemia have a reduced plasma volume, the whole blood glucose concentration may be significantly reduced even when the plasma concentration is normal.[1]

Negro et al.,[1,16] found that concurrent hypoglycemia placed infants with polycythemia at the greatest risk of impaired neurological function.

The finding of a decrease in cerebral glucose supply and uptake associated with normoglycemia leads to speculation that this could be one of the reasons for the neurological sequelae observed in human polycythemic newborns.[16]

Lower levels of renal cholecalciferol metabolites have also been described in infants with polycythemia/hyperviscosity. Both 1,25-dihydroxyvitamin D and 24,25-dihydroxyvitamin D were significantly lower in infants with polycythemia compared to healthy controls.[16]

A significant increase in red blood cell mass will favor hemoglobin catabolism, and hyperbilirubinemia commonly develops.[16]

Gastronutrition

Neonates exhibit an increased concentration of products metabolized by the liver from cholesterol, such as bile acids, causing feeding disorders, vomiting, and, as a more serious complication, necrotizing enterocolitis.

In a randomized study, Black et al. observed that 6% of untreated infants with polycythemia had gastrointestinal symptoms, while 51% of those who received salinopheresis had severe gastrointestinal symptoms, including enterocolitis.

Hematological

Thrombocytopenia may be observed. In a review study from the Netherlands, thrombocytopenia occurred in 51% and severe thrombocytopenia affected 91% of 140 newborns with polycythemia.[13]

Ultimately, polycythemia increases blood density, impairing microcirculatory flow and leading to neurological, gastrointestinal, cardiopulmonary, renal, thrombotic, and metabolic manifestations.[13,14]

Nephrourinary

In the kidneys, there is an alteration in renal flow and a decrease in glomerular filtration, causing fluid retention, decreased diuresis, hematuria, proteinuria, and thrombosis of the renal vein. Alkalay and colleagues[35] It was suggested that hyperviscosity may interfere with the kidneys' ability to convert 25-hydroxyvitamin D into its dihydroxylated metabolites.[1,16,34]

Treatment

Currently, treatment is initiated according to symptoms and clinical situation; these recommendations are not based on hematocrit levels, but rather primarily on the patient's clinical status and multisystem involvement.

The published measures consist of intravenous fluid administration and the possible consideration of partial volume exchange transfusion in some severe cases.[1,16]

The management of asymptomatic neonatal polycythemia is debatable due to a lack of evidence demonstrating that aggressive treatment improves long-term outcomes. Before determining the treatment for polycythemia, it is essential to confirm that the patient is experiencing true polycythemia and not dehydration or blood transfusion. Complete laboratory tests should always be performed to determine if the patient is experiencing associated hypoglycemia, hypocalcemia, or hyperbilirubinemia, and these should be treated immediately.[1,16]

Two treatment patterns have been described for asymptomatic and symptomatic polycythemia: conservative management with rehydration and partial exchange transfusion.

If the etiology is dehydration, rehydrate the patient over 6-8 hours.[11]

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Systematic literature reviews were conducted by De Waal et al. and Dempsey and Barrington. The objective of both reviews was to determine whether crystalloid solutions were as effective as colloid solutions when performing partial exchange transfusion in neonates with polycythemia. The conclusion of both reviews was that crystalloid solutions are as effective as colloid solutions. Crystalloid solutions have the additional benefits of no risk of transmitting blood borne diseases or causing anaphylaxis, rapid and easy availability, and lower cost. Therefore, crystalloid solutions should become the standard of care.[1,16]

Partial exchange dilution

a) Perform it through the umbilical catheter when possible[11]

b) The total amount of blood to be exchanged is calculated as follows:

(Actual Hct – Ideal Hct x blood volume/ Real Hct)[11]

Where blood volume is equal to the patient's weight in kilograms multiplied by 80-100 mL.[11]

Physiological saline solution is the fluid of choice.[11]

The American Academy of Pediatrics specifies: "The accepted treatment for polycythemia is exchange dilution. However, there is no evidence that exchange dilution affects long-term outcomes. "[11]

Definition of the problem to be addressed

Polycythemia is a relatively common condition seen in neonatal units, influenced by various maternal-fetal factors. The nonspecific nature of its signs and symptoms often leads to delayed diagnosis and, consequently, delayed medical management, resulting in treatment and the development of serious complications that negatively impact the newborn's quality of life. Therefore, identifying associated risk factors is crucial for timely intervention and the prevention of complications.

