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**Case Report**

# Atypical cause of apnoea in a neonate born at 29 weeks

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## ABSTRACT

A 29 weeks gestation, male, weighing 1270 grams was born through a vaginal delivery. He was intubated at birth and received 2 doses of surfactant. He had multiple failed extubation attempts on 1st, 7th, 28th, and 54th day of life on account of recurrent apneic spells. The apneic spells continued well beyond 36-37 weeks post menstrual age. He was noted to have subtle signs like poor suck-swallow, and gag reflex for which a pediatric neurology consult was sought. Detailed assessment revealed signs of bulbar weakness, and facial weakness. Magnetic resonance imaging of the brain showed an exophytic space occupying lesion in the medulla oblongata. Liberal excision biopsy was performed; confirming a definitive diagnosis of neuroepithelial tumor, which was difficult to classify, but had features favoring primitive/embroyonal tumor with focal glial differentiation, which carries a guarded prognosis. The child is presently 2 years old and receiving palliative home care. Apnea/s are frequent events in neonates. They can be attributed to a multitude of causes involving all major systems like cardiac, respiratory, neurological, gastro-intestinal, systemic causes like electrolyte imbalance, sepsis, prematurity etc. Apnea of prematurity is one of the common causes. Physicians should be mindful of the usual pathophysiological course of the apnea of prematurity, and its persistence beyond the usual age, especially in the presence of subtle clinical and radiological signs, should warrant further workup.

**Keywords:** Neonates, Preterm, Apnea, Brain tumor

## INTRODUCTION

Apnea in neonates, is a common symptom. It is observed in 15-25% of all the neonates admitted to the neonatal intensive care unit (NICU).[1] The incidence of apnea depends on gestational age, weight and underlying pathology, etc.[1,2] Of all the causes, apnea of prematurity is one of the most common. The incidence of apnea of prematurity is inversely related to gestational age (GA) of the neonate. Almost all the neonates born before 28 weeks of GA, up to 85% of those born at 30 weeks of GA and around 20% of those born at 34 weeks, are affected by apnea of prematurity.[3] Most of the prematurity-related apneic episodes resolve by 34-35 weeks post-menstrual age (PMA); the exception being presence of chronic lung disease or in neonates born at < 28 weeks, wherein they may last till 43-44 weeks PMA.[4] The authors, report a case of premature neonate, who continued to have "Apneic spells" beyond the expected PMA. An informed written consent was obtained from the child's parents.

## CASE REPORT

A male neonate, weighing 1270 grams (3rd Centile), was born through a vaginal delivery at 29 weeks GA, in our hospital. His mother was a primigravida lady of South-East Asian descent, with no significant family history of note in a non-consanguineous marriage. The mother had received antenatal care in another hospital; however, those records were not available to us. She presented in established labor with ruptured membranes, and received two doses of steroids, and prophylactic antibiotics prior to delivery. The child was born with Apgar scores of 3, 6 and 8 at 1, 5, and 10 minutes of life, respectively. He was

intubated at birth and received a dose of surfactant at 30 minutes of life. Empiric antibiotics and caffeine citrate were started soon after admission.

He had a failed extubation at 12 hours of life, followed by increasing oxygen requirement, for which he received a second dose of surfactant at 14 hours of life, with good response. He was extubated to non-invasive ventilation with pressure control (NIV-PC) on 4th day of life (DOL). Antibiotics were discontinued after negative blood culture at 48 hours. Trophic feeds were initiated on first DOL. He was re-intubated, due to repeated apnea episodes associated with desaturation and bradycardia, on 7th DOL.

However, he remained hemodynamically stable. Second line antibiotics were administered empirically for 48 hours, and discontinued after documenting serial normal inflammatory markers, and negative blood culture. He reached full enteral feeds by 10th DOL. He was extubated, a third time to NIV-PC on 14th DOL. He couldn't tolerate continuous positive airway pressure (CPAP) or high flow nasal cannula (HFNC) and remained on NIV-PC.

The child deteriorated again on 28th DOL with recurrent apneas and was re-intubated. These apneas occurred in clusters, were associated with desaturation and bradycardia, not responding to stimulation and needing positive pressure ventilation. There was no correlation between feeding volume, and/or timing with the episodes. He was again started on empiric antibiotics pending blood culture.

As before, the blood workup for sepsis was inconclusive. The chest X-ray findings were not significant, and an echocardiography was normal. A cranial ultrasound scan, done on 3$^{rd}$ DOL showed mildly dilated lateral ventricles (R-13.4 mm and L-14.3 mm) as well as 3rd ventricle but no intracranial bleed.

