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Delhi - NCR

President's Message

At the outset let me express my deepest gratitude to every member and office bearer for giving me this opportunity to serve the Pediatric Respiratory Society Delhi and NCR. Working on this position is a big responsibility and I take a lot of inspiration from my distinguished predecessors in discharging my duties to the best of my abilities.

I had been involved with PRS activities for many years now. I noticed the dedication of past office bearers in administrative work and academic activities related to our society. To continue with same spirit and zeal I shall encourage and expect same dedication and involvement of my teammates. During few video meetings with my office bearers we had discussed and taken few important decisions. The most important decision was to start publication of Pediatric Respirology Journal. I believe this was most the awaited and will enhance the academic achievements of PRS. I request all the members to submit review articles, case reports, and interesting clinical or radiological images for publication. Please encourage your fellows and residents to write articles and case reports for this journal.

We have also planned to start awareness and education sessions in different schools about childhood asthma. This initiative is to educate growing children about the disease process and to remove misconceptions and taboos related to the disease and inhalation therapy.

It has been decided to organize annual PRS meeting on 16 October 2022 (Sunday) at Swarn Jyanti Auditorium, Lady Harding Medical College and Kalawati Saran Children Hospital. I sincerely thank Dr Varinder Singh and Dr Kamal Kumar Singhal for making all the efforts to ensure availability of auditorium. We need blessings and guidance of all seniors and experienced organizers of previous annual meetings. Also request to all the executive members to help and participate in the organization of this annual event.

Finally request you all to participate in bimonthly PRS clinical meetings regularly and insist on your junior colleagues also to attend the same.

We will also make all efforts to organize our bimonthly clinical meetings regularly. Please encourage your residents and fellows to attend this meeting.

Regards

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The Journal of Pediatric Respirology is a peer-reviewed, quarterly journal that publishes one review article, two case reports and one clinical / radiological image which provides new information.

Manuscript Submission

Submission of a manuscript implies that the work described has not been published before; that it is not under consideration for publication anywhere else; that its publication has been approved by all co-authors, if any, as well as by the responsible authorities – tacitly or explicitly – at the institute where the work has been carried out. The publisher will not be held legally responsible should there be any claims for compensation.

Online Submission

Please send the article by 5th July via email to : neetu.talwar@fortishealthcare.com;
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Please refer to the following instructions for the manuscripts:

Case Reports:

Clinical cases highlighting some unusual or new but "clinically relevant" perspectives of a condition, highlighting some new or unusual aspect regarding etiopathogenesis, clinical recognition/ reasoning in establishing clinical diagnosis, investigations in establishing etiological diagnosis or management of a condition that adds to the existing body of knowledge.

Clinical Images:

Only clinical photographs with/without accompanying skiagrams or pathological images are considered for this section. The image should clearly identify the condition and display the classical characteristics of the clinical condition.

First page file

The 'First Page File should carry

- The title of the article
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- The name of the department(s) and institution(s) to which the work should be attributed
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- Source(s) of support in the form of grants, equipment, drugs etc. should be disclosed
- A short running title of no more than 50 characters (count letters and spaces) must be provided. For example, your manuscript heading "Pediatric Lung Ultrasound (PLUS) In The Diagnosis of Community-Acquired Pneumonia (CAP) Requiring Hospitalization"may be given a short title as: "Lung ultrasound in hospitalized pneumonia children" which is of 45 characters.
- Acknowledgements - Acknowledgements may be made to contributions that need acknowledging but do not justify authorship, such as: General support by a department chair, Acknowledgements of technical help
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Case Report

- **Abstract and keywords:** A structured abstract is to be sent in case of Case Report (up to 250 words). It should be brief (up to 250 words) have the following headings: Background (what is unique about the case and how does it add to existing literature), Clinical description (salient clinical symptoms and clinical findings), Management (salient confirmatory diagnostic test, specific intervention(s) and outcome) and Conclusion (the primary lesson learnt from this case). Three to five key words should be written in case report which should be different from those used in title.
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other differential diagnoses. The 'Management and Outcome' section should outline the diagnostic approach (with associated reasoning), details of confirmatory and relevant supportive investigations, any diagnostic challenges encountered, prognostic tests (when applicable), type and details of therapeutic interventions (as per TIDieR guidelines) with rationale and supporting levels of evidence. The outcomes should be both objective and subjective based on the treating clinician's judgement and patient respectively (whenever possible). If applicable, a figure or graph depicting timelines of clinical progression, therapeutic interventions and/ or clinical outcomes should be included. The 'Discussion' should include in-depth rationale of establishment of diagnosis and intervention, strength's and limitations of management, The concluding paragraph should be the primary lessons that emerged during the management of the case.

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- Up to 4000 words (excluding tables, references and abstract). The manuscript may have about 80 references.
- For Review articles non structured Abstract of 250 and 150 words respectively with 4-6 key words.

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- Introduction, Material and methods, Results, Discussion, Conclusions
- Standard references with accurate bibliographic information and punctuations
- Tables with title, legends or foot notes on a separate page
- Check your manuscript for any of the gross grammatical mistake
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- Manuscript pages should have continuous line numbers. Number all pages in sequence, including the abstract, figure legends and tables.
- Images (jpeg, gif, tiff, etc)
- Submit good quality images. Each Image should be less than 1MB in size.

Picture of the Month

Should have a brief case history and a commentary, all fitting on one page along with the pictures.Commentary should run to maximum 500 words with 5 latest references, running to maximum 1.5 pages. No Abstract is required.

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- Use 12-point font size (Times New Roman) and leave margins of 2.5 cm (1 inch) on all sides. The whole manuscript should be formatted in ‘portrait’ layout.
- Use italics for emphasis.
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- Use the table function, not spreadsheets, to make tables.

References

Follow **Indian Pediatrics** authors instructions.