Problem statement

It is vitally important to identify the associated maternal and fetal risk factors, as well as the signs and symptoms, to make a timely clinical diagnosis and confirm it with a hematocrit measurement greater than 65% to prevent complications, reducing morbidity and mortality.

Research question

What are the main risk factors associated with developing neonatal polycythemia in newborns at the National Institute of Perinatology in the Neonatology service?

Goals

General objective

Describe the factors associated with neonatal polycythemia in the Neonatology service of the National Institute of Perinatology in the last 7 years.

Specific objectives

To describe the associated risk factors and morbidity in newborns discharged from the neonatology service during January 1, 2016 to May 31, 2023 at the National Institute of Perinatology.

(i) Describe the frequency of maternal pathology associated with neonatal polycythemia.

(ii) Describe the frequency of placental pathology reported in association with neonatal polycythemia.

(iii) Describe the frequency of clinical and biochemical data in patients with neonatal polycythemia.

(iv) Describe the treatment used in the management of neonatal polycythemia.

(v) To determine the existence of an association between clinical, biochemical, and treatment variables.

Materials and methods

Research classification (type of study): Observational, retrospective, retro-elective, and descriptive.

Population Universe

Records of newborns with a discharge diagnosis of neonatal polycythemia

Eligible population

Newborns discharged from the Neonatology service from January 1, 2016 to May 31, 2023.

Inclusion criteria

(i) Locatable records of patients discharged from the Neonatology service of the National Institute of Perinatology with a diagnosis of neonatal polycythemia.

(ii) Both sexes

(iii) A venous hematocrit greater than 65% or a hemoglobin value greater than 22 g/dL (220 g/L).

Exclusion criteria

(i) Records of patients who have been discharged voluntarily before completing treatment

(ii) Incomplete files

(iii) Death

Place or site of study

The participating population will be from the Neonatology service of the National Institute of Perinatology, Isidro Espinosa de los Reyes.

Method

A list of the medical records of patients discharged from the Neonatology service with a diagnosis of Neonatal Polycythemia (P611) was requested from the medical records department. The list was supplemented with the Neonatology service's electronic statistics. The records were located in the medical records department, and the required data were reviewed. The form created for this purpose was completed, obtaining demographic, clinical, hematological, and patient progress data. The data were then compiled into an Excel spreadsheet for processing.

Sample size

A list of the medical records of patients discharged from the Neonatology service with a diagnosis of neonatal polycythemia (P611) will be requested from the medical records department. This includes newborns discharged from the Neonatology service between January 1, 2016, and May 31, 2023.

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Sample calculation

A convenience sampling will be carried out that will include all locatable cases of newborns discharged from the Neonatology service of the National Institute of Perinatology from January 1, 2016 to May 31, 2023.

Statistical analysis

In the first phase, a database was created using Microsoft Excel. This database was then exported to SPSS version 21, the statistical software used for data analysis. The results of the study are presented in tables, charts, or graphs.

Descriptive statistics were performed on qualitative variables, expressed as frequencies and percentages. For quantitative variables, the mean and standard deviation were determined. The parametric or non-parametric distribution of qualitative variables was determined using the Kolmogorov-Smirnov test for one sample. To determine statistical association, the Chi-square test (for qualitative variables). An alpha value of 0.005 was established to determine statistical significance.

Results

One hundred and ten patients were included, with a mean gestational age of 36.3 weeks ±3.1 weeks of gestation, with a minimum of 26 and a maximum of 40.5 weeks of gestation. 36.4% of the patients were female and 63.4% were male.

