Review Article

The Risks of Forceful Feeding: Lentil Soup Aspiration and Its Association to Hypersensitivity Pneumonitis (HP) in Weaning Young Children

Rahat Bin Habib[1], ARM Luthful Kabir[2]

Abstract

Forceful feeding practices, especially during the weaning period, are prevalent in South Asian countries, such as Bangladesh, where lentil soup is commonly introduced to young children. However, this practice can lead to accidental aspiration of food, resulting in hypersensitivity pneumonitis (HP), an immune-mediated inflammatory lung disease. This article explores the association between lentil soup aspiration due to forceful feeding and HP in pediatric patients. The pathophysiology of HP involves immune complex-mediated (Type III) and delayed-type hypersensitivity (Type IV) reactions, triggering inflammatory responses in the lungs, which may lead to chronic inflammation and fibrosis. Case studies reveal that male infants are particularly vulnerable, as their anatomical characteristics increase the likelihood of retaining aspirated particles in the lungs, which can result in granuloma formation and fibrotic lesions. Clinical features of aspiration pneumonia include persistent cough, fever, and dyspnea. Investigations such as chest radiographs, high-resolution computed tomography, and bronchoalveolar lavage are essential for diagnosis, revealing typical radiological patterns and granulomatous inflammation. Treatment includes corticosteroids, proton pump inhibitors, and prokinetic agents, along with preventive strategies focusing on safe feeding practices. Educating caregivers about proper feeding techniques is crucial in preventing aspiration pneumonia and ensuring long-term respiratory health for children.

Keywords: Hypersensitivity Pneumonitis, Aspiration, Granuloma Formation, Chronic Inflammation, Fibrosis

Introduction

Forceful feeding to young children, especially during the period of weaning, is a common practice in our country. Hypersensitivity Pneumonitis (HP) is an inflammatory lung disease triggered by organic particles, which, with repeated exposure to chronic inflammation, fibrosis, and other respiratory complications in pediatric patients [1,2,3]. This condition is relevant in regions like Bangladesh, India, Pakistan, and other countries in Southeast Asia. This article explained the association between lentil soup aspiration due to forceful feeding and HP in young children.

In South Asia, particularly in Bangladesh, lentil soup is widely introduced during weaning. Mothers and near relatives often resort to forceful feeding methods using manual and bottle feeding, where children are restrained, and their mouths forcibly opened, which can lead to accidental aspiration and the combined effects of gastroesophageal reflux[4]. Studies indicate that such occult aspiration due to force-feeding practices can initiate persistent inflammatory responses in the lungs, potentially resulting in fibrosis in certain cases[5,6].

1. Assistant Professor, Dept. of Paediatrics, Shaheed Sayed Nazrul Islam Medical College, Kishoreganj, Mymensingh
2. Professor, Dept. of Paediatrics, Ad-din Medical College & Hospital, Moghbazar, Dhaka

Correspondence: Dr. Rahat Bin Habib, Assistant Professor, Dept. of Paediatrics, Shaheed Sayed Nazrul Islam Medical College, Kishoreganj, Mymensingh. Cell: +8801912368180, e-mail: ssmcdmc@gmail.com

Received Date: 20 July, 2024

Accepted Date: 23 August, 2024

It's found that male infants are particularly vulnerable to this form of HP due to anatomical factors such as increased airway resistance, which may facilitate the retention of antigens in the respiratory tract, thereby leading to chronic inflammation and granuloma formation, cellular infiltrates, leading to fibrotic lung lesions characteristic of chronic HP[7,8,9].

Pathophysiology of HP

HP represents a complex immune-mediated inflammatory response that primarily affects the lungs. HP's immunopathogenesis mainly involves immune complex-mediated hypersensitivity (Type III) and delayed-type hypersensitivity (Type IV) reactions[10]. When lentil soup is aspirated into the lower respiratory tract during forceful feeding, lentil proteins and other plant antigens enter the alveoli and bronchioles. These foreign antigens are recognized as non-self by the immune system, triggering a cascade of immune responses[11,12,13].

In immune complex-mediated hypersensitivity (III), inhaled lentil proteins bind with antibodies, typically IgG, forming immune complexes that deposit within the lung parenchyma. This complex deposition activates the complement system, leading to a release of inflammatory mediators, such as cytokines and chemokines, which attract neutrophils and other inflammatory cells into the alveolar spaces[14,15]. If antigen exposure persists, this acute inflammatory process can evolve into chronic inflammation.