Following should be observed while citing the references:

- References should be numbered consecutively in the order in which they are first mentioned in the text.
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- References should be cited in the text consecutively and should be identified by numbers in square brackets. Some examples: 1. Negotiation research spans many disciplines [3]. 2. This result was later contradicted by Becker and Seligman [5]. 3. This effect has been widely studied [1-3, 7].

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- All tables are to be numbered using Arabic numerals.
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- For each table, please supply a table caption (title) explaining the components of the table.
- Identify any previously published material by giving the original source in the form of a reference at the end of the table caption.
- Footnotes to tables should be indicated by superscript lower-case letters (or asterisks for significance values and other statistical data) and included beneath the table body.
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REVIEW ARTICLE

EMPYEMA THORACIS IN CHILDREN

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Background

In a healthy state, a small amount of fluid lies between the parietal and the visceral pleura. The fluid continues to be filtered and reabsorbed by the lymphatic system within a very delicate balance of hydrostatic and oncotic pressure forces. When an imbalance occurs, fluid will accumulate at a rate faster than the lymphatic system is capable of draining.

Types of fluids in the pleural space

The aetiology of the fluid accumulation will determine the nature of fluid. A transudate results from a change in the transpleural pressure balance, and the resulting fluid will have a relatively low leukocyte count, lower levels of protein (1). However, exudate results from either increased vascular permeability of pleural capillary beds or impaired lymphatic drainage, such as seen in infection resulting in an acidic, high in white blood cells and lactate dehydrogenase but low in glucose (2).

The most widely used criteria to differentiate between transudate and an exudate are the Light's criteria which have been used since long in adults. They are summarised in Table 1 (3). Criteria favouring exudate are as shown on Table 1:

Pleural fluid protein/serum protein	>0.5
Pleural fluid LDH/serum LDH	>0.6
Pleural fluid LDH	>2/3upper limit normal serum LDH

Table 1: Light's criteria

Other differences between transudate and exudate are summarised in Table 2.

Characteristic	Exudate	Transudate
Pathogenesis	Increased vascular permeability in any inflammatory process	Ultrafilterate of plasma , no increased vascular permeability
Protein content	High (>4g/dl)	Low (<3g/dl)
Gross appearance	Thick/turbid/pus like	More often clear fluid
Cells	Highly cellular, most often polymorphonuclear cells	Cell count is low, usually mesothelial cells
Conditions seen	Pus as in empyema	Ascites/ pleural effusion in congestive heart failure , nephrotic syndrome

Table 2: Differences between transudate and exudate

By far, the most common type of pleural effusion in children is a para-pneumonic effusion (4). This results when there is a disruption of equilibrium between pleural fluid secretion and pleural lymphatic drainage due to underlying pneumonia. It classically presents as a syndrome of progressive pneumonia not responding to treatment and can be divided into the following three stages (5).

i. Exudative stage- simple parapneumonic effusion with low leukocyte count
ii. Fibrinopurulent stage- deposition of fibrin resulting in septations and loculations. An increase in leukocytes occurs and microorganisms invade the pleural space, resulting in purulent material filling the pleural cavity. A parapneumonic effusion that contains pus is called empyema.
iii. Organisational stage - fibroblasts enter the pleural cavity forming tight fibrous membranes.

Empyema is defined as pus in pleural space. Its most common cause is bacterial pneumonia. Viral pneumonias and Mycoplasma infections can also result in parapneumonic effusion but are less likely to require surgical interventions. Empyema thoracis constitutes approximately 5-10% of cases seen by paediatrician in India (6,7).

In an analysis of 150 cases from a tertiary care centre in north east India, mean age of presentation was 4.74 ± 3.53 years and two thirds of the children were under 5 years with male predominance (8).

Risk factors

Children with risk factors such as - immunodeficiency, malignancy, genetic conditions such as down's syndrome, congenital heart disease and cystic fibrosis – are at an increased risk of death (9, 10).

Etiopathogenesis

The reasons for development of empyema in healthy children are multifactorial. Transcription factors nuclear factor (NF- κB) plays a key role in host innate and adaptive immune responses and may play a role in the development of empyema (11).

The bacterial load may be another factor playing a role. Esposito and co-workers demonstrated that in their cohort of 72 otherwise well children less than 5 years of age with community acquired pneumonia (CAP) due to Streptococcus (S.) pneumoniae, and those with high bacterial loads are more likely to have an associated parapneumonic effusion (12).

Furthermore, Munoz-alma et al-were able to show that bacterial load correlated with serum inflammatory markers (CRP), hospital length of stay, and number of hours of pleural drainage (13,14). Delay in initiation of appropriate antibiotics may be a reason for the evolvement of empyema (9). Precipitating viral infections may be another factor that may be contributing to the development of empyema (9).

Microbiology

Common causative organisms of empyema are Streptococcus pneumoniae and Staphylococcus aureus, Escherichia coli, Haemophilus influenza and Klebsiella pneumoniae, Streptococcus pyogenes and uncommon causative organisms are Mycobacterium tuberculosis and cryptococcus neoformans (15).

However, with availability of better and newer antibiotics and introduction of newer vaccinations, profile of bacteria causing empyema has also changed. Empyema caused by cephalosporin resistant S. pneumoniae and methicillin resistant Staphylococcus aureus has also been increasing (16).

Mycobacterium tuberculosis is an important differential diagnosis in patients with chronic empyema thoracis (17, 18). Parasitic causes (Entamoeba histolytica, Echinococcus granulosus and Paragonimus westermani) should be considered if the empyema is bilateral (19).

Other causes of empyema include Streptococcus pyogenes and other streptococcal species, including S. viridans and S. milleri. Gram negative anaerobic organisms and specific fungi are known to cause empyema in rare cases usually following risk factors such as aspiration, immunocompromised state or nosocomial infection (20).