The most frequently associated maternal comorbidity was preeclampsia, present in 39 patients, with a higher frequency of severe preeclampsia. The next most frequent comorbidity was diabetes mellitus, followed by maternal obesity (Table 3 & 4).

Table 3 Associated maternal comorbidity

	Frequency	Percentage
Diabetes Mellitus	36	32.7
Systemic Arterial Hypertension	21	19.1
Gestational hypertension	2	1.8
Preeclampsia without signs of severity	13	11.8
Preeclampsia with severe characteristics	26	23.6
HELLP syndrome	2	1.8
Obesity	26	23.6
Premature rupture of membranes	8	7.6
Hypothyroidism	7	6.4
Substance abuse	5	4.5
Oligohydramnios	3	2.7
Rh negative non-alloimmunized	3	2.7
Thrombocytopenia	4	3.6
Nephropathic	4	3.6
Advanced maternal age	4	3.6
Cancer	4	3.6
Placenta previa	3	2.7
Cholecystitis associated with pregnancy	2	1.8
Cerebral venous thrombosis in pregnancy	1	0.9
Hydrops	1	.9
Polyhydramnios	1	.9
Post-operative laser photocoagulation	1	.9
Anemia	1	0.9
Thrombocytopenia	2	1.8
Teenager	1	.9
Epilepsy	1	.9
Heart patient	1	.9
Heterozygous protein S deficiency	1	.9
Thrombosis	1	.9

Table 4 Placental pathological findings

	Frequency	Percentage
Chorangiosis	32	29.1
Decidual infarcts	21	19.1
Without alterations	14	12.7
Ischemic chorionic villi	5	4.5
Hypoxia of the trophoblast + hypotrophic	15	13.6
Vascular malperfusion	10	9.1
Hypotrophic with increased nodal volume	13	11.8

Table 4 is the most frequent placental pathology was chorangiosis in 29.1%, followed by decidual infarcts in 19.1%.

In the results the most frequent delivery method was cesarean section in 80.9% of cases, 12.7% were twin pregnancies.

Of all the births of neonates with polycythemia, only 25.5% underwent delayed clamping of the umbilical cord.

In 3.6% of cases, fetal-fetal transfusion was found, in which only one case was laser photocoagulation of the placenta performed.

53.6% presented intrauterine growth restriction, which may be associated with maternal pathology because the most frequent condition was preeclampsia with severe symptoms followed by diabetes mellitus.

Most of the newborns treated only required initial resuscitation steps without the need for supplemental oxygen and could be in rooming-in. Polycythemia was initially suspected due to the flushing and associated risk factors of maternal pathology. These patients, being asymptomatic, were managed with close monitoring and increased fluid intake via enteral route, ensuring feedings.

30.9% of patients required CPAP, of whom 30.9% were admitted to the nicu and required management involving increased enteral and intravenous fluids.

The initial hemoglobin measurement had a mean of 22.14 ±1.48 and the hematocrit measurement of 68.86 ± 3.29

In more than 70% of patients with polycythemia, there was a decrease in platelet count leading to thrombocytopenia with elevated erythroblasts.

The different hematocrit values per day after treatment were taken into account, and the graph shows that the delta change in hematocrit between the last recorded value and the first value was -0.33 with a range of -4.30 to +2.40.

There was no statistically significant difference in hemoglobin delta or hematocrit according to the treatment used (Table 5).

Table 5 Results of the clinical presentation and treatment used

		Frequency	Percentage
	Down syndrome	10	9.1
	Prematurity	9	8.2
Clinical data	Ruddiness	93	84.5
	Polypnea	62	56.4
	Tachycardia	26	23.6

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Table 5

	necrotizing enterocolitis	9	8.2
	Cyanosis	11	10.0
	Hyperbilirubinemia	99	90
	Hypoglycemia	32	29.1
	Brain abscess	1	0.9
	Sepsis	21	19.1
Treatment	Oral administration	93	84.5
	Intravenous Fluids	67	60.9
	Salinopheresis	14	12.7
	Phototherapy	92	83.6

The most frequent clinical finding was hyperbilirubinemia in 90% of cases, followed by flushing in 84.5%. Similarly, symptomatic patients began to present with apnea, desaturation, tachypnea, or increased oxygen requirements; tachypnea was documented in 56.4% and tachycardia in 23.6%. Only 10% presented with cyanosis.

