Management of community acquired pneumonia (CAP) in children: Clinical practice guidelines by the Paediatrics Association of Nigeria (PAN)

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Preamble

Against the background of the subsisting high childhood mortality indices in Nigeria, vis-a-vis the global efforts in the last 10 years to stem the tide (as articulated in the Millennium Development Goals), there has been a corresponding need to address the common causes of deaths in under-fives. In Nigeria, as is the case in many countries in sub-Saharan Africa, pneumonia remains a common cause of under-five mortality, accounting for 17% of deaths in this age-group. Indeed, it has been estimated that globally, a child dies from pneumonia every 20 seconds. With these in mind, and indeed the laudable goal of a 66% reduction of the national under-five deaths by the year 2015, a prompt recognition and management of pneumonia (in this vulnerable paediatric population) has become imperative. This was the logic that informed the current initiative of the Paediatric Association of Nigeria, aimed at formulating diagnostic, treatment and control policies with respect to paediatric community acquired pneumonia (CAP). This document is targeted primarily at health care providers working in Nigeria in centres with limited facilities as well as those working in tertiary hospitals.

Definition

Pneumonia is the inflammation of lung parenchyma due to pathogenic micro-organisms such as bacteria, viruses and fungi. Clinically, it is also defined as a condition typically associated with fever, respiratory symptoms, and evidence of parenchymal involvement, either by physical examination or the presence of infiltrates on chest radiograph. In order to facilitate early recognition, prompt treatment and referral of children with pneumonia, the World Health Organization (WHO) definition of pneumonia relies on simple clinical signs, such as tachypnoea and lower chest in-drawing (Table 1). According to this definition, severity classification of pneumonia in under-fives is non-severe and severe (Table 1).

Classification of pneumonia

Pneumonia can be classified using several parameters:

a. Source of infection: Community-acquired pneumonia (CAP) and hospital acquired infection (nosocomial) pneumonia. CAP is defined as pneumonia in a previously healthy child who acquired the infection outside a health facility or develops the illness within 48 hours of admission into a health facility.

b. Duration of Pneumonia: Pneumonia can be classified as "acute" (less than two weeks duration) and "chronic". Chronic pneumonias tend to be either mycobacterial or fungal. A microbiological classification involves knowledge of the aetiological agents.

c. Anatomical area(s) of involvement: Usually recognized based on chest radiographic parameters:

d. Lobar pneumonia: characterized by the presence of a smooth, dense homogenous opacity of a single lobe, or a segment of a lobe, of a lung. The aetiological agent is often Streptococcus pneumoniae. Multilobar pneumonia involves more than one lobe, often causing a more severe illness.

e. Bronchopneumonia: there are patchy changes in the lung around the bronchi or bronchioles.

f. Interstitial pneumonia: this involves the areas in between the alveoli. It is more likely to be caused by viruses or by atypical bacteria

g. Microbiological classification: This is based on the organism isolated/identified. This is exemplified by viral, bacterial, fungal, mycoplasmal and chlamydial pneumonia.
Epidemiology

Pneumonia is the leading cause of death in children under five years around the world, accounting for ~20% of all under-five mortality globally. Indeed, more than 155 million new episodes of clinical pneumonia occur in children under 5 years of age annually with about 10% of these being of sufficient severity to be life-threatening requiring hospitalization. In 2011, for example, an estimated 1.3 million children under five years died from pneumonia. Nigeria ranked fifth among the countries with the highest absolute number of new cases of clinical pneumonia in 2008 with an estimated 6.1 million new cases. By 2010, the estimated number of under-five children dying from pneumonia in Nigeria was more than 120,000, a disease burden that constitutes the highest in Africa. The burden of disease is mainly in the younger age groups. Furthermore, while 81% of deaths from pneumonia happen in children younger than 2 years, disease incidence has been shown to fall less rapidly with age than does mortality from the disease. Also, the global pneumonia incidence shows a higher prevalence among boys than girls with the largest differences recorded in South Asia regions. Indeed the recent series by Abdulkarim in Ilorin reported a male to female ratio of 1.5:1.