In an analysis of 150 cases of empyema from north-eastern part of our country (21), pleural culture was positive in 48 (32%). Streptococcus pneumoniae was isolated in 31 (64.6%) cases followed by Staphylococcus aureus in 11 (22.9%) patients. Other organisms isolated included Klebsiella pneumoniae (3 cases, 6.3%), Haemophilus influenzae type b (2 cases, 4.2%) and Enterococcus (1 case, 2%).

Clinical manifestations of Empyema

By and large the most common presentation of empyema is fever, cough, dyspnoea and productive cough (22, 23). Child may be tachypnoeic, with shallow breathing to minimise pain caused by deep inspiration. Infection in lower lobes may present with abdominal pain. The child may splint the painful side by lying on the affected side or by refusing to move and change position. Chest examination reveals decreased chest expansion, dullness to percussion, classically described as stony dullness and decreased breath sounds on the affected side.

Children presenting with pneumonia and scarlatiniform rash, toxaemia,-with signs of circulatory failure or low leukocyte count, should be considered to be infected with Group A Streptococcus (GAS) bacteria resulting in streptococcal toxic shock syndrome (STSS). Risk factors for GAS empyema include recent corticosteroid usage and varicella infection (24). These children should be admitted in the PICU for meticulous cardiopulmonary monitoring.

Investigations

The first-line treatment for bacterial pneumonia includes antibiotics and respiratory support. Empyema should be considered in children who do not respond to appropriate antibiotics within 48 hours as evaluated by ongoing fever intensity, levels of inflammatory markers, and respiratory distress. A chest x-ray done in such a case may help to ascertain if pneumonia has worsened or the patient has developed a pleural fluid collection. Fluid will collect in dependent areas; hence an erect or lateral decubitus film may allow easier identification of a dependant opacification, loss of costophrenic angle or a meniscus suggestive of fluid. When an erect X ray is compared with a decubitus film, failure of the fluid to shift with a change in position is suggestive of loculated fluid. A chest x ray suggestive of right pleural collection is shown in Figure 1.

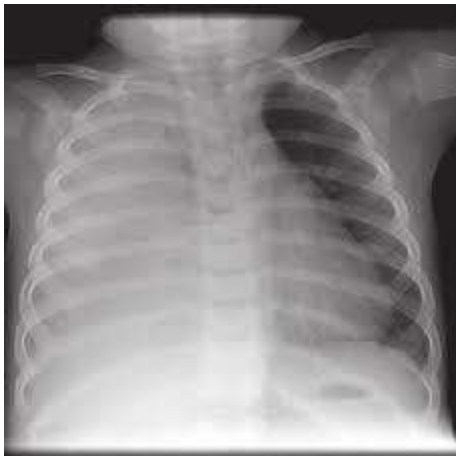

Figure 1: Right side pleural effusion with tracheomediastinal shift to left

Children who are not clinically improving or who have a chest x-ray suggestive of an effusion should undergo chest ultrasonography (USG) to allow a more accurate assessment of pleural fluid volume and will also better characterize the fluid. Ultrasound will also enable the grading of empyema. Jaffe and co-workers developed an ultrasound scoring system for children with empyema based on complexity (25).

Ultrasound scoring system: Grades I and II effusions did not have septations, whereas Grade III and IV empyemas were more complex:

Grade I was defined as anechoic

Grade II was defined as echoic fluid without septation.

Grade III defined as having thick septations

Grade IV had greater than one- third of the effusion comprising solid components.

These ultrasound findings are clinically relevant and may help to determine which effusions need further intervention, including those that are less likely to resolve with antibiotics alone. If the fluid is found to be complex and loculated, further interventions such as insertion of a chest drain or surgical intervention should be considered (26). Figure 2 shows a ultrasonography of lung showing anechoic pleural effusion with thickened pleura.

Ultrasound also allows fluid volume to be estimated and enables the identification of optimal sites for chest drain insertion; it is also more sensitive than CT in detecting early loculations and septations. Multi-loculation of empyema is defined on the basis of USG/CECT finding showing presence of two or more loculations that were separated and without communication. Loculations were defined by one or more of the following criteria- failure of effusion to layer on decubitus film, fixed fluid in abnormal locations, septations seen on USG or CT scan (present prior to instrumentation).

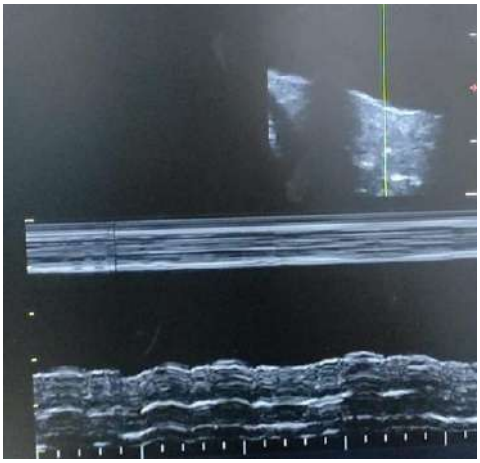

Figure 2: Ultrasonography of lung showing anechoic pleural effusion with thickened pleura (bright white line) and associated collapse of lung (shred sign) in top portion. Lower portion shows M-mode appearance with sinusoidal pattern of pleura.

Contrast-enhanced computerized tomography (CECT) chest should be done to delineate parenchymal and mediastinal pathologies with greater sensitivity, such as cavitary necrosis, pneumatoceles and mediastinal lymph nodes particularly in suspected pulmonary tuberculosis. CECT chest does not alter the management of empyema thoracic in the majority of situations (25).

Initial blood investigations often demonstrate raised white cell count, inflammatory markers such as CRP, ESR or procalcitonin. Secondary hypoalbuminemia from loss of protein into pleural space or malnutrition may occur with large effusions. Reactive thrombocytosis is also very common and resolves after few weeks.

