ABSTRACT

Bacterial pneumonia is an acute lower respiratory infection related to an inflammation of the pulmonary parenchyma determining a radio-clinical syndrome of lobar or segmental condensation [1].

Frequent reasons for hospitalization in pediatric settings, their distribution is worldwide with a high prevalence in Africa. Streptococcus Pneumoniae (STO.P) or Pneumococcus, first infectious cause of pediatric mortality, is responsible for 15% of deaths before 5 years. In Africa, it is the cause of 21% of deaths, 28% if we include the neonatal period, or nearly a third of deaths. A real public health problem, pneumococcal pneumonia is the subject of numerous guidelines from the World Health Organization (WHO). The emergence of strains of Pneumococcus with reduced sensitivity to penicillin, the difficulty of their biological objectification and the co-detection of several pathogens in the same sample complicate their management [2]. Radiography, an available and inexpensive technique, makes it possible to diagnose parenchymal involvement, obtain an etiological orientation, guide antibiotic therapy, ensure follow-up, and thus avoid complications.

The general objective of this work is to evaluate the role of standard radiography in the management of bacterial pneumopathies in children.

The specific objectives are:

- To list the pathogens involved in the different age groups after the introduction of the pneumococcal vaccine in the Extended Vaccination Program (EVP) of Senegal,
- To describe the radiological syndromes characteristic of these pathogenic agents.

The prognosis of bacterial pneumopathies in children being severe, it is imperative to make an early diagnosis. Unfortunately, non-specific clinical signs, late and random bacteriological results do not allow a good etiological orientation. The fine study of radiological syndromes, confronted with epidemiological and clinical data, has the merit of guiding probabilistic antibiotic therapy and avoiding complications.
Keywords
Radiography, Bacterial pneumonias, Respiratory infections.

Introduction
Bacterial pneumonias are acute lower respiratory infections related to inflammation of the pulmonary parenchyma leading to a radiological syndrome of lobar or segmental condensation [1]. Frequent reasons for hospitalization in pediatric settings, their distribution is worldwide with a high prevalence in Africa. Streptococcus Pneumoniae (STO.P) or Pneumococcus first infectious cause of pediatric mortality is responsible for 15% of deaths before 5 years. In Africa, it is the cause of 21% of deaths, 28% if we include the neonatal period, nearly a third of deaths. A real public health problem, pneumococcal pneumonia is the subject of numerous World Health Organization (WHO) guidelines. The emergence of strains of Pneumococcus with decreased sensitivity to penicillin, the difficulty of their biological objectification and the co-detection of several pathogens in the same sample complicate their management [2]. Radiography, which is available and inexpensive, makes it possible to diagnose parenchymal involvement, obtain etiological orientation, guide antibiotic therapy, ensure follow-up, and thus avoid complications.

The general objective of this work is to assess the role of radiography in the management of bacterial pneumonia in children.

The specific objectives are:
− To list the pathogens involved in the different age groups after the introduction of the pneumococcal vaccine in the EPV of Senegal,
− To describe the radiological syndromes characteristic of these pathogenic agents.

Materials and Methods
A retrospective, descriptive, analytical study, from January 1, 2012, to May 31, 2018, concerned children’s medical records of patients aged 0 to 15 years, hospitalized for bacterial pneumonia at CHNEAR in Dakar, level III national pediatric reference center of the health pyramid of Senegal. We included children who received standard frontal chest X-rays and whose samples (blood culture, pleural fluid, pericardial fluid) made it possible to isolate a germ. We excluded children who were not hospitalized, children followed for chronic pathologies or presenting incomplete files.

The x-rays, carried out in inspiration and in a vertical position, anteroposterior in infants, postero-anterior in children, in a bone-lung room equipped with a digital x-ray table and a wall potter, were read by the same pediatric radiologist.

The parameters studied were epidemiological characteristics (age, sex), clinical (medical-surgical history, site, co-morbidities, signs, complications), biological (blood count, C-reactive protein), bacteriological (isolated germ) and radiological (lung volumes, elementary parenchymal or pleural lesions, mediastinal anomalies). They were confronted to identify specific radiological pictures of the germs. Data entry and analysis were performed on a computer with Epi info software version 7.2.2.6. and the design of the figures by Microsoft Excel 2007. We calculated frequencies, means, standard deviations and compared frequencies using the chi-square test, where p <0.05 was considered statistically significant.