Aetiological Agents

The aetiological agents of CAP could be divided into bacterial, viral and fungal.

a. Bacteria

Common bacterial agents
- Streptococcus pneumoniae
- Haemophilus influenzae
- Staphylococcus aureus

Less common bacterial agents
- Klebsiella pneumoniae
- Mycoplasma pneumoniae
- Chlamydomphilia pneumoniae
- Non-typhoidal salmonella
- Non-typeable Haemophilus influenzae

b. Viruses

Common Viral agents
- Respiratory syncytial virus (RSV)
- Influenza A & B virus
- Parainfluenza

Less common viral agents
- Adenovirus (ADV)
- Human metapneumovirus
- Measles virus

c. Others

Mycobacterium spp
Pneumocystis jiroveci

Globally, the common pathogens of CAP and the corresponding paediatric population are:

General population of children

The two commonest bacteria are Streptococcus pneumoniae (30–50% of pneumonia cases) and H. influenzae type b (Hib; 10-30% of cases), and the main viral cause is RSV, but estimates of their relative importance vary in different settings.

HIV-infected children

Pneumocystis jiroveci and Mycobacterium tuberculosis are important causes of pneumonia, though bacterial causes remain the major cause of pneumonia mortality.

Severely malnourished children

Klebsiella pneumoniae, S. aureus, S. pneumoniae, E. coli, and H. influenzae are the major aetiological agents with very few data on the role of respiratory viruses and M. tuberculosis.

School-aged children

Mycoplasma pneumoniae is an important cause of pneumonia in school-aged children.

However, in Nigeria pathogens causing CAP in the under-fives are as shown in the table below:

<table>
<thead>
<tr>
<th>Table 1: Aetiological agents of childhood pneumonia in Nigeria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Studies</td>
</tr>
<tr>
<td>---------</td>
</tr>
<tr>
<td>Abdulkarim et al 2013</td>
</tr>
<tr>
<td>Falade et al 2009</td>
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<tr>
<td>Johnson et al 2008</td>
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<tr>
<td>Tagbo et al 2005</td>
</tr>
</tbody>
</table>

Results of aetiology of pneumonia studies in Nigeria are at variance with the global trend due to a) high pre-consultation antibiotic use; b) use of human blood to prepare blood agar; and c) lack of current method to distinguish coagulase negative Staphylococcus from S. aureus.
Predisposing/Risk Factors

Risk factors for CAP include the following:

<p>| Table 2: Risk Categories of Community Acquired Pneumonia (modified from Rudan et al Bull WHO 2008) |
|---------------------------------------------------------------|---------------------------------------------------------------|</p>
<table>
<thead>
<tr>
<th>Definite Risk Factors</th>
<th>Likely Risk Factors</th>
<th>Possible Risk Factors</th>
<th>Risk Factors for neonate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malnutrition (WAZ score &lt; -2)</td>
<td>Parental smoking</td>
<td>Mother’s education</td>
<td>Premature rupture of membranes</td>
</tr>
<tr>
<td>Low birth weight</td>
<td>Zinc deficiency</td>
<td>Day care attendance</td>
<td>Low birth weight</td>
</tr>
<tr>
<td>Non-exclusive breastfeeding</td>
<td>Mother’s experience as a caregiver</td>
<td>Outdoor air pollution</td>
<td>Preterm delivery</td>
</tr>
<tr>
<td>Lack of measles immunization</td>
<td>Concomitant disease, e.g.</td>
<td>Lack of exclusive breastfeeding</td>
<td></td>
</tr>
<tr>
<td>Household air pollution</td>
<td>Sickle cell disease, e.g.</td>
<td>Concomitant heart disease, e.g.</td>
<td></td>
</tr>
<tr>
<td>Overcrowding</td>
<td>Immunodeficiency states</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Pathophysiology and Pathogenesis of Pneumonia

The respiratory tract is replete with both specific and non-specific protective mechanisms which act in concert to keep the airways and alveoli free of both particulate materials and microbes.

The non-specific defense mechanisms include the nasal hair and nasal turbinates, the vocal cord, glottis, mucusiliary clearance and the cough reflex. Others include humidification, neutrophils, resident alveolar macrophages, airway secretions including lysozymes, iron binding proteins, complements and surfactant.

The specific defense system involves the coordinated activities of B and T lymphocytes resulting in activation of cytotoxic T cells and specific antibodies.

