Journal of the African Society for Paediatric Infectious Diseases
Volume 2

CASE REPORT

Does a feeding nasogastric tube cause hypoxaemia?

Akinrinoye OO*[1], Subair YO[1], Falade AG[1,2]
[1]Department of Paediatrics, University College Hospital, Ibadan, Nigeria
[2]Department of Paediatrics, College of Medicine, University of Ibadan, Ibadan, Nigeria
*Corresponding author: akinrinoye@gmail.com

How to Cite this article:
Akinrinoye OO, Subair YO, Falade AG. Does a feeding nasogastric tube cause hypoxaemia? Journal of the African Society for Paediatric Infectious Diseases. 2023;2:1-6. DOI: https://doi.org/10.15641/jafspidVol2pp1-6/1669

Article Information

Received: 30 April 2023
Accepted: 31 July 2023

Key words
Feeding nasogastric tube, hypoxaemia

Abstract

The use of feeding nasogastric tube has been very beneficial in management of sick infants to ensure optimal intake but is not without untoward effects. Persistent hypoxaemia is not well known to be associated with nasogastric tube feeding. We report two cases of persistent hypoxaemia in tube-fed patients with resolution on removal. Clinicians need to have a high index of suspicion for possible association between nasogastric tube feeding and persistent hypoxaemia after excluding common causes of hypoxaemia. Regular pulse oximetry and prompt removal of the tube is recommended once patient can take considerable quantity of feeds and fluid orally.

Introduction

Poor feeding is a common manifestation of severe illness, and this includes infants and children with severe community-acquired pneumonia (CAP), who are vomiting everything or refusing feeds.[1] An age long alternative is the use of nasogastric tube (NGT) or orogastric feeding until they are able to take adequately, as also used in early phase of nutritional rehabilitation.[2] This method of feeding is associated

with a few complications, which include vomiting, gagging, aspiration pneumonitis, and hypoxaemia, especially during insertion.[1, 3] But it is not clear if it causes persistent hypoxaemia. There have been contradictory reports on the possible association between use of NGT and hypoxaemia among neonates, with scanty mention among infants and children.[3-5] We observed persistent hypoxaemia and oxygen dependence in two patients with successfully treated severe CAP while on NGT feeding, and resolution of the hypoxaemia on removal of the tube.

Case presentation 1

OA, a 14-month-old male toddler presented to our facility on account of a fever of 4 days' duration, cough of 2 days and difficulty with breathing of one day. Fever was high grade, intermittent, associated with reduced appetite and refusal of feeds except breast milk. Cough was paroxysmal but non-barking. There was neither post-tussive vomiting, nor nasal discharge.

He subsequently developed difficulty with breathing. He had a positive history of cough in his older sibling which started earlier. No history suggestive of aspiration of a foreign body, choking on feeds or bluish discolouration of the lips or extremities.
At the onset of the illness, the mother gave paracetamol syrup; and oral dispersible artemether /lumefantrine. When the symptoms persisted, he was taken to a primary healthcare centre (PHC), where he was given oral cefuroxime 5 ml twice a day; and ibuprofen 5mls three times a day. However, on persistence of difficulty in breathing, he was taken to Adeoyo Maternity Teaching Hospital, Yemetu, Ibadan (AMTHYI) from where he was referred to the University College Hospital (UCH), Ibadan Nigeria due to a lack of bed space.

The pregnancy, labour, and delivery history was unremarkable. He was exclusively breastfed for 4 months. The mother commenced pap (porridge) and water at 4 months, other family diet at 6 months of age and he is currently on family diet and breastfeeding.

He was fully immunized according to the National Programme on Immunization schedule. The developmental history was appropriate for age.

On examination, he was febrile (axillary temperature, 38.1˚C), anicteric, not pale, acyanosed, well hydrated and had no pedal oedema. The tonsils were slightly enlarged and hyperaemic.
His weight was 9.7kg (0.1 z-score), length was 84cm (2.42 z-score.), mid-upper arm circumference was 14cm (-0.28 z-score.), and occipito-frontal circumference was 47cm (1 z-score).

There was no stridor, wheezing, or grunting. His chest was symmetrical. He was mildly dyspnoiec, tachypnoeic (respiratory rate, 54 breaths per minute), and has vesicular breath sounds both lung fields. The transcutaneous haemoglobin oxygen saturation (SpO_2) was 89% on room air. His pulse was 140 beats per minute, regular and full volume. The first and second heart sounds were heard and there was no murmur.