Results

Epidemiological
54 files were retained out of 73. The mean age of the patients was 37.5 months. They were mostly male (61.1%), peri-urban (59.3%) and low socio-economic status (81.5%). 48 children (88.9%) were up to date with their EPV and 21 (38.8%) had received the pneumococcal vaccine.

Clinical
53 children had deteriorated general condition (98.1%), 49 had fever (90.7%) and 5 had clinical anemia (9.3%). Respiratory functional signs were dyspnea in 51 children (94.4%), cough in 42 (77.8%). There were digestive signs including vomiting (15 cases or 27.8%) and diarrhea (9 cases or 16.7%).

The respiratory physical manifestations were distress (50 children or 92.6%), hypoxemia (54%), pleural fluid effusion syndrome (87%), pulmonary condensation syndrome (64.8%), bronchial syndrome (40.7%) and gaseous pleural effusion syndrome (9 children or 16.7%). The physical signs were also digestive with abdominal bloating (4 children or 7.4%), hepatomegaly and oral thrush (1 case each, or 1.9%) and neurological with 1 meningeal syndrome. Among the 19 children who presented a particular ground, 15 were malnourished or 27.8%. Human immunodeficiency virus (HIV) infection, heart disease, sickle cell anemia and low birth weight each accounted for 2 cases (3.70%). Three children had pericardial effusion (5.6%). The other comorbidities were malaria, abscessed collections, meningitis, urinary tract infection and oral thrush.

Biological
The complete blood count showed anemia in 44 children (81.48%), leukocytosis in 49 (90.74%) and leukopenia in 1 (1.85%). The C-reactive protein essayed in 43 patients (79.63%) was positive in 42 (97.67%) and normal in a child (2.3%).

Bacteriological
Of the 54 children, 38 or 70.37% had benefited from a puncture and/or pleural drainage. The pathogen was isolated from pleural fluid 47 times (87.03%), from blood 7 times (0.12%), and from pericardial fluid 3 times (0.05%). Bacteriological examination of the CSF was contributory in 1 case (0.02%). The pathogens isolated were Streptococcus Pneumoniae (STO.P) for 21 children (38.9%), Staphylococcus Aureus (STA.A) for 20 (37%), Gram-negative non-fermenting bacillus (NFGNB) for 4 (7, 4%), Staphylococcus Sp (STA.SP), Streptococcus Sp (STO.SP), Staphylococcus Saprophyticus (STA.SA) and Klebsiella Pneumoniae (KB.P) in equal proportions (2 cases or 3.7%) and Escherichia Coli (E.Coli) (1 case or 1.9%). Unlike pathogens present exclusively in pleural fluid, pneumococcus was found in all pathological fluids. Staphylococcus was found in pleural fluid and blood (Table 1).
Radiological
Lung damage
According to the side
Parenchymal involvement was found on the right in 23 children (42.6%) and on the left in 19 (35.2%). They were bilateral in 12 cases (22.2%).

According to the location
The upper lobes were affected 19 times (25%), the middle lobe 9 times (11.8%) and the lower lobes 48 times (63.1%). The upper lobe-middle lobe association was 7% and the middle lobe-lower lobe 6%. Hemithorax involvement was 59.2% on the right and 40.7% on the left (Table 2).

According to the Type
The following radiological syndromes were noted: alveolar, cavity, interstitial, broncho-alveolar or broncho-alveolo-cavity. The latter two were isolated or associated with pulmonary and digestive hyperventilation in 12 patients (22.22%) (Table 3).

Extra-pulmonary involvement
Pleural
45 children (83.33%) presented with pleural fluid effusion. It was free in 10 cases (15.52%) and encysted in 21 cases (38.89%).

Digestive
Digestive hyperventilation accompanied pulmonary hyperventilation in 12 children, or 22.22%. An ascension of the right diaphragmatic dome was noted in 2 cases. An abdominal ultrasound confirmed liver abscess.

Cardiac
Three children presented with cardiomegaly (7.4%) with a "carafe heart" appearance in favor of pericardial effusion confirmed by ultrasound.

Others
One child had an abscess of the soft parts of the abdomen and right thigh (1.85%).

Correlations
Radio-epidemiological
Radiological lesions and age
Among the 17 children (31.48%) who presented an alveolar syndrome, 5 (29.41%) were aged 0 to 30 months or 15.63% of radiological lesions, 7 (41.18%) from 30 to 72 months or 63.64%

Table 1: Distribution of Pathogens in Samples.