Microbes are introduced to the airway via inhalational or haematogenous route. When microbes evade the non-specific defense system they provoke an inflammatory response leading to exudates of plasma, neutrophils, lymphocytes, macrophages and inflammatory debris.

The inflammatory debris narrows the airway and increases airway resistance. The debris also causes partial or total occlusion of the smaller airways with resultant atelectasis and hyperinflation of some alveoli leading to increased work of breathing and wheezing. Furthermore, the increased alveolar diffusion barrier causes significant ventilation-perfusion mismatch and intrapulmonary shunt.

These pathophysiologic events are responsible for (1) tachypnoea (2) increased work of breathing (3) crepitations (4) reduced air entry (5) dull percussion note (6) wheeze/rhonchi and (7) fever.

Seeding of bacteria to the blood and other organs could also occur causing organ-specific manifestations, such as meningitis, septic arthritis, acute osteomyelitis, while the inevitable increase in pulmonary vascular resistance coupled with increased myocardial oxygen requirement may cause heart failure.

Clinical Features

The main objective of the initial clinical assessment is to decide if the child’s history and physical examination findings are suggestive of CAP.

Clinical Assessment

1. Relevant history questions:
   - Host-related factors: Age, immunization status, lack of exclusive breastfeeding, low birth weight, severe malnutrition
   - Environmental Factors: household air pollution e.g. firewood burning, passive tobacco smoking, season of the year, overcrowding and poor ventilation
   - Co-morbidities: heart disease, sickle cell disease, HIV infection, gastro-esophageal reflux disease

2. An initial physical examination should be performed for signs of respiratory illness and for fever. These signs include tachypnoea, evidence of increased work of breathing, cyanosis, auscultatory signs such as decreased breath sounds, crepitations and bronchial breath sounds. Other features to be looked for include evidence of other organ involvement such as heart failure (tachycardia, tender hepatomegaly), acute osteomyelitis, septic arthritis and meningitis

The following points should be noted, however:

1. Respiratory rates are best determined over a full 60-second period and inconsistencies require repeated observations. This is required in view of the effects of the peculiar behavioral and physiologic factors in children.
2. No single clinical finding is sufficient in determining the presence or absence of pneumonia; combinations of clinical findings are more useful.
3. The best individual examination measures in children less than 5 years are: nasal flaring (age < 12 months); oxygen saturation 90% or less in room air; tachypnoea; and retractions. The absence of tachypnoea alone or of all other signs of respiratory illness is highly suggestive of the absence of pneumonia.
4. Among children less than 5 years, especially in neonates and those with severe malnutrition pneumonia may be present without signs of respiratory illness.

| Table 3: Respiratory Rate Cut-offs for Children According to Age Groups |
|---------------------------------------------------------------|---------------------------------------------------------------|
| Age groups | Approximate normal respiratory rates (bpm) | Tachypnoea threshold (bpm) |
| Less than 2 months | 40 to 60 | ≥60 |
| 2 up to 12 months | 25 to 40 | ≥50 |
| 1 up to 5 years | 20 to 30 | ≥40 |
| ≥5 years | 15 to 25 | ≥30 |

Classification of severity of pneumonia

Children with pneumonia usually present with cough and/ or difficult breathing, fast breathing and fever. These children may either have severe or non-severe pneumonia, as defined below. This classification forms the basis of subsequent management:
a. Pneumonia (non-severe)

- Mild chest indrawing: (i.e. lower chest wall goes in when the child breathes in)
- Chest auscultation signs: decreased breath sounds, bronchial breath sounds, crackles or crepitations

b. Severe pneumonia

These children will have, in addition to the features of non-severe pneumonia, at least one or more of the following:

- Central cyanosis, or oxygen saturation 90% or less on pulse oximetry in room air
- Severe respiratory distress (e.g. grunting, very severe chest indrawing)
- Chest auscultation signs: decreased/absent breath sounds or vocal resonance as in pleural effusion, pleural rub
- Signs of pneumonia with a general danger sign: Inability to breastfeed or drink, lethargy or unconscious, convulsions.
- Presence of complications or co-morbidities: e.g. congestive heart failure, severe malnutrition and sickle cell disease

Clinical chest examination is useful in providing anatomical diagnosis (Table 4):

