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Case Report

MALIGNANT PERSISTENT PULMONARY HYPERTENSION – A CASE REPORT

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ABSTRACT

Persistent pulmonary hypertension is a phenomenon with 2 in 1,000 live births. Persistent pulmonary hypertension involves defective lung parenchymal development, heart valvular defects, or may also be syndromic. It can also be idiopathic. The most common factor is improper clamping of the umbilical cord on parturition. This case report discusses the condition of a male neonate with severe tachypnea and hypoxia due to PPTH at birth. A male neonate born to anon consanguineous parents on the day of birth presented with severe tachypnea. It was normal vaginal delivery conducted in the ambulance. There was a delay in clamping the umbilical cord, it was clamped with an elastic rope. The neonate and the mother were rushed to the obstetric ward within the time duration of 20 min. The neonate presented with hypoxia, hypocalcemia, and severe tachypnea. The neonate was shifted to the intensive care unit and was supported with high flow oxygen 2 L/min. 2D echo was performed; there was no significant anomaly noted except tricuspid valvular regurgitation. The pulmonary pressure was recorded to be 76 mm/hg which gradually reduced to 26 mm/hg on the 6th day after birth. Improper or impaired fall in pulmonary vasculature resistance that occurs after birth and increase of systemic vascular resistance is due to the removal of the placenta from circulation. Increased pulmonary vasculature with decreased or reversal of shut at the foramen ovale or ductus arteriosus leads to PPHTN. Proper clamping of the umbilical cord within 60 s becomes crucial. Delayed or improper clamping can lead to severe PPHTN.

Keywords: Pulmonary hypertension, Umbilical cord clamping, Hypoxia.

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INTRODUCTION

Pulmonary hypertension is a well-known condition of newborn which occurs due to various reasons such as defective development of lung parenchyma, heat development, involvement of chromosome 7, or iatrogenic reasons also. Persistent pulmonary hypertension is related to elevated vascular resistance, resulting in vascular shunting of blood hypoxemia. The treatment modalities include optimal oxygenation, avoiding respiratory and metabolic acidosis, blood pressure stabilization, sedation, and pulmonary vasodilator therapy. Circulatory transition during birth is crucial to maintain gaseous exchange from the placenta to the lung tissue. About 8–10% of combined ventricular output and 13–21% in human fetuses perfuse the fetal lungs due to high pulmonary vascular resistance [1]. Clamping of the umbilical cord removes the low-resistance placental circulation, increasing systemic arterial pressure. Simultaneously fast reducing pulmonary resistance and increase pulmonary blood flow promote pulmonary vasodilation ventilates the lung with sufficient oxygenation [2]. It was observed that delayed cord clamping with a decrease in the blood Ph levels altered acid-base parameters and lactate values compared to immediate cord clamping [3]. Delayed cord clamping has been useful in preterm births, while it increases the pulmonary vascular resistance leading to persistent pulmonary hypertension [4]. This case report explains the delayed cord clamping of 15–17 min which resulted in the slow decline of pulmonary vascular resistance, leading to respiratory distress and hypoxia.

CASE REPORT

The case study has been presented as a detailed report after getting informed patient consent. A 30-year-old mother with Gestational Diabetes mellitus delivered a body in the ambulance on the way to the hospital. It was a normal vaginal delivery. Neonate cried after birth but the clamping of the umbilical cord was delayed. APGAR score was not available. Downes score was 3/10. The baby was severely severe tachypnea. The umbilical cord was clamped with an elastic rope and cut after reaching the hospital with a time interval of 15–17 min.

Observation

The baby presented with severe tachypnea, hypoxia, and reduced blood Ph. Inj Vitamin K 1 mg was given intramuscularly. There was no significant congenital anomaly noted, baby was on continuous positive airway pressure (CPAP) with Fi O_2 21%, CPAP-5, later 2L/min was given. Blood culture was done using an aerobic bacterial method, no growth was observed after 48 h of incubation. C-reactive protein was also tested negative. The baby was investigated with 2D echo and other blood parameters were evaluated for abnormalities. There was elevated total bilirubin 4.4 mg/dl, while direct bilirubin was normal. Hypocalcemia was noted 7.5 mg/dl. Blood gas analysis revealed reduced Ph 7.314 (slightly acidic) evident hypoxia was noted 42.5 mm/hg. No hypercapnia was noted. Blood electrolyte analysis is done. Hyperkalemia 5.5 mmol/hypernatremia 123 mmol/L, and hypocalcemia 0.88 mmol/L (ionized calcium) increased chloride levels. Metabolite levels showed an increase. Increased serum lactose 3.6 mmol/L and decreased glucose level 66 mg/dl ABG analysis revealed reduced O_2 saturation 81.2% partial O_2 which was 104.6 mm/Hg, and P O_2 (a/A) e 40.6% Ph(st)e 7.322. C-reactive protein was noted reactive. Complete blood count was normal. There were no hemi parasites seen. 2D Echo clearly showed tricuspid regurgitation with pulmonary pressure up to 73 mm/Hg. The baby was administered 2 L/min O_2 repeat 2D echo after 6 days revealed pulmonary pressure to be 26 mm/Hg. The serum T4 and thyroid stimulating hormone was normal in repeated evaluation on the 4th and 6th day after birth. There was a reduced ionized calcium level of 0.9 mmol/L. The baby was discharged after giving O_2 therapy and reviewed for 3 years. There were no significant breathing difficulties or breathlessness noted. The milestones are normal with the normal dietary pattern (Fig. 1).