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>NFGNB</th>
<th>E. COLI</th>
<th>KB. P</th>
<th>STO. P</th>
<th>STA. A</th>
<th>STA.SA</th>
<th>STO.SP</th>
<th>STA.SP</th>
<th>Total</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pleural fluid</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>18</td>
<td>16</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>47</td>
<td>87,03</td>
</tr>
<tr>
<td>Blood</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pericardial fluid</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3</td>
<td>0.12</td>
</tr>
<tr>
<td>Cerebrospinal fluid</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0.02</td>
</tr>
<tr>
<td>TOTAL</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>24</td>
<td>21</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>58</td>
<td>87.22</td>
</tr>
</tbody>
</table>

Table 2: Distribution of lung lesions by lobe.

<table>
<thead>
<tr>
<th>Lung</th>
<th>Upper lobe</th>
<th>Middle lobe</th>
<th>Lower lobe</th>
<th>Hemithorax</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>%</td>
<td>Nombre</td>
<td>%</td>
<td>Number</td>
</tr>
<tr>
<td>Right</td>
<td>13</td>
<td>17.1</td>
<td>9</td>
<td>11.8</td>
</tr>
<tr>
<td>Left</td>
<td>6</td>
<td>7.8</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>19</td>
<td>25</td>
<td>9</td>
<td>11.8</td>
</tr>
</tbody>
</table>

Table 3: Distribution of Lung Damage by the Type.

<table>
<thead>
<tr>
<th>Pulmonary radiological syndromes</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alveolar syndrome</td>
<td>17</td>
<td>31.48</td>
</tr>
<tr>
<td>Broncho-alveolar syndrome</td>
<td>12</td>
<td>22.22</td>
</tr>
<tr>
<td>Broncho-alveolar syndrome / hyperventilation</td>
<td>9</td>
<td>16.67</td>
</tr>
<tr>
<td>Cavity syndrome</td>
<td>4</td>
<td>7.41</td>
</tr>
<tr>
<td>Broncho-alveolar-cavity syndrome</td>
<td>8</td>
<td>14.81</td>
</tr>
<tr>
<td>Broncho-alveolar-cavity syndrome / hyperventilation</td>
<td>3</td>
<td>5.56</td>
</tr>
<tr>
<td>Interstitial syndrome</td>
<td>1</td>
<td>1.85</td>
</tr>
<tr>
<td>Total</td>
<td>54</td>
<td>100</td>
</tr>
</tbody>
</table>

Table 4: Distribution of Pleural Damage.

<table>
<thead>
<tr>
<th>Pleural radiological syndrome</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Free fluid effusion syndrome</td>
<td>10</td>
<td>15.52</td>
</tr>
<tr>
<td>Encysted fluid effusion syndrome</td>
<td>21</td>
<td>38.89</td>
</tr>
<tr>
<td>Mixed effusion syndrome</td>
<td>12</td>
<td>22.22</td>
</tr>
<tr>
<td>Gas effusion syndrome</td>
<td>9</td>
<td>16.67</td>
</tr>
<tr>
<td>None</td>
<td>2</td>
<td>3.7</td>
</tr>
<tr>
<td>Total</td>
<td>54</td>
<td>100</td>
</tr>
</tbody>
</table>
of the lesions, 3 (17.65%) were older children or 42.86% of the lesions and 2 (11.76%) were adolescents or 50% of the lesions. Of the 21 patients (38.89%) who presented with broncho-alveolar syndrome, 15 or 71.43% concerned the newborn-infant group. Broncho-alveolar syndrome was isolated in 7 children (58.83%) or 21.88% of the radiological lesions. It was associated with pulmonary hyperventilation in 8 children (88.9%) or 25% of lesions.

Among the 11 patients who presented with broncho-alveolar-cavity syndrome (20.37%), 10 or 90.9% were aged 0 to 30 months. In 7 cases, it was isolated (87.5%) and corresponded to 21.88% of the radiological lesions. In 3 patients, it was associated with hyperventilation, broncho-alveolar-cavity syndrome was caused by STA.A in all cases. The isolated cavity syndrome was due to STA.A and NFBGN (50% each). The only case of interstitial syndrome involved an adolescent (Table 5).

Radiological lesions and vaccination.