8.2% presented enterocolitis during their hospitalization, not during the event of neonatal polycythemia, as well as neonatal sepsis in 19.1%.

It was documented that 29.1% of patients presented with hypoglycemia, both symptomatic and asymptomatic with respect to polycythemia. Management involved increasing enteral nutrition, which resolved the hypoglycemia. Only one case was reported of a patient in the NICU who required salinopheresis and management of hypoglycemia due to persistent signs and symptoms, which resolved with salinopheresis.

Regarding treatment, 83.6% required phototherapy, and salinopheresis was documented in 12.7% of the study population.

The performance of advanced resuscitation was statistically significantly associated with salinopheresis (chi square p= 0.043).

Other factors statistically significantly associated with salinopheresis were those described in the following Table 6:

Table 6 Saline apheresis procedure performed according to clinical data

	Salinopheresis	**Salinopheresis**	**p-value**
	Yeah N=14	**No N= 94**	
Advanced resuscitation	4	54	0.043
CPAP	7	26	0.020
Polypnea	14	48	0.001
Tachycardia	12	14	<0.001
NEC	5	4	<0.001
Cyanosis	5	6	0.001
Hypoglycemia	9	23	0.002
Oral administration	4	89	<0.001
No oral intake	10	5	<0.001
Brain abscess	1	0	0.009
Sepsis	9	12	<0.001

CPAP, continuous positive airway pressure; NEC, Necrotizing enterocolitis

Redness had a statistically significant association with hematocrit (mean of 69.1% in patients with redness vs 67.3% in patients without redness (Ttest) (Figure 1).