The structure of lentils, particularly their cellulose-rich shell, with repeated exposure, can intensify this reaction to delayed-type hypersensitivity (IV). Here, antigen-presenting cells process lentil proteins and present them to T-helper cells, which activate macrophages and lead to granuloma formation. The cellulose acts as a granuloma-inducing substance, resulting in the formation of multiple patches or nodular lesions throughout the lungs. Chronic inflammation in response to these antigens can further result in fibrotic changes within the alveolar walls and bronchioles, creating a restrictive lung pattern, compromised gas exchange, and progressive respiratory dysfunction[16,17].

Histologically, these nodules are characterized by granulomas surrounding the small bronchi or bronchioles, containing acute and chronic inflammatory cells with accompanying fibrotic tissue. This fibrosis progressively disrupts normal lung architecture, causing long-term damage[18].

Case Studies

Food aspiration in childhood, particularly with hypersensitivity pneumonitis (HP), remains significantly understudied. While case reports suggest a link between lentil aspiration and lung inflammation, a critical gap exists, especially in regions like Bangladesh. Here, lentils are a dietary staple, and forceful feeding practices are prevalent, potentially increasing aspiration risks.[19,20,21].

In some case reports, children subjected to forceful feeding at the weaning stage often experienced episodes of coughing, choking, and respiratory distress shortly after being fed lentil soup. Physical examination and clinical findings frequently revealed hypoxemia and persistent respiratory symptoms, including cough, fever, and shortness of breath[22]. Bronchoalveolar lavage and high-resolution imaging typically confirmed inflammatory responses consistent with HP, and in many instances, granulomas were detected, often surrounding bronchioles and small airways. This pathological finding is attributed to the unique antigenic composition of lentils, whose cellulose-rich outer layer acts as a granuloma-inducing substance when aspirated into the respiratory tract[23,24].

One retrospective study observed that aspiration-induced HP in children often exhibited faster symptom onset and more severe progression than HP triggered by other inhaled antigens, possibly due to a combination of immune response immaturity and structural characteristics of the infant airway, which can trap particulates and antigens more readily in children than adults[25].

Such case studies underscore the importance of raising awareness among healthcare providers and caregivers about the risks of forceful feeding, especially with lentil-based foods. Comprehensive clinical protocols to assess aspiration risk, especially in infants with pre-existing respiratory conditions, may help prevent HP and its associated complications, including chronic lung damage and pulmonary fibrosis[26].

Clinical features

Aspiration pneumonia resulting from forceful feeding of lentil soup during weaning presents distinctive clinical features. Key symptoms include persistent cough, dyspnea, and fever following lentil aspiration. The cough is often unrelenting and can worsen after feeding sessions, particularly when forceful feeding is involved. Dyspnea manifests as labored breathing or increased respiratory effort, which may cause concern among

caregivers and is often accompanied by tachypnea. Fever generally appears within hours to days post-aspiration and may be persistent[27].

Taking a comprehensive history is critical to establishing a link between the child's symptoms and aspiration events during the weaning period. Inquiry about feeding practices and any episodes of gagging, choking, or coughing during meals can provide essential clues, as these events are often overlooked when diagnosing respiratory issues in infants and young children[28].

Investigations

The investigation of forceful feeding-induced aspiration pneumonia in infants reveals distinctive radiological and cytological findings. In cases where lentil aspiration during weaning causes pneumonia, chest radiographs (CXR) and high-resolution computed tomography (HRCT) scans provide critical diagnostic insights. CXR findings predominantly show involvement of the perihilar and lower lung lobes, with a high prevalence of reticulonodular patterns (44%), isolated consolidation (22%), and combined findings (33%)[29]. CT imaging further elucidates parenchymal abnormalities, where nodular patterns appear in 78% of cases, consolidations in 67%, reticular features in 22%, and ground-glass opacities in another 22%[30]. Notably, HRCT often reveals irregular consolidations and micronodules, particularly in the lower lobes and perihilar regions, reflecting the typical food aspiration pattern in infants[31].

Bronchoalveolar lavage (BAL) evaluation, including bacterial, fungal, and mycobacterial cultures, is essential to rule out infectious causes in patients from regions where tuberculosis and fungal infections are prevalent[32]. BAL cytology is valuable in assessing inflammatory responses, revealing leukocytosis in 89% of patients, with neutrophilic predominance in 57% and lymphocytic involvement in 31%[33].