Among the children with parenchymal damage, 2 had received the pneumococcal vaccine and 2 were not vaccinated. Among the children with pleural involvement, 4 were vaccinated and 13 were unvaccinated. Encysted pleural fluid effusion syndrome was observed in 2 vaccinated (50%) and 9 unvaccinated (69%) patients. Fluid-free pleural effusion was present in 1 vaccinated (25%) and 3 unvaccinated (23.07%) children. Mixed fluid pleural effusion syndrome was noted in 1 vaccinated (25%) and 1 unvaccinated (7.69%) child.

Radio-clinics

In this study, classic respiratory signs were noted. The digestive signs (57.8%) reported were close to the percentage of lower lobe radiological locations (63.1%). The difference could be explained by the number of children who could not report abdominal pain.

Radio-bacteriological

Pulmonary radiological damage and germs

The alveolar syndrome was due to STO.P (76.47%), BGNF (11.76%), E.Coli (5.88%). STA.A (25%) and STO.P (50%) were responsible for an isolated broncho-alveolar syndrome. The broncho-alveolar syndrome associated with hyperventilation was due to STA.A (55.56%), STA.SA (11.11%), STA.SP (11.11%) and STO.P (22.22 %). The isolated broncho-alveolar cavity syndrome was due to STA.A (75%) and STA.SA (12.5%). Associated with hyperventilation, broncho-alveolar-cavity syndrome was caused by STA.A in all cases. The isolated cavity syndrome was due to STA.A and NFBGN (50% each). The only case of interstitial syndrome was due to STO.SP (Table 6).

Pleural Radiological Damage and Germs

Fluid-free pleural effusion was caused by STO.P (40%) and STA.A (20%). Encysted pleural fluid effusion was caused by STO.P (52.38%) and STA.A (33.3%). Mixed pleural effusion was related to STA.A (50%), STO.P and NFGNB (16.67% each), STA.SA and STA.SP (8.33% each). The gaseous pleural effusion in 2 patients was due to STA.A in one and NFGNB in the other (Table 7).

Epidemi-bacteriological

Age and germs

STO.P had reached 7 newborns and infants or 21.88%, 8 small

Table 5: Distribution of Pulmonary Radiological Lesions According To Age.

<table>
<thead>
<tr>
<th>Alveolar syndrom</th>
<th>0-30 months</th>
<th>30-72 months</th>
<th>72-108 months</th>
<th>108-180 months</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>5</td>
<td>15,63%</td>
<td>29,41%</td>
<td>17,65%</td>
<td>47,15%</td>
</tr>
<tr>
<td>Broncho-alveolar syndrom</td>
<td>7</td>
<td>58,33%</td>
<td>21,88%</td>
<td>25%</td>
<td>11,76%</td>
</tr>
<tr>
<td>Broncho-alveolar Syndrom / hyperventilation</td>
<td>8</td>
<td>88,99%</td>
<td>25%</td>
<td>0%</td>
<td>11,11%</td>
</tr>
<tr>
<td>Broncho-alveolar-cavity syndrom</td>
<td>7</td>
<td>87,5%</td>
<td>21,88%</td>
<td>0%</td>
<td>14,29%</td>
</tr>
<tr>
<td>Broncho-alveolar-cavity syndrome / hyperventilation</td>
<td>3</td>
<td>100%</td>
<td>9,38%</td>
<td>0%</td>
<td>12,5%</td>
</tr>
<tr>
<td>Cavity syndrom</td>
<td>2</td>
<td>50%</td>
<td>25%</td>
<td>0%</td>
<td>12,5%</td>
</tr>
<tr>
<td>Interstitial syndrom</td>
<td>0</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Total</td>
<td>32</td>
<td>59,26%</td>
<td>20,37%</td>
<td>19,96%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Percentage of lesions in the age group

Percentage of lesions in radiological involvement
Table 6: Distribution of pathogens according to lung lesions.

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Alveolar syndrome</th>
<th>Broncho-alveolar syndrome</th>
<th>Broncho-alveolar syndrome Hyperventilations</th>
<th>Broncho-alveolo-cavity syndrome</th>
<th>Cavity syndrome</th>
<th>Interstitial syndrome</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>NFGNB</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>E. COLI</td>
<td>11.76%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>100%</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td>KB. P</td>
<td>5.88%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>STO.P</td>
<td>0%</td>
<td>0%</td>
<td>16.7%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>STA.A</td>
<td>13</td>
<td>6</td>
<td>65%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>STASA</td>
<td>1</td>
<td>3</td>
<td>5%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>STO.SP</td>
<td>5</td>
<td>2</td>
<td>27%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>STA.SP</td>
<td>0%</td>
<td>0%</td>
<td>75%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>17</td>
<td>12</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>54</td>
</tr>
</tbody>
</table>

Table 7: Distribution of Pathogens According to Pleural Damage.