DISCUSSION

Ventilation of lungs at birth reduces pulmonary vascular resistance, timely removal of the low vascular resistant placenta from the systemic circulation at birth increases systemic vascular resistance [5]. The sequence of events occurring at normal vaginal delivery is related to reduced fetal pulmonary vascular resistance, while in elective cesarean

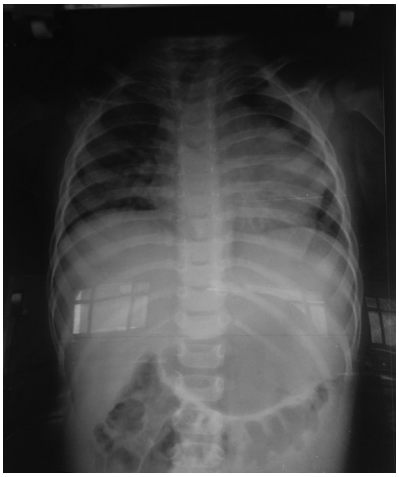

Fig. 1: X ray of the neonate with persistent pulmonary hypertension

section shows reduced pulmonary vascular resistance as in delayed cord clamping [6].

During normal vaginal delivery, there is a sequence of hormonal changes occurring which prepares the fetus to change into a neonate. One of the main changes include the absorption of lung alveolar fluid that prepares it for gaseous transition [7]. Reabsorption of sodium through the amiloride-sensitive sodium channels in the alveolar epithelial cells. Mechanical factors are also involved in alveolar fluid reabsorption. This rapid reabsorption of alveolar fluid is replaced by air after birth facilitating gaseous exchange. Elective cesarean section has reduced the risk of respiratory distress secondary to transient tachypnea of the newborn, surfactant deficiency, and pulmonary hypertension morbidity. In normal vaginal delivery, the hormonal sequence of events occurring at parturition leads to timely clamping of the umbilical cord after cessation of umbilical cord pulse. There is a rapid decrease in the ratio of the pulmonary artery and aorta pressure. This ratio is altered with delay in the cord clamping resulting in increased pulmonary vascular resistance left ventricular blood flow increases defective right to left shunt through foramen ovale. This causes severe hypoxia, malignant transient tachypnea in the neonate. The dynamic nature of the pulmonary circulation and the oxygen across the placenta maintains fetal oxygen saturation.

Maternal arterial oxygen saturation with 100% oxygen saturation transfers 25–28 mm/Hg partial pressure in the fetal left ventricular and ascending aorta blood perfusing the fetal brain. Higher maternal oxygen administration with partial pressure above 400 mm hg also increases the venous pressure to 40–50 mm/hg in the ascending aorta of the fetus [5]. This clearly shows that maternal hyperoxemia does not affect the fetal arterial oxygen partial pressure very much.

Pulmonary vascular resistance is known to change throughout the fetal life with varying circulating blood levels in the lungs. During the full-term the vasoconstriction drops the amount of combined ventricular output with 19–23% pulmonary vasculature, as weeks progress during the delivery the pulmonary blood vessels develop sensitivity to oxygen, leading to fetal hypoxic pulmonary vasoconstriction and elevation of pulmonary vascular resistance [6,8]. This case report shows that normal vaginal delivery in the ambulance without proper expertise led to the delayed clamping of the umbilical cord. The neonate was born without any congenital anomaly to a non-consanguineous parent. The NVD was conducted in the ambulance without proper instrumentation for umbilical cord clamping. The umbilical cord was clamped after 15–17 min after delivery after reaching the hospital. The neonate developed hypoxia, malignant tachypnea. There was a reduced oxygen saturation of 81.2% partial O_2 [9].

The neonate presented with reduced oxygen saturation and reduced blood Ph. The carbon dioxide partial pressure was maintained within normal limits. There was severe persistent pulmonary hypertension that was reduced with the administration of 2L oxygen at the neonatal intensive care unit. There were electrolyte level variations showing hyponatremia, hypocalcemia, and respiratory acidosis.

CONCLUSION

Hospital delivery is an important factor for safe parturition. Developing countries like India still reports normal vaginal delivery conducted by untrained persons without proper facilities. This case report is an eye-opener for the necessity to prevent delayed cord clamping to avoid persistent pulmonary hypertension.

AUTHORS' CONTRIBUTIONS

All authors equally contribute to the article.

CONFLICTS OF INTEREST

None.

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PATIENT CONSENT

Obtained.

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