Lung biopsies typically reveal aspiration pneumonia features with ill-defined inflammatory foci around bronchioles and alveoli, where vegetable tissue fragments, lentil particles, and cellulose wall fragments are visible [34]. Lung biopsy post-lentil aspiration shows foreign body granulomas with starch-rich cores from lentils, indicating a granulomatous inflammatory response. The biopsy reveals centrilobular nodules, a pattern resembling hypersensitivity pneumonitis (HP), with nodular inflammation and multinucleated giant cells characteristic of granulomatous reactions[35]. Chronic cases often show established fibrosis, resulting in persistent radiological findings[36].

Additionally, hypersensitivity to lentil proteins is confirmed through elevated serum IgG specific to lentil proteins, measured by immunoassays like enzyme-linked immunosorbent assay (ELISA), with levels often ranging from 21 milligrams of antigen-specific antibodies per litre (mgA/L) to over 200 mgA/L. Immunoglobulin E (IgE) specific to lentil proteins is also measured in some cases, further indicating the immune sensitivity contributing to pneumonitis[37].

Clinicians are encouraged to consider hypersensitivity pneumonitis as a differential diagnosis in pediatric patients from regions with forceful feeding practices, especially when there are symptoms of fever, persistent pneumonia, and multiple radiologic nodules.

Treatment and Management

Educating caregivers on weaning counselling for proper feeding techniques and recognizing the risks of forceful feeding plays a key role in preventing recurrent aspiration incidents. Initial assessments focus on ruling out other potential infections or underlying conditions before starting specific treatments for aspiration-induced HP[38].

Once infection has been ruled out, corticosteroids, such as prednisolone, are initiated to control inflammation. Prednisolone is typically started at an appropriate dose and then tapered every 1 to 2 weeks based on clinical response. For most children, the maximum duration of corticosteroid therapy spans up to six months to minimize potential long-term side effects. Additionally, proton pump inhibitors (PPIs), such as lansoprazole, are prescribed to 89% of patients to manage gastroesophageal reflux, which can exacerbate aspiration risk. Prokinetic agents, including domperidone, are prescribed to about 33% of children to enhance gastric emptying, further reducing the chance of aspiration[39].

Close follow-up and monitoring are essential for evaluating the response to treatment and adjusting therapy as necessary. Regular imaging and symptom tracking are advised to assess ongoing inflammation or signs of fibrosis, which may occur in chronic cases. Early intervention and preventive counseling prove to be highly effective in improving outcomes, preventing recurrence, and reducing the risk of chronic lung damage in affected infants and young children[40].

Prevention

Preventing aspiration pneumonia related to forceful feeding involves educating caregivers on safe weaning

practices and the dangers of forced feeding. Proper feeding techniques and allowing self-paced eating reduce aspiration risks significantly. Counseling caregivers about gentle, responsive feeding can prevent HP and improve overall child health[41].

Conclusion

The practice of forceful feeding during weaning, particularly in South Asia, where lentil soup is commonly introduced, has been identified as a significant risk factor for aspiration pneumonia and subsequent development of hypersensitivity pneumonitis (HP) in young children. Given the potential for long-term lung damage, it is critical to emphasize early identification, thorough investigation, and timely intervention in affected pediatric patients.

Investigation of suspected aspiration-induced HP in children should involve a comprehensive approach, including radiological assessment through chest X-rays and high-resolution computed tomography (HRCT) to identify characteristic lung changes, such as reticulonodular patterns, consolidations, and micronodules. Bronchoalveolar lavage (BAL) and lung biopsy play a crucial role in confirming the diagnosis, revealing granulomatous inflammation and foreign body reactions indicative of HP. Additionally, serum IgG and IgE testing for lentil proteins can be an important diagnostic tool in confirming hypersensitivity to lentils.

From a treatment perspective, a well-structured management plan is necessary to address both the acute inflammatory response and the prevention of further damage. Corticosteroids, such as prednisolone, should be administered to control inflammation, with gradual tapering based on clinical response. For children at high risk of aspiration due to gastroesophageal reflux, proton pump inhibitors (PPIs) and prokinetic agents may be prescribed to alleviate reflux and enhance gastric emptying, thereby reducing the likelihood of aspiration. Close follow-up and regular imaging are essential to monitor disease progression and adjust treatment as needed, particularly in chronic cases where fibrosis may occur.

Most importantly, education and preventive counseling for caregivers are key to reducing the incidence of aspiration-related HP. Promoting safe and responsive feeding techniques and discouraging forceful feeding practices are critical steps in safeguarding pediatric respiratory health. By implementing these measures, healthcare professionals can significantly reduce the risk of aspiration pneumonia, HP, and associated chronic respiratory complications in young children[42].