The repeat cranial ultrasound scan at 28th DOL, showed very similar findings of dilated lateral ventricles but normal 3$^{rd}$ ventricle. He was extubated to NIV-PC on 31st DOL. He continued being on NIV-PC, with failure to tolerate CPAP and HFNC because of recurrent apnea episodes, some requiring positive pressure ventilation.

The characteristics of the apnea episodes were like before. The child was noted to have a somewhat weak suck-swallow reflex, which was attributed to prematurity. Serial head circumference measurements were not significant. A third cranial ultrasound scan was performed and was same as the second one. He continued being on maintenance dose of caffeine.

He deteriorated again and was re-intubated on 54th DOL (PMA-36 weeks +6 days), for recurrent apneic episodes. The blood workup was inconclusive, and he was managed on same line of management, as before. Chest X-ray was not suggestive of bronchopulmonary dysplasia (BPD). The child was also noticed to have somewhat weak gag reflex with some doubtful signs of upper motor neuron facial palsy. Because of the doubtful neurological signs with a marginally abnormal cranial ultrasound, a pediatric neurology consultation was sought.

Neurology assessment revealed head circumference growing along the centile with no change in trajectory. He had subtle facial dysmorphology (long philtrum, retrognathia, very high arched palate) and signs of bulbar weakness (paucity of tongue and palatal movements, absent gag reflex, and occasional drooling of saliva).

He had facial weakness with preserved eye closure suggesting a central cause. There was no response to loud sounds. He had axial hypotonia with normal peripheral muscle tone and symmetrical limb movements.

Neuroimaging was recommended to identify or exclude a structural cause for the bulbar dysfunction before considering any other investigations. A brain magnetic resonance (MR) scan was performed on 60th DOL (PMA, 37 weeks + 5 days).

It showed an exophytic space occupying lesion, in the medulla oblongata slightly towards the left side. It had relatively increased signal in T1W1, low signal in T2W1, but iso-intense to brain cortex in both T1 and T2 WI. There was faint diffusion restriction on DWI. This lesion caused mass effect on the surrounding structures with no surrounding edema (Figure 1). A differential diagnosis of exophytic glioma of medulla oblongata or primitive neuroectodermal tumor (PNET) was considered and the baby was transferred to a sister tertiary level hospital on 68$^{th}$ DOL for further management.

***Management and outcome***

A liberal excision biopsy was performed at the referral center. Staining and molecular studies established a definitive diagnosis of a neuroepithelial tumor, which was difficult to classify, but features, favoring primitive/embroyonal tumor with focal glial differentiation.

A multi-disciplinary team–including pediatric oncologist and neurosurgeon decided on palliative line of management considering the poor prognosis and high risk of the further surgery, chemotherapy or radiotherapy. Parallelly, the child failed 2 more extubation attempts at/on 75$^{th}$ and 80$^{th}$ DOL and was tracheostomized on 85th DOL and transferred back to us on 90th DOL for continuation of palliative care.

Presently, the child is two years old, tracheostomized, with gastrostomy tube in-situ, and is receiving palliative care at home. Serial MR scans demonstrated a slow growing tumor, which is unresectable and the child continues to receive palliative care.

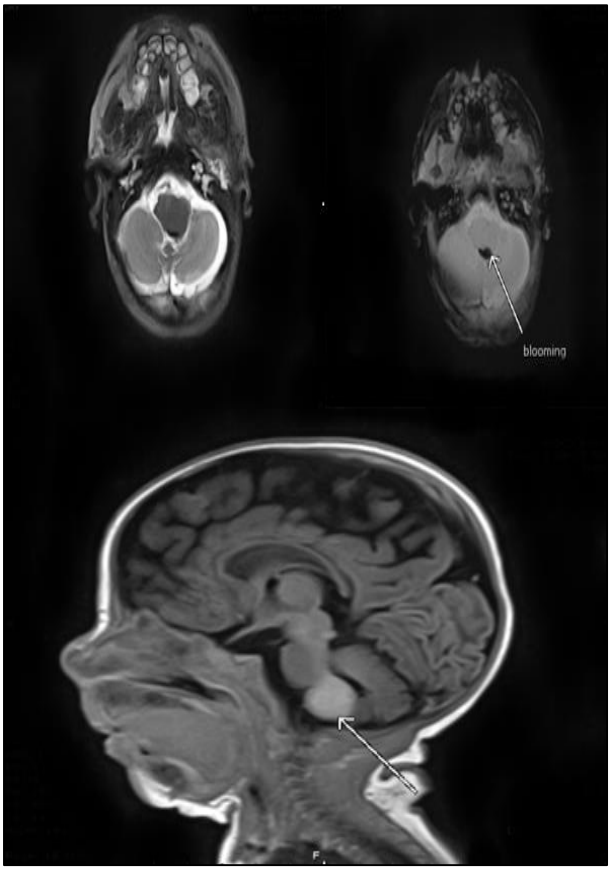


**Figure 1: Magnetic resonance images showing an exophytic space occupying lesion in the medulla oblongata slightly towards the left side**