<table>
<thead>
<tr>
<th>Signs</th>
<th>Lobar pneumonia</th>
<th>Bronchopneumonia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest deformity</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Chest movement</td>
<td>Diminished or absent</td>
<td>Normal</td>
</tr>
<tr>
<td>Mediastinal shift</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Vocal fremitus</td>
<td>Increased</td>
<td>Normal</td>
</tr>
<tr>
<td>Percussion note</td>
<td>Dull</td>
<td>Resonant</td>
</tr>
<tr>
<td>Breath sound</td>
<td>Bronchial or vesicular</td>
<td>Vesicular</td>
</tr>
<tr>
<td>Added sound</td>
<td>Crepitations (crackles)</td>
<td>Crepitations (crackles)</td>
</tr>
<tr>
<td>Vocal resonance</td>
<td>Increased</td>
<td>Normal</td>
</tr>
</tbody>
</table>

**Diagnostic Evaluation**

Management of CAP can be in a community or hospital settings. Community setting includes: the home, health centres, community pharmacy shops/stores; as against hospital setting, i.e. emergency departments, out-patient and in-patient departments. Emphasis on community setting is on treatment of symptoms and prevention of progression to severe cases of pneumonia in order to avoid hospitalization.

For a child with suspected CAP in the community, there are no indicators for any general investigations. Investigation of any sort is not necessary and where this has been done, it has not contributed to outcome of management. We recommend instruction on respiratory rate count for mothers, pharmacy assistants and community health workers as part of capacity building for recognition of varying severity of the disease and appropriate referral option. Also recommended is the knowledge of the haemoglobin genotype of the child.

At hospital setting, aims of management include aetiological diagnosis, anatomical/pathological diagnosis and determination/correction of effects of the CAP on the child.

**Supportive Investigations**

- Anthropometry – weight, height, mid upper arm circumference
- Bedside determination of respiratory rate and pulse rate
- Pulse oximetry for oxygen saturation: Helpful in monitoring response to therapy and detection of cyanosis. Acceptable cut-off value for discontinuing oxygen therapy is SPO\textsubscript{2} 90% or more.
- Acute phase reactants (APR), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), procalcitonin (PCT). While these non-specific inflammatory markers may be of clinical benefit, their usefulness in differentiating the cause or indeed the severity of the CAP is doubtful.

**Diagnostic Imaging**

Diagnosis of CAP is commonly achieved by carefully considering the symptoms and signs, and in the majority of cases, further investigations are uncalled for, especially in resource-poor countries.

Plain chest radiograph is the commonest ancillary investigation for confirmation of CAP. Its main value is the identification of opacities in the chest radiograph. Indications for chest radiograph in CAP include:

a. Presence of significant chest retractions
b. Failure to respond to initial course of antibiotic therapy at 48 hours
c. Suspected CAP with complications, e.g. pleural effusion, pneumothorax
d. Progressive symptoms despite antibiotic therapy

In general CAP requiring hospitalization is an indication for requesting a chest radiograph. There is controversy regarding the timing and the specific views of the chest radiograph required. The majority of clinicians favour chest radiograph for severe pneumonia at presentation, while others favour chest radiograph as a pre-discharge recommendation. Follow-up chest radiographs are unnecessary in children who recover uneventfully from CAP. Commonly, anteroposterior (AP) view is all that is required. In the Nigeria situation, simultaneous AP and lateral views are preferred in order to assess additionally the hilum, paratracheal and paravertebral structures. Where massive effusion is suspected, lateral views should also be obtained following a substantial drainage of the effusion.

Possible chest radiograph findings indicative of CAP include: lobar infiltrates, interstitial infiltrates (bacterial,
viral, atypical pneumonia), lobar consolidation, atelectasis, nodular infiltration, hilar adenopathy, pneumatoceles, etc.

However, the ‘gold standard’ for the diagnosis of pneumonia is chest radiography. Nevertheless, some of the limitations of chest radiography include:

1. Interpretation of the chest radiographs in pneumonia varies as some studies classify only cases with alveolar consolidation as pneumonia, others include the presence of any pulmonary parenchymal infiltrates.
2. Poor agreement between radiologists on the presence or absence of infiltrates in paediatric chest radiographs even when standard reporting formats are used.
3. Chest radiographs predict the post-mortem diagnosis of pneumonia in severely malnourished children with 100% specificity but only 50% sensitivity.
4. Facility for chest radiography is not available in most health facilities in developing countries.