References

1. Lynch DA, Rose CS, Way D, King Jr TE. Hypersensitivity pneumonitis: sensitivity of high-resolution CT in a population-based study. AJR. American journal of roentgenology. 1992; 159(3): 469-472.
2. Patel AM, Ryu JH, Reed CE. Hypersensitivity pneumonitis: current concepts and future questions. Journal of allergy and clinical immunology. 2001; 108(5):661-670.
3. Selman M, Pardo A. Role of epithelial cells in idiopathic pulmonary fibrosis: from innocent targets to serial killers. Proceedings of the American Thoracic Society. 2006;3(4):364-372.
4. Khan JR, Awan N, Sheikh MT. A multilevel and spatial analysis of the infant and young child feeding practices and associated factors among the under-2 aged children in Bangladesh. Child Care in Practice. 2022;28(2):178-195.
5. Schuyler M, Cormier Y. The diagnosis of hypersensitivity pneumonitis. Chest. 1997;111(3): 534.
6. Selman M, Chapela R, Raghu G. Hypersensitivity pneumonitis: clinical manifestations, pathogenesis, diagnosis, and therapeutic strategies. In Seminars in respiratory medicine 1993 14 (5);353-364.
7. Townsel CD, Emmer SF, Campbell WA, Hussain N. Gender differences in respiratory morbidity and mortality of preterm neonates. Frontiers in pediatrics. 2017; 5:6.
8. Doershuk CF, Matthews LW. Airway resistance and lung volume in the newborn infant. Pediatric Research. 1969;3(2):128-34.
9. Hirschmann JV, Pipavath SN, Godwin JD. Hypersensitivity pneumonitis: a historical, clinical, and radiologic review. Radiographics. 2009;29(7): 1921-1938.
10. Girard M, Lacasse Y, Cormier Y. Hypersensitivity pneumonitis. Allergy. 2009;64(3):322-334.
11. Silva CI, Churg A, Müller NL. Hypersensitivity pneumonitis: spectrum of high-resolution CT and pathologic findings. American Journal of Roentgenology. 2007;188(2):334-344.