When thoracocentesis is performed, pleural fluid should be sent for pH, glucose, cytology, LDH, gram stain, culture and molecular testing like such as PCR. Pleural fluid may often be sterile owing to prior administration of antibiotics. A differential cell count may help in differentiating between bacterial, mycobacterial or non-infectious causes, as lymphocytes rather than neutrophils may predominate in the last two.

Pleural fluid for pH must be handled meticulously like arterial blood for pH. Uncomplicated parapneumonic effusion show pH more than 7.30, glucose more than 60 mg/dl and LDH less than 1000 IU/L (mostly less than 500 IU/L). Complicated parapneumonic effusion or empyema is characterized by pH less than 7.10, glucose less than 40 mg/dl and LDH more than 1000 IU/L. Most cases of anaerobic infection have malodorous empyema. Protein level and specific gravity is rarely helpful in differentiating stages of empyema. In some cases, with frank pus, organisms are neither seen on gram stain nor grown in culture. Such cases must raise a suspicion of chylous effusion which should be excluded by testing the fluid for neutral fat, pH and sedimentation values after centrifugation. Purulent empyema has acid pH and cell fragments will sediment where a chylous effusion will have a neutral pH and remain opaque after centrifugation. Tuberculous empyema can be confirmed by stains for acid fast bacilli in fewer than 25% cases but pleural biopsy and culture can diagnose more than 90% cases.

Management

The aim of treatment of empyema thoracis is to treat the microbial cause, sterilize the pleural fluid, and allow re-expansion of the lung to support the return to normal lung function. This occurs either through the gradual reabsorption of excess pleural fluid over time or by active drainage by tube thoracostomy or surgery.

All children with empyema should be admitted to the hospital and treated with high-dose intravenous (IV) antibiotics and respiratory support if required. Choice of antibiotics will depend on the severity of clinical presentation, the local incidence of microorganisms, and whether or not the child has any specific risk factors such as immunocompromised state, aspiration or hospital-acquired infection.

In the absence of an identified causative organism, the choice of antibiotic should be determined by local community-acquired pneumonia guidelines and should include coverage of S. pneumoniae, S. pyogenes, and S. aureus. IV antibiotics are given until the child is afebrile, failure to respond to antibiotics should warrant consideration of an alternative regime. Data regarding duration of antibiotics are scarce, however, BTS (British thoracic society) guidelines note that IV therapy is generally given until the patient has been afebrile for at least 24 hours and it should continue until the chest drain is removed (27). Ongoing oral antibiotics are recommended for a minimum of 1 week, with some guidelines advocating up to 4 weeks of oral antibiotics (28). The choice of oral antibiotics should be determined on the basis of the organism identified or the class of IV antibiotics that led to clinical improvement.

Other aspects of management of empyema in children include management of hydration with IV fluids as required, analgesia, and antipyretics for comfort. The analgesic regimen should be optimised to encourage early mobility and maximize opportunities for lung expansion. Supplemental oxygen should be given if saturations fall below 93% in room air. Chest physiotherapy does not play a role in the management of empyema, but physiotherapy support with early mobilization, deep breathing and coughing is important.

Most cases of empyema will respond to conservative management, defined as antibiotics alone or with chest drain insertion, particularly for patients with small effusions and no respiratory compromise. The rates of response to conservative management are varied; in one case series Gocmen et al reported a 92% success rate in treating empyema with antibiotics and drainage alone (29). However, most centres do not report this high success rates. Even if conservative management is successful, this approach results in a more prolonged stay in hospital compared with treatment by surgery or with fibrinolytics.

Children who are acutely unwell, with severe respiratory distress, who are hypoxic or have moderate to large empyema should not be managed with antibiotics alone; surgical intervention should be considered early. The specific intervention will depend on the level of service and clinical experience of the treating staff. The most commonly performed interventions include insertion of a chest drain or thoracoscopic VATS (video-assisted thoracoscopic surgery) (30).

Patients with loculated fluid, or frank pus, or smear positive fluid, or fluid with pH less than 7.10, glucose less than 40 mg/dl and LDH more than 1000 IU/L, require chest tube drainage. There is sufficient evidence to suggest that a small chest tube (< 14F or pigtail is

sufficient to drain an empyema. Smaller percutaneous drains have the advantage that they can be easily inserted under sedation by the Seldinger technique, are less painful and allow early mobilization. The initial drainage rate should be monitored; chest drains should be connected to an underwater drainage system which must be kept below the level of the patient's chest (27). The drain should be removed if there is clinical resolution and should never be clamped if bubbling, as this is an indication of air leak, and clamping will precipitate the development of a pneumothorax.

If a chest drain is inserted, intrapleural fibrinolytics may be used, as they shorten the hospital stay compared with standard chest drainage, especially in those with multiloculated empyema. Fibrinolytics are infused via the chest catheter into the pleural space to allow lysis of pleural septations and fibrous strands and to clear lymphatic stomata so as to re-establish physiologic drainage from the pleural space. Urokinase, streptokinase, tissue plasminogen activator, and alteplase, all have been used in adults and have been found safe in clinical trials in children as well. However, as of now, there is insufficient evidence to favor the use of fibrinolytics (30). Fibrinolytics are instilled intrapleurally via the chest tube and the chest tube is clamped for 4 hours. The dose may be repeated if chest drain > 100 ml and multiloculated septations are persisting on ultrasonography.

The surgical options available are video-assisted thoracoscopic surgery (VATS), thoracoscopic debridement and irrigation, and decortication. VATS is effective in lysis of adhesions, especially in multiloculated empyema, however, it is useful only when taken up early in the course, not useful once the organizing stage has set in. Thoracoscopic debridement and irrigation may also be useful in multiloculated empyema is reserved for cases with a fibrotic visceral peel that restricts the expansion of the underlying lung. Decortication is useful if the above measures have failed.