There is no sufficient evidence to recommend the routine use of ultrasound and computerized tomography scan in CAP.

**Isolation of Microbiologic Agents**

This is a desirable investigation in children with CAP in order to avoid antibiotics misuse and development of bacterial resistance. Available methods include blood culture, pleural fluid culture, nasopharyngeal culture, sputum (induced using 5% normal saline) culture, etc. However, the gold standard for sample recovery is lung puncture aspirate from infected region of the lung. Emphasis should be on the less invasive sampling methods. Although new molecular diagnostic tests are available, e.g., polymerase chain reaction (PCR), their usefulness in our hospital setting is limited.

**Recommendations for Management of Community Acquired Pneumonia**

**Introduction**

Results of aetiological studies of CAP in Nigeria, as well as its complications are essential to formulate its management. It is paramount to consider the management as first/alternative and second lines, which will fit into management at primary, secondary and tertiary levels.

A stepwise approach to management is preferred: In children with a history of fever, cough, and/or difficult breathing:

**Step 1:** Count the respiratory rate for one full minute when the child is awake and calm, or asleep. If the breathing is fast, consider pneumonia.

**Step 2:** Look for evidence of increased work of breathing (difficult breathing): in-drawing of the lower chest wall when the child breathes in and nasal flaring.

**Step 3:** Check for cyanosis (bluish discolouration) by looking at the tongue and buccal mucosa. Document the oxygen saturation using a pulse oximeter.

**Step 4:** Palpate for the position of the trachea.

**Step 5:** Percuss the chest for dullness, or hyper-resonance.

**Step 6:** Auscultate for bronchial breath sounds, crepitations or rhonchi.

**Step 7:** Look for complications such as heart failure (tachycardia, tender hepatomegaly), pleural effusion (stony dull percussion note, reduced/absent breath sound over the region of either or both chest regions), pneumothorax (hyper-resonance and reduced/absent breath sound over the upper and lateral region of the involved lung field).

**Step 8:** Look for signs of other organ involvement. Ask/determine if convulsion, lethargy, inability to drink or feed or not responding to calls is present. Presence of these features or any of the complications listed above indicates severe pneumonia.

**Step 9:** Classify the severity of pneumonia (using WHO classification; see above).

**Step 10:** Decide on who needs hospitalization. Criteria for management in the hospital are:

- Age less than 2 months
- Severe pneumonia
- Presence of complications or comorbidities
- SpO₂ 90% or less in room air.

**Step 11:** Decide on relevant investigations:

- Chest radiography is NOT required in children with pneumonia to be managed as outpatient.
- Do chest radiography in children with pneumonia needing hospitalization, more so in those children suspected of having complications such as parapneumonic effusion (pleural effusion, empyema) or pneumothorax.
- Routine full blood count is NOT required for children suspected of having pneumonia to be managed in the outpatient.
- A full blood count should be obtained for all children with severe pneumonia or sick enough to be hospitalized.
- Because malaria is a common co-morbidity in this environment, screen for malaria parasite.
- Blood culture should be obtained in sick children requiring hospitalization.
- Serum electrolytes, urea and creatinine, and random blood sugar should be obtained in children with severe pneumonia.
### Step 12: Give systemic antibiotics to all children with pneumonia