## DISCUSSION

Apnea is classically defined as "cessation of respiratory effort or airflow for >20 seconds or for shorter duration when accompanied by bradycardia or hypoxemia".[3] The common underlying causes for apnea in neonates include. Neurological–birth trauma, drugs, intracranial hemorrhage, seizures, perinatal asphyxia, pulmonary–respiratory distress syndrome, pneumonia, pulmonary hemorrhage, obstructive airway issues, air leak syndromes; cardiac–congenital cyanotic heart disease, patent ductus arteriosus, congestive heart failure, shock; gastro-intestinal–gastroesophageal reflux, necrotizing enterocolitis. systemic conditions–hypo/hyperthermia, anemia, sepsis, metabolic acidosis, hypoglycemia, hypocalcemia, hypo/hypernatremia, inborn errors of metabolism, prematurity.[2,5,6]

Apnea of prematurity is one of the commonest reasons for apnea in neonates. The underlying cause is attributed to the immaturity of the nervous system. It is conventionally a diagnosis of exclusion, to be labelled only after the underlying secondary causes are excluded. The prevalence of apnea of prematurity increases with decreasing GA, varying from 10-20% in neonates born at 34 weeks, to 60-85% in neonates born at 28 weeks, and almost 100% in those born <28 weeks GA.[1] Apneas in neonates, can be categorized in 3 types. Central cessation of respiratory effort with no obstruction. Obstructive the child has persistent breathing efforts, without airflow, due to obstructed airway, mixed–mostly starts as obstructive apnea followed by central apneas. They account for ~40%, 10% and 50% of the apnea episodes, respectively.[5] The apneic spells of prematurity usually stop by 37 weeks in 92%, and by 40 weeks in 98% of neonates.

However, the apneic spells can persist well beyond 38 weeks in extremely preterm neonates born at 24-26 weeks GA, and in neonates with underlying chronic lung disease.[4] Even in this subgroup, there is a dramatic decline in the severe spells after 43 weeks, though they may continue showing clinically silent intermittent hypoxia.[4]

In our case, the persistence of recurrent apneas and extubation failures, beyond the physiological course, presence of subtle clinical signs weak suck-swallow, gag reflex, presence of signs of facial nerve paresis, and subtle cranial ultrasound findings, prompted us to evaluate this child with MR scan, which clinched the diagnosis.

The respiratory control network extends from the caudal medulla and roof of 4th ventricle, to the dorsolateral pons, and lastly to nucleus of the solitary tract. Respiratory control is mediated by the feedback from central (located within the respiratory control network) and peripheral chemoreceptors (located mainly in carotid bodies).[7]

The nuclei of the 5th, 7th, 9th cranial nerves also lie in the medulla and the pons.[7] The authors determine retrospectively, that the recurrent apneas and desaturations were probably of mixed nature, on account of the proximity of the brain tumor to the critical centers – respiratory control network and nuclei of cranial nerves involved in airway protection.

Brain tumors, which present in the neonatal age group are rare and represent around 0.5% to 2% of all pediatric brain tumors.[8] Most of the brain tumors in neonates are incidentally diagnosed in the antenatal period by ultrasound. With the advances of fetal medicine fewer and fewer children are diagnosed postnatally.[8] Those diagnosed postnatally, present with increased intracranial pressure, hydrocephalus, seizures, irritability, drowsiness, apneic spells, vomiting and neurological deficits.[9]

The common brain tumors presenting in neonatal period are germ cell tumors (teratomas), choroid plexus tumors (papilloma, carcinoma), astrocytic tumors and embryonal tumors (embryonal tumors with multilayered rosettes (primitive neuroectodermal tumors (PNETs), atypical teratoid/rhabdoid, medulloblastoma], neuronal and mixed neuronal-glial tumors.[10] The commonest outcome of neonatal brain tumors is death within 5 years of diagnosis.[10]

## CONCLUSION

Physicians should be mindful of the usual pathophysiological course of the apnea of prematurity, and its persistence beyond the usual age, especially in the presence of subtle clinical and radiological signs, should warrant further workup.

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