Historically, surgical intervention was recommended only once conservative management with antibiotics alone had failed, or in the case of chest tube drainage and fibrinolytics with disease progression or if there was ongoing clinical toxicity. This is the recommended approach in the BTS guidelines as there are no evidence-based criteria for the timing of surgical intervention.

There is no difference in clinical outcomes between children who are treated with chest catheter drainage and fibrinolytics and VATS (30).

VATS may be preferred option in some children, if they can be identified early, who are at risk for failed fibrinolytic treatment. Livingstone et al have reported that children with positive blood culture, requirement for immediate intensive care admission, and those without complex septations may be at greater risk of fibrinolytic treatment failure (31).

Monitoring progress

Routine daily imaging as chest x ray is not recommended for monitoring the progress of empyema, as it does not alter management. More useful indicators of improvement are– defervescence, decreased respiratory distress and decreasing oxygen requirements and respiratory support.

Follow up of empyema

Fortunately, children rarely die from empyema, unlike adults where mortality is as high as 20% (32), however the burden of empyema on healthcare resources is substantial (33-35). Chest x-rays usually are abnormal at the time of discharge but this does not alter management. A delayed CXR should be performed in all children approximately 6 weeks after the event to ensure that the condition is resolving. Most CXR will return to near normal by 6 months.

Conclusion

Empyema thoracis constitutes 5-10 % cases seen by a paediatrician in our country. The condition carries significant morbidity. Hence, children with empyema should be identified early especially in the setting pneumonia which is not responding to appropriate antibiotics. Most of the children can be managed conservatively. Chest radiograph may be abnormal at discharge, but most of these patients recover radiologically on follow up in 6-8 months.

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CASE REPORT

Inflammatory Myofibroblastic Tumor of the Lung

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Background: An inflammatory myofibroblastic tumor (IMT) although rare, is a common cause of primary lung tumor in children. **Case characteristics:** This is a case of chronic cough and hemoptysis inaneleven-year-old child with right middle lobe radio – opacity. **Outcome:** Complete recovery after surgical resection of the medial segment of the right middle lobe. **Message:** Not all persistent radio-opaque shadows are due to tuberculosis.

Keywords: Inflammatory myofibroblastic tumor, primary lung tumor, radio-opaque shadows

Inflammatory myofibroblastic tumor (IMT) is a benign, quasineoplastic lesion with no definitive etiology, and consists of inflammatory and myofibroblastic spindle cells(1). There is very little data about its incidence, prevalence, natural history, and preferred treatment (2). This case is being reported because of its rarity with a relatively common initial presentation of persistent pneumonia.

Case Report

An 11-year-old female child had month-long symptoms of excessive cough with mucoid expectoration and hemoptysis, one year prior to presentation. She had been evaluated, and had normal counts and an ESR of 34 mm, with a negative Mantoux test. Chest x-ray had revealed a right-sided, lower-zone, round, homogenous opacity. In view of the symptoms, the child was empirically treated with anti-tuberculosis treatment for 6 months without demonstrating tubercular bacilli in sputum/respiratory secretions. Her cough persisted. After nine months, fever

recurred and there was another episode of hemoptysis. There was no history of any tuberculosis contact, bleeding diathesis, rashes, or joint pain. General examination revealed pallor and tachycardia. On respiratory system examination, there were crepitations on the right side. Chest x-ray showed persistence of right-sided, lower-zone, homogenous, round opacity (Figure. 1).

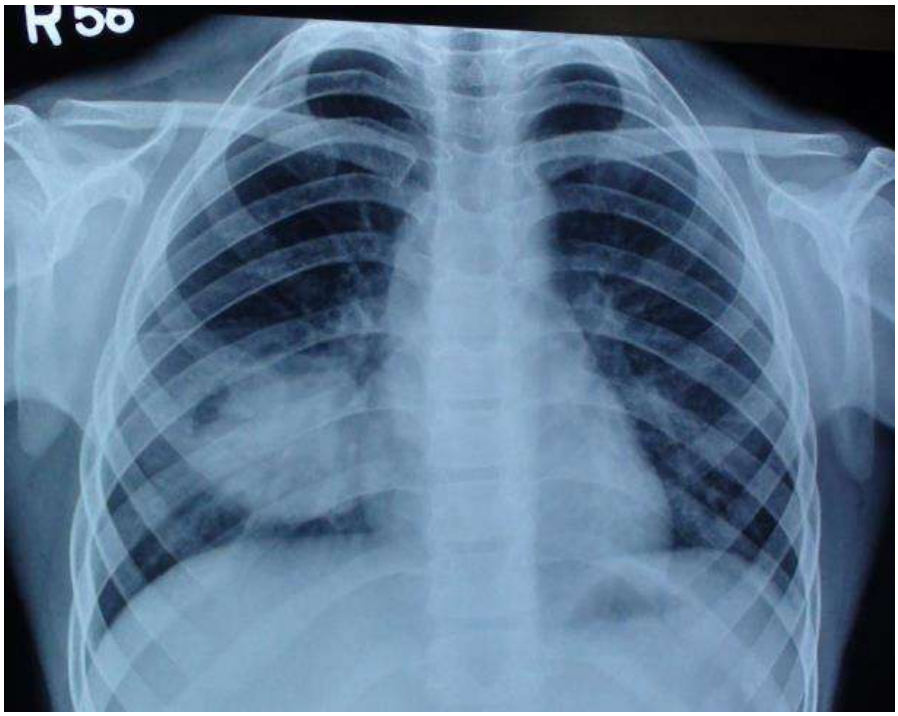

Figure. 1 - Plain radiograph of the chest: Round, well-circumscribed homogenous opacity in the right lower zone.