<table>
<thead>
<tr>
<th>Category of children</th>
<th>Outpatients</th>
<th>Inpatients</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>≤2 months</strong></td>
<td>Admit and treat as neonatal sepsis</td>
<td><strong>First line</strong></td>
</tr>
<tr>
<td></td>
<td>High dose Oral amoxicillin (90mg/kg/day in 2 divided doses) for at least 5 days</td>
<td>IV amoxicillin (150mg/kg/day in 3 divided doses) AND IV/IM gentamicin (5-7.5mg/kg once daily) for at least 5 days</td>
</tr>
<tr>
<td></td>
<td>Oral amoxicillin-clavulanic acid (amoxicillin component 90mg/kg/day in 2 divided doses) OR oral cefuroxime (20-30mg/kg/day in 2 divided doses) for at least 5 days</td>
<td>IV ceftriaxone (50-100mg/kg/day every 12-24 hours), OR IV cefotaxime (100-200mg/kg/day in 4 divided doses), OR IV/IM gentamicin (5-7.5mg/kg once daily) AND IV cloxacillin (100-200mg/kg in 4 divided doses) OR IV cefuroxime (150mg/kg in 3 divided doses) AND IV/IM gentamicin (5-7.5mg/kg once daily) for at least 5 days.</td>
</tr>
<tr>
<td><strong>HIV-infected children</strong></td>
<td>High dose Oral amoxicillin (90mg/kg/day in 2 divided doses) for 10 days</td>
<td>IV amoxicillin (150mg/kg/day in 3 divided doses) AND IV/IM gentamicin (5-7.5mg/kg once daily) PLUS high dose co-trimoxazole (20mg/kg/day of trimethoprim) for at least 10 days</td>
</tr>
<tr>
<td><strong>Children with sickle cell disease</strong></td>
<td>High dose Oral amoxicillin (90mg/kg/day in 2 divided doses) for at least 5 days</td>
<td>IV amoxicillin (150mg/kg/day in 3 divided doses) AND IV/IM gentamicin (5-7.5mg/kg once daily) PLUS oral erythromycin (60-100mg/kg/day in 4 divided doses) for at least 5 days</td>
</tr>
</tbody>
</table>

**Notes:**
Step down to appropriate oral antibiotics when improvement is sustained. For instance, cefpodoxime after ceftriaxone; Target pathogens in outpatients' treatment are *S. pneumoniae* and Hib; whereas in cases on admission, these as well as *S. aureus* and other bacilli are included; Maximum dose of gentamicin should not exceed 120mg; Chloramphenicol is not included in the antibiotic protocol because of its toxicity in the face of effective alternative antibiotics;

*Alternatives: Consider alternatives when first line drugs are not available or applicable or child has not responded to the first line drugs.

**Other Supportive Measures**

- Clear the airway using gentle suction as needed, always mouth before nose.
- Give supplemental oxygen if oxygen saturation is 90% or less, in room air or signs of severe respiratory distress are present. If pulse oximetry is not available give oxygen if signs of respiratory distress and or cyanosis are present.
- Give oxygen via nasal prongs or nasal catheters: 0.5-1L/min for children 0-2 months, 2-3L/min for children 3 months to 5 years, maximum of 4L/min for older children.
- Allow small frequent feeds/fluids if tolerated; feeding may also be done using appropriate size nasogastric tube.
- If feeds are not tolerated give intravenous isotonic fluid. Ensure it contains at least 5% glucose (e.g. 5% dextrose in 0.9% saline or ringer’s lactate with added glucose).
- For high grade fever (temperature ≥39°C), give paracetamol 10-15mg/kg 4-6 hourly, and ibuprofen if required.
- If widespread rhonchi is present (high-pitch continuous sound during expiration only or during both phases of respiration) give first dose of short acting bronchodilator such as salbutamol or albuterol and re-assess.
- Nursing care should be provided at least every 3 hours: check vital signs including oxygen saturation.
- The doctor should review the child at least twice daily.
When to Consider Referring a Child with Pneumonia to a Tertiary Centre/Getting a Specialist’s Review

- If child’s clinical state does not improve after 48 hours or worsens within this period
- When the child requires mechanical ventilation at presentation
- If oxygen saturation is persistently <90% despite supplemental oxygen
- If blood pressure remains low If the child has altered mental status

When to consider transfers to a Critical Care Unit

- When the child requires mechanical ventilation
- If blood pressure remains low or child requires inotropic agent(s) to maintain normal blood pressure
- If the child has altered mental status
- If oxygen saturation is persistently <90% despite supplemental oxygen
- Presence of other organ failure

When to Consider Discharge

- When clinical features such as fast breathing, respiratory distress and fever have resolved for at least 24 hours
- Child is feeding by mouth and tolerates oral medications and
- Caregiver is comfortable about discharge from hospital and capable of administering oral medication(s) if any