The abdomen was full, moved with respiration, soft, and with no area of tenderness. The liver was palpable 4 cm below the right costal margin, firm, smooth and non-tender. No other abnormality was observed.

The diagnosis was acute severe CAP not in heart failure, and acute tonsillopharyngitis. He was placed on supplemental oxygen 1l/min, and intravenous antibiotics (cefuroxime 50mg/kg/dose 8 hourly and gentamycin 5mg/kg/day).

The chest radiograph showed situs solitus, levocardia, right perihilar adenopathy, patchy opacities on both lung fields, the cardiophrenic and costophrenic angles were clean and intact. The packed cell volume (PCV) was 31%; malaria rapid diagnostic test (RDT) was negative, and the full blood count (FBC) showed leucocytosis (16,000/µL) with neutrophil predominance (neutrophils, 66.1%;

lymphocytes, 26.4%; monocytes, 6.3%); eosinophil, 1.2% and platelets, 280,000/ μL). The blood culture was sterile. The serum chemistry results showed metabolic acidosis of 10 mmol/L, Na-134mmol/L, K-3.7mmol/L, Cl-100mmol/L, Urea- 32mg/dl, and Cr-0.9mg/dl. The Gene X-pert test for *Mycobacterium tuberculosis* complex using gastric lavage was negative.

During the first 15 hours of admission, the patient's SpO_2 was between 95% and 99% on 1l/min of oxygen. An NGT was passed because he was not tolerating feeds orally. Correct placement of the NGT was confirmed by aspirating gastric content (checking that the pH was between 1-5) and hearing a whooshing sound when a stethoscope was placed over the patient's epigastrium while instilling a 30cc air bolus. He was subsequently weaned off oxygen. The SpO_2 on room air was between 95% and 98% for the subsequent 5 hours (26 hours into admission). However, 27 hours into admission, the patient desaturated (SpO_2 88% on room air) shortly after being fed via NGT, and aspiration pneumonitis was suspected. He was recommenced on intranasal oxygen at 1L/min.

Between day 2 and 6 on admission, the patient remained mildly dyspnoeic (mild lower chest wall indrawing), and he was still on oxygen support. The breath sounds were vesicular with few coarse crackles on the right middle and lower lung zones. He was saturating between 95 and 100% on oxygen. On day 7 of admission, the patient was afebrile, breath sounds were normal, respiratory rate ranged between 42 and 46 breaths/min, with no added sounds but still desaturating on room air while the NGT was *in situ*, and thus oxygen was continued at 1l/min. Possibility of impairment of oxygenation due to subtle obstruction from NGT was suspected during the consultant ward round. The NGT was removed, saturation improved significantly and remained normal (between 95 and 99% on room air) till discharge on the following day. The patient was discharged on day 8 with SpO_2 of 96% on room air.

Case presentation 2

AA was a 15-week-old (real age, 5 weeks + 5 days) preterm female infant who presented with cough of two days' duration, fast breathing of 12 hours and 2 episodes of vomiting.

She developed cough 2 days prior to presentation. Cough was neither barking nor paroxysmal. Fast breathing started 12 hrs after onset of cough, no choking on feeds, no bluish discoloration of skin or extremities. She had associated vomiting (2 episodes), which contained recently ingested feeds and not bloody. No passage of loose stool, and no fever. She had had BCG and Hepatitis B vaccine at 8 weeks. Other aspects of history were not remarkable.

At presentation, examination revealed a small-for-age infant, axillary temperature was 36.7^{o}C, mild pallor, anicteric, acyanosis, optimal hydration status and no peripheral oedema. The SpO_2 was 90% on room air.

The anthropometry was within normal limits: weight, 3.1 kg (-2.78 z-score); length, 50 cm (-2.49 z-score); and occipitofrontal circumference, 38 cm (0.66 z-score). There were abnormalities only in the respiratory system. There was neither stridor nor wheezes. She was not grunting, but dyspnoeic with subcostal recession; tachypnoeic with respiratory rate 72 breaths/min, breath sounds were normal, and there were bilateral coarse crackles. Other systems were essentially normal. The diagnosis was preterm female neonate, severe pneumonia not in heart failure. The differential diagnosis was aspiration pneumonitis.