Other investigations included: Hb-9.4 g/dl; TLC-8000 thousand/µl, 66% neutrophils; 22% lymphocytes, 10% eosinophils, 2% basophils, ESR 20 mm, platelet count 358 thousand/µl, liver and renal function tests were normal, Echinococcus antibody test (Elisa) for hydatid serology was negative. Flexible bronchoscopy was done under local anesthesia, using Olympus BF Type 3C160, a 3.8 mm flexible bronchoscope. Airways were normal. Bronchoalveolar lavage (BAL) was collected, after which there was active bleeding from the right middle lobe. Immediately cold saline (4^0C) wash was given – 4 aliquots of 10 ml each. But the bleed persisted and therapeutic lavage with epinephrine (1:10 000 solution) was done after which it stopped (3). Bronchoalveolar lavage did not reveal any evidence of bacterial, fungus, or Mycobacterial infection. Cytopathology examination revealed no evidence of malignant cells.

CECT thorax revealed a well-circumscribed oval lesion in the right middle lobe with a large heterogeneously enhancing component. It was seen to communicate with the lateral segmental bronchus of the right middle lobe (Figure. 2).CT pulmonary angiography was normal.

Figure. 2- CECT Thorax: Soft tissue mass in the right middle lobe, which is enhancing, and has communication with the bronchi.

Expert opinion of the pediatric thoracic surgeon was sought and resection of the medial segment of the right middle lobe was advised. Surgical exploration revealed a tumor present within the lung parenchyma. The tumor was composed of prominent whorls of spindle-shaped cells punctuated by groups and sheets of foamy histiocytes. Interspersed lymphocytes and plasma cells were seen. Immuno-histochemistry revealed spindle cells to be diffusely positive for vimentin, smooth muscle actin was positive in a few areas, synaptophysin, myogenin, and Alk-1 were negative, Ki-67 was positive in intermixed lymphocytes, and few (1-2%) tumor cells. Inference on histopathology was an IMT of the fibrohistiocystic variant.

Our child had a complete recovery and was asymptomatic till 2 years' post-surgery, after which she was lost to follow-up.

Discussion

Our patient presented as a case of chronic, recurrent cough, fever, and hemoptysis, with persistent opacity in the right lung. The differential diagnoses at this point after investigations and CECT chest, were an oval mass - likely tumor in the right middle lobe, infection superimposed on an underlying congenital lung anomaly, hydatid cyst, metastasis from a tumor, or lymphoproliferative disorders. Intravenous antibiotics were given in view of the clinical presentation, but there was no response. The CECT chest helped to define the pathology and the course of further management. However, diagnosis was possible only after a histopathological examination of the tissue.

Although lung tumors are rare in the pediatric age group, IMT is the most common isolated, primary, tumor-like lesion of the lung among children and adolescents (4). They represent 20% of all primary lung tumors and more than 50% of all benign masses in children (5). This entity was first described by Brunn in 1939 and named as IMT by Umikeret al in 1954 because mimics malignant neoplasm clinically, radiologically and histopathologically (6) As per the World Health Organization (WHO) definition, IMT is "a lesion composed of a proliferation of myofibroblastic spindle and stellate cells with abundant eosinophilic cytoplasm mixed with infiltrative plasma, inflammatory cells, lymphocytes and eosinophils (7). Previously, IMT was also called inflammatory pseudotumor (IPT), plasma cell granuloma, and post inflammatory tumor (8).These tumors could arise from infections like the Ebstein Barr virus, bacterial infections, malignancies, autoimmune diseases, and due to excessive inflammatory reactions after surgery, however, the exact etiology and pathogenesis remain elusive (1). Apart from the lungs, it could be located in the pelvis, abdominal cavity, and retroperitoneal space. It is locally invasive or recurrent and rarely metastasizes.

The symptoms depend on the location of the tumor. As it grows slowly, the patient may be completely asymptomatic and is diagnosed on chest radiography. Clinical presentation could be one suggestive of chronic inflammation such as a low-grade fever, and weight loss, with investigations revealing anemia, thrombocytosis, polyclonal hypergammaglobulinemia, and elevated sedimentation rate or with cough, chest pain, and hemoptysis indicating bronchial irritation(8, 9). Chest x-ray findings could reveal a peripheral lesion, a solitary nodule usually in lower lobes, or rarely multiple nodules. HRCT chest reveals a solid mass with a homogeneous or heterogeneous enhancement that appears as a single, well-defined, lobulated mass. Punctate

calcifications mimicking malignant tumors are sometimes seen (10).Interventional bronchoscopy would be very useful in endobronchial lesions for diagnostic (biopsy samples) as well as therapeutic purposes (removal of tumor obstruction).

Surgical removal is the first choice of treatment. As per literature, a complete but conservative surgical excision, to prevent a recurrence could be done. In select cases, radical surgeryis preferred as slow progression to sarcoma and even metastasis can happen though rarely. (8, 11) Chemotherapy is preferred in cases of unresectable, multifocal, or metastatic disease and has a response rate of 50–60%, while radiotherapy is offered as a palliative approach, alone or in combination with chemotherapy treatment in extensive disease. (11) Parenteral steroids should be used with utmost precaution as the use of prednisone could worsen the lesions due to immunosuppression. (8)

Key Message

Not all persistent radio-opaque shadows are due to tuberculosis. Awareness of this lesion is essential not only for appropriate diagnosis and treatment but also to attempt a pre-operative recognition so that the child is spared from unnecessary radical surgery or harmful chemoradiotherapy.