12. Stark JM, Mueller GA. Lung defenses: intrinsic, innate, and adaptive. Kendig and Chernick's Disorders of the Respiratory Tract in Children. Philadelphia, PA: Elsevier Health Sciences. 2012: 89-109.
13. Roberts RC, Moore VL. Immunopathogenesis of hypersensitivity pneumonitis. American Review of Respiratory Disease. 1977;116(6):1075-1090.
14. Lacasse Y, Cormier Y. Hypersensitivity pneumonitis. Orphanet journal of rare diseases. 2006;1(1):25-29.
15. Barrera L, Mendoza F, Zuñiga J, Estrada A, Zamora AC, Melendro EI, Ramírez R, Pardo A, Selman M. Functional diversity of T-cell subpopulations in subacute and chronic hypersensitivity pneumonitis. American journal of respiratory and critical care medicine. 2008;177(1):44-55.
16. M. Caillaud D, M. Vergnon J, Madroszyk A, M. Melloni B, Murris M, C. Dalphin J. Bronchoalveolar lavage in hypersensitivity pneumonitis: a series of 139 patients. Inflammation & Allergy-Drug Targets-Inflammation & Allergy). 2012;11(1):15-19.
17. Bourke SJ, Dalphin JC, Boyd G, McSharry C, Baldwin CI, Calvert JE. Hypersensitivity pneumonitis: current concepts. European Respiratory Journal. 2001; 18(32): 81S-92S.
18. GARDNER GM. Hypersensitivity Pneumonitis: Clinical Manifestations, Pathogenesis, Diagnosis, and Therapeutic Strategies. Radiology. 1994 ;192(1): 170-177.
19. Quirce S, Vandenplas O, Campo P, Cruz MJ, de Blay F, Koschel D, Moscato G, Pala G, Raulf M, Sastre J, Siracusa A. Occupational hypersensitivity pneumonitis: an EAACI position paper. Allergy. 2016; 71(6):765-779.
20. Weir K, McMahon S, Barry L, Ware R, Masters IB, Chang AB. Oropharyngeal aspiration and pneumonia in children. Pediatric pulmonology. 2007;42(11):1024-1031.
21. Sampson HA. Update on food allergy. Journal of allergy and clinical immunology. 2004;113(5): 805-819.
22. Furlong K. The role of nutritional factors in acute respiratory tract infections. University of Toronto (Canada); 2015.
23. Özdemir Ö. Various clinical presentations of food allergy in children. Asthma Allergy Immunology. 2014;12(2):70-82.
24. Mukhopadhyay S, Katzenstein AL. Pulmonary disease due to aspiration of food and other particulate matter: a clinicopathologic study of 59 cases diagnosed on biopsy or resection specimens. The American journal of surgical pathology. 2007; 31(5):752-759.
25. Yi ES. Hypersensitivity pneumonitis. Critical reviews in clinical laboratory sciences. 2002;39(6):581-629.
26. Silveyra P, Fuentes N, Rodriguez Bauza DE. Sex and gender differences in lung disease. InLung Inflammation in Health and Disease, Volume II 2021; 227-258.
27. Collaco JM, McGrath-Morrow SA, editors. Pediatric swallowing and feeding: assessment and management. Pulmonary manifestations and management considerations for aspiration. Pediatric Swallowing and Feeding: Assessment and Management. 2019;1:453.
28. Weir K, McMahon S, Barry L, Masters IB, Chang AB. Clinical signs and symptoms of oropharyngeal aspiration and dysphagia in children. European Respiratory Journal. 200 ;33(3):604-611.
29. Tutor JD, Gosa MM. Dysphagia and aspiration in children. Pediatric pulmonology. 2012 ;47(4): 321-337.
30. Hassen M, Toma A, Tesfay M, Degafu E, Bekele S, Ayalew F, Gedefaw A, Tadesse BT. Radiologic diagnosis and hospitalization among children with severe community acquired pneumonia: a prospective cohort study. BioMed research international. 2019;(1):6202405.
31. Arvedson JC. Swallowing and feeding in infants and young children. GI Motility online. 2006 .
32. Senda M, Harada N, Kanda K, Kako T, Urano M, Ito M. Pneumocephalus caused by Enterobacter cloacae meningoencephalitis following cerebral infarction. Neurology and Clinical Neuroscience. 2025;13(1): 66-68.
33. Lacasse Y, Cormier Y. Hypersensitivity pneumonitis. Orphanet journal of rare diseases. 2006;1(1):25.
34. Gami A, Rinaldi K, Degefe YT, Vosoughi AS, Lee D, Maleki Z. Bronchoalveolar Lavage in a Pediatric Population: A Correlation of Clinical Symptoms and Cytomorphologic Findings. American journal of clinical pathology. 2022;157(5):678-684.

35. Owayed AF, Campbell DM, Wang EE. Underlying causes of recurrent pneumonia in children. Archives of pediatrics & adolescent medicine. 2000; 154(2): 190-194.

36. Haque T, Basera P, Singh T, Arif D. Lentil aspiration pneumonitis. The Southwest Respiratory and Critical Care Chronicles. 2024;12(52):29-32.

37. Komiya K, Ishii H, Umeki K, Mizunoe S, Okada F, Johkoh T, Kadota JI. Impact of aspiration pneumonia in patients with community-acquired pneumonia and healthcare-associated pneumonia: a multicenter retrospective cohort study. Respirology. 2013; 18(3): 514-521.

38. James JM. Respiratory manifestations of food allergy. Pediatrics. 2003;11(11);625-1630.

39. Adil E, Al Shemari H, Kacprowicz A, Perez J, Larson K, Hernandez K, Kawai K, Cowenhoven J, Urion D, Rahbar R. Evaluation and management of chronic aspiration in children with normal upper airway anatomy. JAMA Otolaryngology–Head & Neck Surgery. 2015;141(11):1006-1011.

40. Brahmer JR, Lacchetti C, Schneider BJ, Atkins MB, Brassil KJ, Caterino JM, Chau I, Ernstoff MS, Gardner JM, Ginex P, Hallmeyer S. Management of immune-related adverse events in patients treated with immune checkpoint inhibitor therapy: American Society of Clinical Oncology Clinical Practice Guideline. Journal of Clinical Oncology. 2018;36(17):1714-1768.

41. Wolter NE, Hernandez K, Irace AL, Davidson K, Perez JA, Larson K, Rahbar R. A systematic process for weaning children with aspiration from thickened fluids. JAMA Otolaryngology–Head & Neck Surgery. 2018;144(1):51-56.

42. Carson BS, Losey RW, Bowes Jr WA, Simmons MA. Combined obstetric and pediatric approach to prevent meconium aspiration syndrome. American journal of obstetrics and gynecology. 1976;126(6): 712-715.