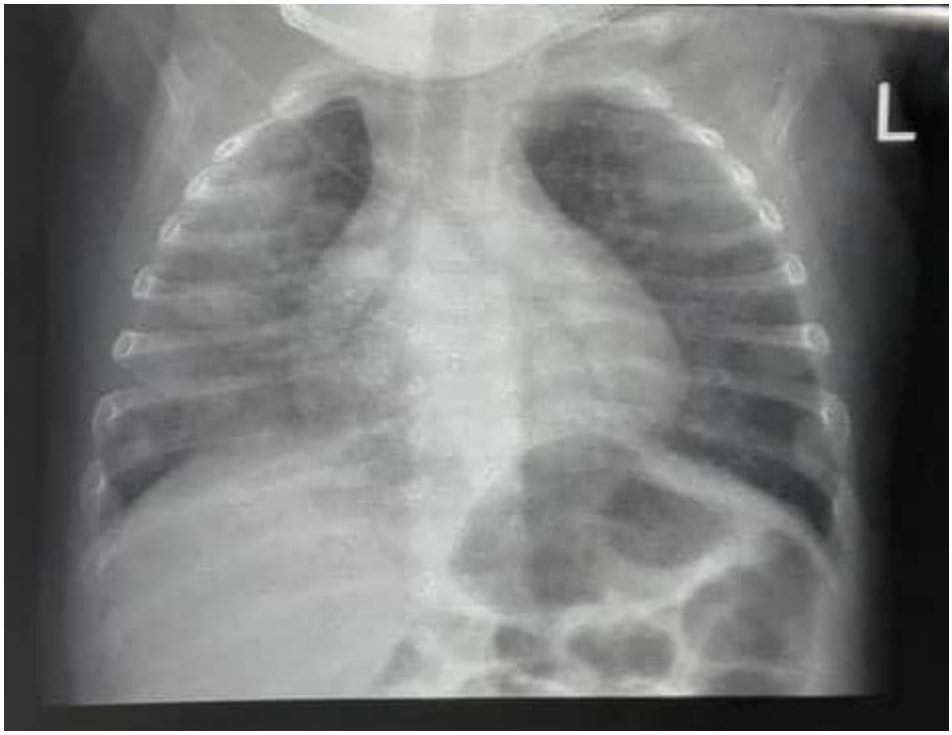
On admission, the random blood glucose was normal (78mg/dl). FBC and differentials showed leucocytosis (14,200/μL) and normal differential counts (neutrophils,17.1%; lymphocytes, 74.4%; monocytes,7.3%, eosinophils, 1.2%), platelets were 430, 000 cells/μL. Pre-transfusion PCV was 23 %, and 32% after about 30mls of blood transfusion the next day. The malaria RDT was negative, and blood film showed no malaria parasites. The chest radiograph revealed areas of widespread patchy opacities and near homogenous opacity involving the right middle lung zone, Figure 1. The electrocardiogram showed left ventricular hypertrophy, but echocardiographywas essentially normal. The serum electrolytes, urea and creatinine showed metabolic acidosis of 15mmol/L while other parameters were essentially normal (Na- 130mmol/L, Cl-104mmol/L, K-4.7mmol/L, Urea-5mg/dl, and Cr-0.3mg/dl).

She was treated thus: oxygen administered via nasal prong at 0.5l/min, which increased the SpO_2 from 90% to 98%; intravenous(iv) cefuroxime 150mg/kg/day 8hourly, iv gentamycin 5mg/kg daily, and iv metronidazole for 1 week.

She had persistent diarrhoea and was managed with tablets zinc sulphate 10 mg daily for 10 days, and oral rehydration solution . Throughout admission, she was euglycaemic. The antibiotics were changed to iv ceftriaxone because of poor clinical improvement, persistent fever, and worsening respiratory distress. The ceftriaxone was also changed after six days of use to iv meropenem and iv clindamycin for eight days; there was a sustained improvement but remained dependent on oxygen.

She was commenced on NGT feeding because of inadequate intake from direct breast-feeding with associated weight loss. She remained hypoxaemic for most of this time and was continued on intranasal oxygen at 1-2 l/min. On the 16th day on admission, temperature had remained normal, dyspnoea subsided, respiratory rate was 52 breaths/min, but she remained hypoxaemic and dependent on oxygen. Possibility of NGT contributing to the hypoxaemia was suspected. The NGT was removed, and hypoxaemia resolved, and improvement maintained till patient was discharged home after spending 18 days on admission.