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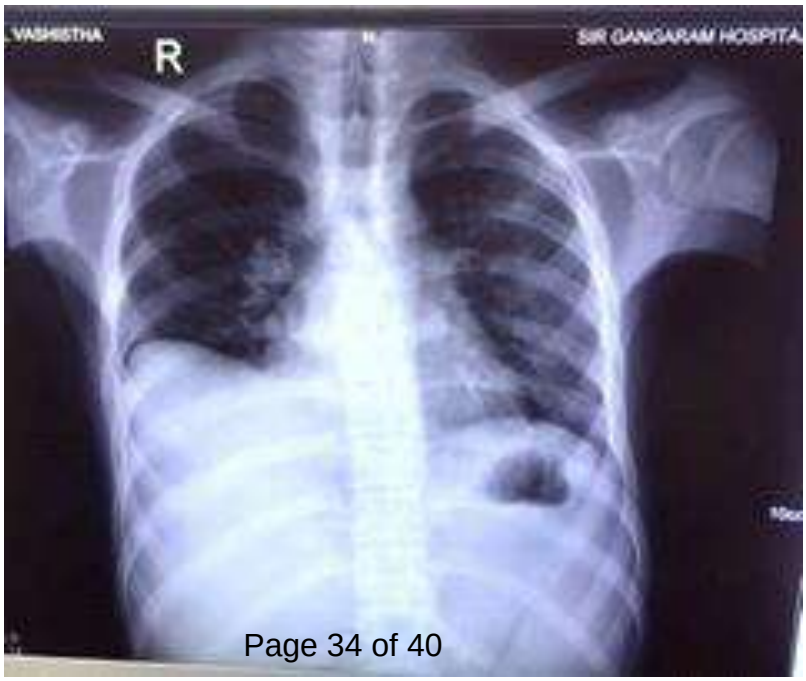
QUIZ-1

Your diagnosis please:



CASE REPORT

Exogenous lipoid pneumonia - A Dash of Oil in the Wrong Place!

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Background: Exogenous lipoid pneumonia as a result of hydrocarbon aspirationis not uncommon in children. **Case characteristics:**Chemical pneumonitis due to accidental lipid ingestion may be initially diagnosed as viral or bacterial bronchopneumonia if history is concealed. **Intervention:**PICU admission and noninvasive ventilation were instituted. **Outcome:** Gradual weaning of respiratory support was done over a period of 10 days of the PICU stay. **Message:** Aspiration of lipids causeshypoxemic respiratory failure and may be diagnosed as infective or interstitial lung disease if history is not revealed in the initial assessment.

Keywords: lipoid pneumonia, aspiration, respiratory support

Lipoid pneumonia can result from the accumulation of exogenous or endogenous lipids in the lungs (1). Exogenous lipoid pneumoniais not uncommon in our country and may occur due to accidental inhalation or aspiration of lipid-containing substances like medications and oily foods. Also, it is a common practice to instill oil in the ears and nose in certain parts of our country, which leads to aspiration of the oil and resultant chemical pneumonitis. Even if correctly diagnosed in time, these cases of pneumonia are often difficult to treat and may progress to acute respiratory distress syndrome and eventually pulmonary fibrosis, apart from complications of secondary infections. We report an interesting case of successfully managed lipoid pneumonia, to increase awareness of this condition and its management.

Case Report

A 4-year-old girl presented to the emergency room with bouts of spasmodic cough, fever, and fast breathing for the last 4 days. Fever was high grade, continuous in nature, and rapid breathing progressively increased prior to admission. She was hypoxemic with a saturation of 88% in room air, resp. rate of 86/min, with significant nasal flaring, suprasternal, intercostal, and subcostal retractions. General examination was normal, except for mild pallor. The anthropometric assessment was just above the 3rd centile for age. Auscultation revealed fine crepitations in bilateral infrascapular area, along with polyphonic wheeze, and bronchial breathing in the left infrascapular area. Except for sinus tachycardia (HR- 182/min), cardiovascular examination was normal. Past history was significant in terms of prolonged NICU stay for 6 weeks due to prematurity (30 weeks), ELBW (birth weight 700g), twin pregnancy, respiratory distress syndrome, surfactant therapy twice, and prolonged mechanical ventilation.Parents denied a history of frequent hospital visits, multisite infections, regurgitation of feeds, rapid breathing, or any exposure to environmental tobacco smoke. There was a history of infrequent nebulizationswith seasonal change, once or twice a year.

At this stage, we considered the possibilities such as mild bronchopulmonary dysplasia with exacerbation due to viral infection, bronchopneumonia, gastroesophageal reflux leading to microaspirations, or airway anomalies. There was an ongoing COVID pandemic, hence acute COVID pneumonia on the underlying chronic respiratory disease was kept as a possibility.

COVID RT PCR performed twice was negative. Laboratory investigations: except for neutrophilic leucocytosis and microcytic hypochromic anemia with high CRP (330 mg/L) - was noncontributory.

She continued to remain tachypneic RR- 80/min, SpO_2 - 90% in room air, and 94-95 % on O_2 by face mask @10 l/min, hence care was escalated to noninvasive ventilation, with CPAP of 10 cmH_2O. On day 5 of PICU admission, during a counselingsession with the mother for prognostication, a history of accidental ingestion of turpentine oil and subsequent choking was revealed. Noninvasive support was given for 8 days and the childwas shifted after 10 days of the PICU stay. Initial chest x-ray(Figure. 1) showed widespread bilateral punctate, ground glass densities involving perihilar, and middle lung zones, that subsequently became more organized to formbilateral middle and lower zone consolidation.

Figure. 1: Chest x ray on day 1 of admission. The child was on NIV support.

CECT chest showed bilateral dense dependent consolidation, along with areas of ground glass opacity, alveolar opacification, and interlobular septal thickening. There was also the appearance of a "crazy paving pattern"(1) in both the lower lobes (Figure. 2A) and the "sandwich appearance"(2)- subfissural zone of healthy lung between two pathological zones in coronal view (Figure.2B).