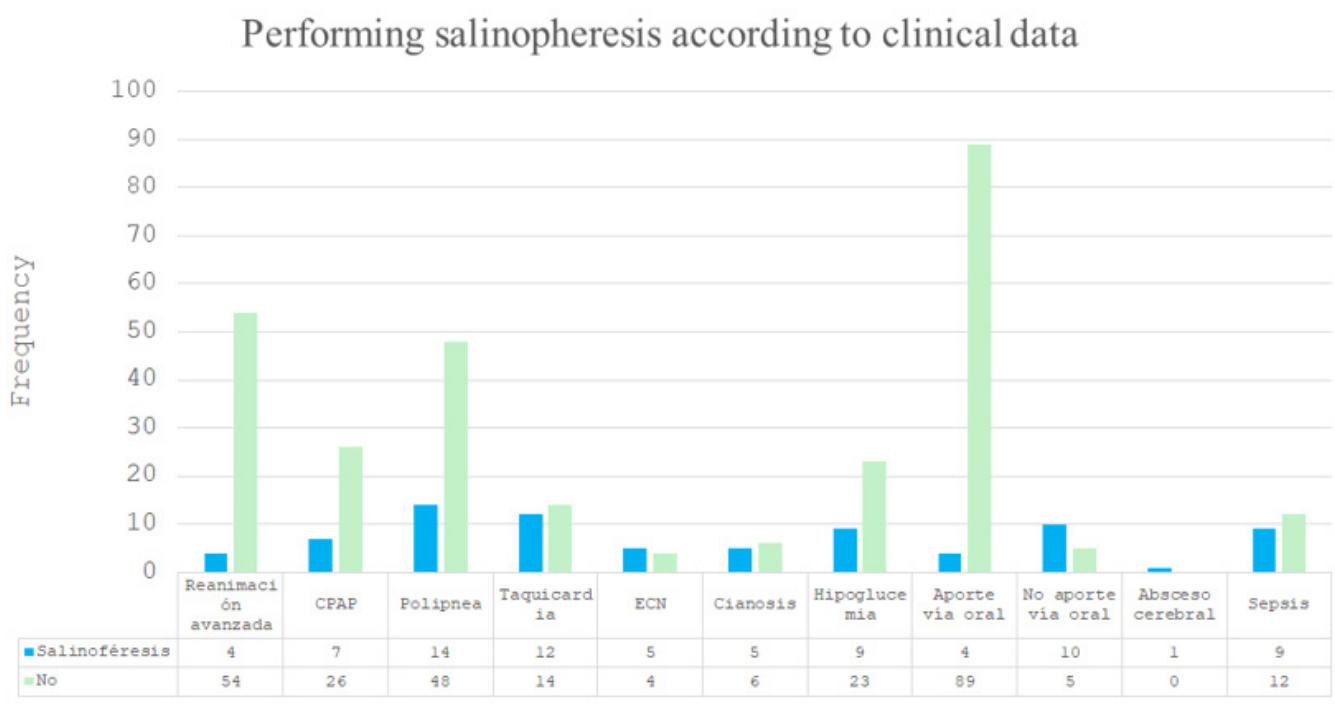

Figure 1 Performing salinopheresis according to clinical data.

Cyanosis, intravenous fluids, and salinopheresis had a statistically significant association with the initial hematocrit.

Redness, polypnea, tachycardia, NEC, hypoglycemia, prematurity, Intravenous fluids and salinopheresis had a statistically significant association with erythroblasts.

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Discussion

In this study, a higher prevalence of male infants was observed, contrary to what has been reported in the literature, which indicates no difference between genders. Cesarean section was more prevalent than vaginal delivery; this can be attributed to the large number of premature infants, twins, and newborns whose mothers had severe preeclampsia, uncontrolled maternal diabetes, obesity, or other maternal comorbidities, given that this is a tertiary care institution. While these are modifiable factors, most of these mothers did not attend our institution for adequate prenatal care during the first trimester.

Most of them debuted with preeclampsia with severe symptoms, according to the literature it is a risk factor associated with morbidity and to develop neonatal polycythemia due to the low hypoxic intrauterine environment to which the baby is subjected.

Although most newborns with neonatal polycythemia were preterm or term, the number of late preterm newborns with neonatal polycythemia is significant, indicating that neonatal polycythemia is not uncommon among late preterm newborns.

The increase in intrauterine erythropoiesis generally results from placental insufficiency and chronic intrauterine hypoxia. This finding was observed during the histopathological study of the placentas, in which chorangiosis stands out. Chorangiosis is an infrequent placental alteration characterized by an abnormal growth pattern of the terminal chorionic villi and has been observed in certain gestational pathologies such as diabetes or preeclampsia, and in situations where there is a certain state of sustained placental hypoperfusion, and where oxygen transfer to the fetus is reduced. This could explain part of the pathophysiology of neonatal polycythemia in these patients, since chorangiosis was the most frequently determined histopathological result in these patients, followed by vascular infarcts and trophoblast hypoxia.

It is vitally important to determine which population is exposed in newborns, and what the symptoms are, since most of them are nonspecific. The most frequently observed symptom was flushing, which should make us suspect that the patient may be experiencing polycythemia and request complete laboratory studies to determine hematocrit, glycemia, calcium and bilirubin, since most patients experienced hyperbilirubinemia requiring phototherapy, and a significant percentage experienced hypoglycemia.

Gestational age was statistically significantly associated with the performance of saline apheresis, and the performance of advanced resuscitation was also statistically significantly associated with saline apheresis. Therefore, patients who have had a complicated course since birth should remain under close observation for biochemical testing and maintain adequate fluid intake to prevent dehydration.

Conclusion

It is vitally important to know the maternal history, and to identify if there is a hypoxic intrauterine environment as one of the risk factors for polycythemia, or any of the maternal morbidities already described in order to identify our risk population and determine if they are candidates for laboratory tests and close monitoring to make early diagnosis and timely treatment to prevent complications from hyperviscosity and/or polycythemia, since most of the risk factors are modifiable.

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Conflict of interest

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