Figure 1, chest radiograph of case 2 at presentation



Discussion

These two cases suggest the possibility of subtle and undetected episodes of hypoxaemia in infants on NGT feeding. Anatomically, the narrowest portion of the respiratory tract (the anterior nasal valve) is found just posterior to the nares, and since the tube is often passed through the upper respiratory passage, and then into the oesophagus, there is a possibility of it causing impairment of oxygenation and inadequate ventilation especially in such patient with severe pneumonia with significant lung consolidation; but this is expected to resolve following treatment with resolution of the infective

process.[3,6]

Many decades ago, Bevan et al[4] demonstrated significant improvement of about 20% in the forced expiratory volume in one second (FEV_1) following removal of nasogastric tube in eight adult patients after operation, which support the belief that an NGT unfavourably affects pulmonary ventilation in adult patients.[4] This belief has not been proven irrefutably to be so in infants or children. Some researchers[2] have also hypothesised that nasogastric route of placing a feeding tube is associated with higher incidence of hypoxaemia and bradycardia in preterm infants compared to orogastric route, but Bohnhorst et al[5] and Watson et al[2] found no difference in the effect of route of placement on development of hypoxaemia.[2]

However, Shiao et al[3] found some degree of breathing compromise in preterm infants with nasogastric tube feeding compared to those on continuous sucking, and recommended close monitoring of oxygen saturation, and heart rate during feeding via NGT.[3] These cases have been presented is to draw attention to the possibility of this interaction among infants especially those with other co-morbidities like pharyngitis as seen in the first case that can cause partial obstruction to the upper airway.[6] The impact of prematurity on the development of hypoxaemia as seen in the second case reported is not so clear, but warrants further studies as available reports in the systematic review by Watson et al[2] are too few to influence practice. It is still not clear whether this association is by chance or if it only occurs in association with cases of pneumonia with significant lung consolidation, and thus requiring further studies. Nonetheless, clinicians need to have a high index of suspicion for the possibility of NGT feeding causing hypoxaemia, when hypoxaemia is persistent than expected, and also ensure more frequent pulse oximetry on patients with NGT feeding and prompt removal once adequate oral/direct intake is established.

Author contributions: All authors contributed to the writing and finalisation of the manuscript.
Funding Sources: Nil
Ethics approval: Not applicable
Consenting statement: The authors confirm that verbal informed consent was obtained from the patients' biological mothers to publish the clinical information and the image in these case reports.
Competing interests: The authors declare no competing interests.

References

1. Krom H, van Zundert SMC, Otten M-AGM, van der Sluijs Veer L, Benninga MA, Kindermann A. Prevalence and side effects of pediatric home tube feeding. Clin Nutr. 2019;38(1):234-9.
2. Watson J, McGuire W. Nasal versus oral route for placing feeding tubes in preterm or low birthweight infants. The Cochrane Database of Systematic Reviews. 2013;2013(2):Cd003952.
3. Shiao SY, Youngblut JM, Anderson GC, DiFiore JM, Martin RJ. Nasogastric tube placement: effects on breathing and sucking in very-low-birth-weight infants. Nurs Res. 1995;44(2):82-8.
4. Mackay WD, Aberd M, Matheson NA, Aberd M. Nasogastric tube and pulmonary ventilation. Arch Surg. 1963;87(4):673.
5. Bohnhorst B, Cech K, Peter C, Doerdelmann M. Oral versus nasal route for placing feeding tubes: No effect on hypoxemia and bradycardia in infants with apnea of prematurity. Neonatology. 2010;98(2):143-9.
6. Sporik RB. Why block a small hole? The adverse effects of nasogastric tubes. Arch Dis Child. 1994;71:393-4.

The *Journal of the African Society for Paediatric infectious Diseases Society* (JAfSPID) is a free, open-access, online journal. *JAfSPID* publishes a wide variety of manuscripts including full-length research articles, short research communications, review articles, commentaries, case reports, medical images, conference reports, short commentaries on a published landmark paper or report, letters to the editor and invited editorials on all aspects of infectious diseases in neonates, children, and adolescents. Contributions are reviewed by one editor. In addition, all research manuscripts, review articles, commentaries, case reports, medical images and conference reports are subjected to double-blind peer-review by at least one external, independent referee.