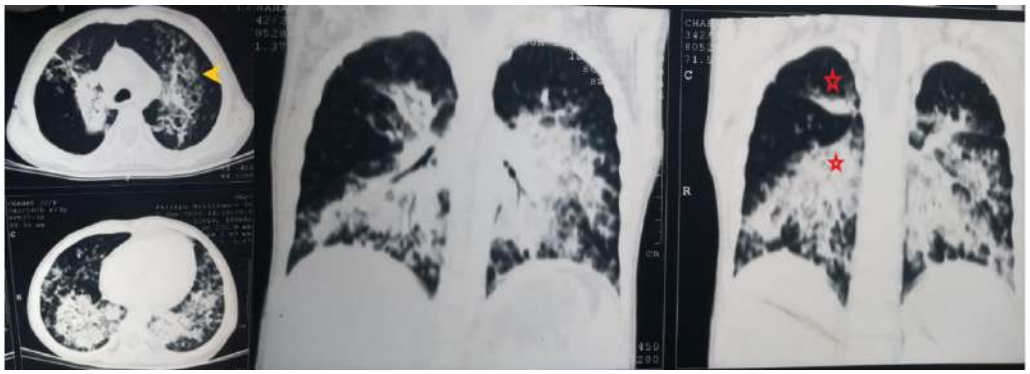

Figure. 2A **Figure. 2B**

Figure. 2A. CECT lung showing bilateral dense dependent consolidation, along with areas of ground glass opacity, alveolar opacification and interlobular septal thickening - crazy pavement pattern (yellow arrow). Figure. 2B. CECT lung showing "sandwich appearance" - subfissural zone of healthy lung between two pathological zones (red stars) in coronal view

As the child improved with pulse steroids and supportive care, we did not performbronchoscopy for apprehensions of pneumomediastinum or air leaks, so it was

classified assuspected exogenous lipoid pneumonia.She was discharged on oral prednisolone at 2 mg/kg/day with tapering over a period of 2 months. On discharge,her respiratory findings had improved with a respiratory rate of 35/minute and oxygen saturation of 94% at room air. Minimal, bilateral, fine crepitations were present with no retractions. However, findings on chest x-ray remained almost the same. Subsequently, the child was lost to follow-up.

Discussion

Lipoid pneumonia could result from the accumulation of both exogenous or endogenous lipids in the lungs. In the endogenous type, also called “cholesterol” or “golden” pneumonia, the fatlike materials are derived from the lung itself. The causecould be due to cholesterol and its esters which are released from the destroyed alveolar cell walls distal to an obstructive airway lesion, from lung tissue damaged by a suppurative process, or due to lipid storage diseases (1).Thepresent case emphasizes that the diagnosis of lipoid pneumonia goes unrecognized when a history of oil ingestion or aspiration is concealed by parents, which can be particularly confusing in a patient witha background of respiratory illness in the past.

Acute exogenous lipoid pneumonia (LP) appears radiologically as diffuse confluent ground-glass opacities, consolidation, with bilateral, segmental, or lobar involvement, predominantly in the middle and lower lobes of the lung (3). They may be apparent as early as 30 minutes of the episode of aspiration or as pulmonary opacities within 24 hours of the aspiration or inhalation event (4). In some cases, particularly those who have aspirated or inhaled a large amount of mineral oils or hydrocarbon products, pneumatoceles may occur within the regions of ground-glass opacities or consolidation. These may appear radiologically within the time window of 2–30 days after the aspiration or inhalation event (5). The most characteristic HRCT finding in lipoid pneumonia is the presence of consolidations with areas of fat attenuation, i.e., negative attenuation values. For instance, mineral oil has a computed tomographic attenuation value of −132 Hounsfield units (6,7).

Several factors increase the risk of exogenous LP such as forced feeding of medications with an oil base, oily food items, associated gastroesophageal reflux, neuromuscular disorders that result in swallowing discoordination or affect the cough reflex; anatomical or structural abnormalities of the pharynx and esophagus, such as Zenker diverticulum, gastroesophageal fistula, hiatal hernia, achalasia; psychiatric disorders; causes

of loss of consciousness. Some cultural practices such as nasal instillation of coconut oil have also been found in a detailed history. This entity may be difficult to diagnose because a history of oil ingestion is often missed or concealed, as happened in our case. The exposure is often identified only retrospectively after the diagnosis is made when a directed history is taken from the patient or their parents.

The clinical presentation of LP may mimic other pulmonary diseases because of its non-specific clinical and imaging signs. Sometimes, incidental radiologic findings may also be present. In our case,chest imaging was suggestive of a bilateral, symmetricalreticulonodular pattern with consolidation in dependent areas. Hence, the chance of aspiration was also considered. In contrast, patients with bronchopulmonary dysplasia or viral pneumonia will have an interstitial pattern, perihilar edematous bronchi, with air trapping.This led to further evaluation of history with parents. Bronchoscopy with bronchoalveolar lavage (BAL) can be successful in establishing the diagnosis of LP by demonstration of a high lipid-laden macrophage index and for rapid clearing of the aspirated oil by repeated bronchoalveolar lavages.However, we did not perform bronchoscopy as the child had a tumultuous PICU course and had responded very well to pulse dose steroids and supportive care. Hence we kept the diagnosis as suspected exogenous lipoid pneumonia due to witnessed choking on turpentineoil by the mother.

Treatment of LP in children is generally supportive, with the symptoms and imaging abnormalities resolving gradually within months after stopping exposure to the offending agent. Therapeutic lung lavage is used in some cases of LP. It might be effective, due to its ability to clear aspirated substances, inflammatory factors, and surfactants, which might impair the normal gas exchange and initiate the lung fibrotic process. (8)

Systemic corticosteroids are used to slow the inflammatory response and lessen the degree of fibrosis(9). However, steroid therapy remains controversial and should be reserved for severe cases. In our case, due to progressive hypoxia, we started the child on steroids. Roles of large volume brochoalveolar lavage and azathaoprine have reported previously. (10)

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QUIZ-1 ANSWER: SUBPULMONIC EFFUSION

DELHI RESPICON - 2022

4th Annual Conference
Organised by
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HIGHLIGHTS….

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- Chronic Lung Disease
- Tuberculosis - Update and Management
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