At Discharge

- Plan to review the child two days after discharge
- Review immunization record and make plans to get the child fully immunized if vaccines have been missed. In addition, children under 2 years should get recommended doses of pneumococcal conjugate vaccines (PCV) or *H. influenzae* Type b conjugate vaccines (Hib vaccine) if not already immunized
- Instruct caregiver to bring child to the hospital when child with cough and catarrh develops fast breathing
- Instruct caregiver to increase frequency of feeding for the next 2 weeks after treatment for pneumonia. Children with moderate to severe malnutrition should receive treatment advice according to standard guidelines

Drugs to Avoid in the Management of Pneumonia

Cough syrups containing antihistamines or opioids such as codeine, hydrocodeine, because they add little to the management of pneumonia and may be toxic in some children

Counselling/Health Education For CAP

Health education and guidance play important roles in the management of children with CAP. Counselling should be ongoing from admission to discharge with family regularly updated on progress of management. Information on what the caregiver should observe in the child and report to the health facility should be communicated. The education should include:

1. The fact that CAP is caused by micro-organisms.
2. Environmental factors that predispose to CAP and CAP-related deaths such as indoor air pollution including passive parental smoking, overcrowding, poor ventilation, poor personal and environmental hygiene. Good hand washing practices should be emphasized.
3. Education about the presenting features with emphasis on the need for early recognition of fast breathing.
4. Immunization against the childhood diseases. Vaccines that protect against pneumonia such as Pneumococcal conjugate vaccine (PCV), *Haemophilus influenzae* type B vaccine (Hib), pertussis, and measles vaccines should be communicated.
5. The importance of Exclusive breastfeeding in the first 6 months of life and adequate nutrition should be explained to the caregiver.
6. Opportunities should be given for the caregivers to express his/her fears, so that cultural and religious beliefs that are detrimental to achieving optimal health and development of the child should be discussed.
7. The need to avoid self-medications including the use of cough mixtures

Prevention

There are proven strategies for the prevention of community acquired pneumonia in children. These include:

**Specific Vaccines**
- Conjugate vaccine for *S. pneumoniae*
- Conjugate vaccine for *H. influenzae* type b
- Influenza vaccine

**Other vaccines**
- Measles containing vaccine (including booster doses)
- BCG vaccine
- Pertussis vaccine (now in pentavalent vaccine used nationwide; including booster doses)

**Measures to reduce risk factors**
- Improved housing: improved ventilation, reduce overcrowding and indoor air pollution
- Improved nutrition
- Exclusive breastfeeding for the first 6 months
- Micronutrients supplementation, including vitamin A and zinc
- HIV prevention – prevention of mother-to-child transmission of HIV
Future Research

Paucity or absence of recent data on various aspects of childhood community acquired pneumonia in Nigeria makes it imperative for research strategies directed at filling the knowledge gaps in:

1. Viral contribution to aetiology of CAP
2. Bacterial super-imposition following an initial viral infection
3. Seasonal variations in the contribution of various organisms to childhood community acquired pneumonia
4. Contribution of Mycoplasma pneumoniae to childhood community acquired pneumonia
5. Severity grading of childhood community acquired pneumonia
6. Antibiotic resistance/sensitivity pattern of common organisms causing community acquired pneumonia
7. Appropriate and relevant clinical scoring tool for pneumonia
8. Usefulness of procalcitonin and C-reactive protein in the diagnosis of CAP
9. Surveillance study on the prevalent pneumococcal serotypes in CAP
10. Surveillance study on the prevalence and the role of non-typeable Haemophilus influenzae in CAP

Appendices

Appendix I: Lung Sound Nomenclature

<table>
<thead>
<tr>
<th>Lung Sound Nomenclature Description</th>
<th>Term</th>
</tr>
</thead>
<tbody>
<tr>
<td>Discontinuous</td>
<td></td>
</tr>
<tr>
<td>Fine (high pitched, low amplitude, short duration)</td>
<td>Fine crackles (crepitations/Rhales)</td>
</tr>
<tr>
<td>Coarse (low pitched, high amplitude, long duration)</td>
<td>Coarse crackles (crepitations/Rhales)</td>
</tr>
<tr>
<td>Continuous</td>
<td></td>
</tr>
<tr>
<td>Continuous, musical sound heard during expiration only or during both phases of respiration</td>
<td>Rhonchi</td>
</tr>
</tbody>
</table>

NB: Wheezes and Rhonchi may be heard in severe Pneumonia

Appendix II: Methods of Oxygen Delivery

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References
