MINI-REVIEW

Neonatal polycythaemia

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ABSTRACT

Neonatal polycythaemia has multifactorial causes, and can be designated as active (increased foetal erythropoiesis) or passive (red blood cell transfusion) polycythaemia. Hematocrit estimated from capillary blood (regularly obtained through "heel sticks" in newborns) is normally the principal laboratory feature facility by which polycythaemia is recognszed. An unusually high proportion of haematocrit builds the risk of hyperviscosity, microcirculatory hypoperfusion, and in the long run multisystem organ dysfunction. A report enclosed in this short communication gives a brief review of neonatal polycythaemia, its causes, management and complications.

KEYWORDS

Neonatal polycythaemia; Hematocrit; Complications; Partial exchange transfusion.

Polycythaemia (erythrocythemia) is an unusual selective height of coursing erythrocyte mass rather than leukocyte and platelet. High haemoglobin levels (≥ 22 g/l) and haematocrit ratios (≥65%) are denominating polycythaemia in newborns [1]. Neonatal polycythaemia usually represents the normal foetal adaptation to hypoxemia instead of genuine haemopoietic stem cell abnormalities. The rate of polycythaemia in healthy term neonates has been accounted to be 0.4% to 5% [2,3]. Venous haematocrit level is utilised as a surrogate marker for viscosity. Haematocrit estimated from capillary blood (regularly obtained through "heel sticks" in newborns) is normally the principal laboratory feature facility by which polycythaemia is recognised. Venous haematocrit determination has been observed to be frequently discordant with capillary values, consistently exceeding venous levels by as much as 10%. Therefore, much of the time, a high capillary haematocrit result ought to be affirmed with a venous haematocrit estimation before choices with regards to clinical management is made for newborn infants [4]. The unusually high proportion of haematocrit builds the risk of hyperviscosity, microcirculatory hypoperfusion, and in the long run multisystem organ dysfunction.

Neonatal polycythaemia may happen in newborn children conceived post-term or small for gestational age, babies of hypertensive or diabetic mothers, twin–twin transfusion syndrome (the recipient infant), and those with chromosomal abnormalities [5]. For the most part, polycythaemia has multifactorial reasons, and therefore, can be designated as active (increased foetal erythropoiesis) or passive (red blood

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cell transfusion) polycythaemia [2]. Increased foetal erythropoiesis (active polycythaemia) is associated with many conditions, such as placental insufficiency, endocrine abnormalities, and genetic disorders. Erythrocyte transfusion (passive polycythaemia) may also be associated with conditions, such as placental-foetal transfusion with delayed cord clamping (DCC) and twin–twin transfusion syndrome [2]. DCC leads to an expanded blood volume being conveyed to the newborn child. When cord clamping is postponed over 3 minutes after birth, blood volume rises 30%. Notwithstanding, potential entanglements of DCC could lead to polycythaemia and hyperbilirubinemia. A few investigations have analysed the frequency of polycythaemia as a potential complication when DCC is rehearsed. An investigation of 242 neonates whose cords were clamped at less than 60 seconds, between 1 minute and just under 2 minutes, or somewhere in the range of 2–3 minutes following birth found that their haematocrit levels at 48 hours after birth were 53%, 58% and 59%, respectively [3]. A later investigation of 73 newborn children demonstrated that DCC at 5 minutes after birth didn't prompt an expanded occurrence of polycythaemia when compared to early cord clamping [5].

Polycythaemia has a wide scope of complications, including numerous organ frameworks, and 50% of newborns with polycythaemia develop one or more symptom. Moreover, most of these symptoms are non-specific and may be attributed to the underlying conditions. Yet, any newborn child with a suggestive component of polycythaemia ought to be screened for polycythaemia [6].

The management of neonatal asymptomatic polycythaemia is disputable; this is because of the absence of proof showing that forceful treatment upgrades long haul results. Before a conclusion of polycythaemia is made, it is obligatory to rule out dehydration and somewhat hypoglycemia [2]. Two patterns of treatment have been described for asymptomatic and symptomatic polycythaemia; conservative management with rehydration and partial exchange transfusion (PET). Asymptomatic infants with haematocrit 60%–70% need only conservative treatment by increasing fluid intake (normal saline), which is frequently administered to neonatal polycythaemia with the aim of preventing the haematocrit from expanding to the levels that require treatment with PET. Recheck haematocrit every 4–6 hours and proceed for this checking for at least 24 hours until the haematocrit level is decreased. However, this exertion is not usually prosperous. A study involving 55 asymptomatic infants with haematocrit values between 65% and 75% demonstrated that treating them with normal saline boluses reduced neither their consequent haematocrit levels nor their requirement for a PET [7]. In asymptomatic patients with a haematocrit level of over 75% on repeated estimations, consider administering PET in spite of the fact that the proof is missing as to its adequacy. In symptomatic patients with a haematocrit level over 65% with side effects owing to polycythaemia and hyperviscosity, consider PET to cure the organ dysfunction [8]. Symptomatic infants should be treated with PET with normal saline if the peripheral venous hematocrit is > 70%, using the following formula:

$$\frac{\text{Infant blood volume}\left(80\ \text{ml/kg}\right) \times \text{measured hematocrit} - \text{desired haematocrit}}{\text{Observed haematocrit}}$$

Although PET is generally regarded to be safe fiats, it is not devoid of risks. Complications have been reported to vary between 0.5 and 3.3%. Many of these complications are transient, such as bradycardia, apnea, severe thrombocytopenia, hypocalcemia and hypokalemia. Recovery is predictable along with appropriate care and monitor. However, critical complications and even death can happen due to cardiovascular collapse, necrotising enterocolitis, bacterial sepsis and pulmonary haemorrhage that can be avoided by careful oxygen saturation and cardiopulmonary monitoring. Necrotising enterocolitis (NEC) is uncommon, however, it tends to mar polycythaemia or hyperviscosity. Truly, about 44% of term newborn children with NEC have polycythaemia. Later information proposed that polycythaemia might not have a huge impact in the advancement of NEC in the term baby, and might be identified with PET with colloid to decrease the haematocrit [9]. Thus, hypoglycemia is the most widely recognised metabolic confusion and is seen in 12%–40% of babies with polycythaemia [10]. Polycythaemia can influence coagulation although disseminated intravascular coagulation is

uncommon. Thrombocytopenia might be noted. In a review study from Netherlands, thrombocytopenia happened in 51% and extreme thrombocytopenia influenced 91% of 140 neonates with polycythaemia [10]. Ultimately, polycythaemia expands the blood thickness, which impedes microcirculatory stream and leads to neurologic, gastrointestinal, cardiopulmonary, renal, thrombotic and metabolic manifestations [5].

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