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Gas effusion</th>
<th>Encysted fluid effusion</th>
<th>Free fluid effusion</th>
<th>Mixed effusion</th>
<th>None</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>NFGNB</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>E. COLI</td>
<td>50%</td>
<td>4.76%</td>
<td>0%</td>
<td>16.6%</td>
<td>0%</td>
<td>74.1%</td>
</tr>
<tr>
<td>KB. P</td>
<td>0%</td>
<td>4.76%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>1.85%</td>
</tr>
<tr>
<td>STO.P</td>
<td>0%</td>
<td>4.76%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>3.7%</td>
</tr>
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<td>STA.A</td>
<td>0%</td>
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<td>0%</td>
<td>16.67%</td>
<td>0%</td>
<td>38.9%</td>
</tr>
<tr>
<td>STASA</td>
<td>0%</td>
<td>33.33%</td>
<td>0%</td>
<td>16.67%</td>
<td>0%</td>
<td>37%</td>
</tr>
<tr>
<td>STO.SP</td>
<td>0%</td>
<td>4.0%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>3.7%</td>
</tr>
<tr>
<td>STA.SP</td>
<td>0%</td>
<td>20%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>3.7%</td>
</tr>
<tr>
<td>Total</td>
<td>2%</td>
<td>41.5%</td>
<td>0%</td>
<td>100%</td>
<td>0%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Comments
Radiological data
Lung damage

Field and comorbidities and germs
9 malnourished children or 60%, presented with Staphylococcus infection, including 7 with STA.A (46.67%) and 2 with STA.SA (13.33%). 4 children (26.66%) had an infection with STO.P. 2 children were infected with NFGNB and KB. P (6.67% each). The 2 SS sickle cell patients or 50%, presented with STO.P. 2 HIV children were infected, one with STA.A and the other with STO.P. The 3 cases of pericarditis and meningitis were due to STO.P. Both abscesses, hepatic and soft tissue were due to STA.A.

Distribution of lesions by side
The predominance of the lesions on the right (42.6% against 35.2% on the left) has been reported by several authors [3-5]. It is explained by the anatomical arrangement of the right mainstem
bronchus, which continues in the direction of the trachea and carries pathogens from the airways [6,7]. The left side is less frequently affected because the bronchus is located transversely [8].

**Distribution of Lesions According To Their Location**
The preferred site of lesions in the lower lobes can be explained by the flow-volume relationship. The lungs of children empties more easily than that of adults and tend to atelectasis [9]. In addition, there is physiological hypoxemia with less ventilation at the bases [10].

**Distribution of Lesions According To Their Nature**
In our series, the alveolar syndrome, radiological translation of the filling of the pulmonary alveoli, is noted in nearly 91% of cases, isolated or not. Multiple lobe involvement or even a hemithorax is common (58.6% on the right and 40.7% on the left). Alveolar syndrome is the main lesion reported by Thiongane in Senegal [3], N’goran in Ivory Coast [11] and Tinsa in Tunisia [12]. As in our series, N’goran reports 58.6% plurilobar disease.

Bronchial syndrome due to alteration of the "muco-ciliated escalator" preventing the drainage of secretions and cavity syndrome by destruction of the pulmonary parenchyma are reported in equivalent proportions by N’goran [11].

Interstitial syndrome most often due to atypical bacteria [13] was not reported and the only case noted in our study was due to Streptococcus Sp.

**Pulmonary Hyperventilation**
Lung hyperventilation, noted in 22% of cases, associated with digestive hyperventilation, was not reported in any study. The patho-physiological explanation may be the heterogeneity of pulmonary aeration leading to alternating atelectasis and "trapping" [14]. In infections with STA.A and NFGNB, it is probably related to their toxin and necrotizing power.

**Pleural involvement**
Due to the anatomical relationship between the lung and the pleura, pleural complications are observed in 20 to 60% of bacterial pneumonias [15]. In our series, pleural effusion syndrome was associated with parenchymal syndrome in 83.3% of cases. N’goran and Tinsa exclusively report a syndrome of free pleural effusion indicating early management. It was less important in the study by S. Oulai, which was prospective and concerned newborns with an average age of 4 days, also reducing the time taken for treatment [16].

In our study, the high rate of encysted pleural fluid effusion syndrome reflects advanced forms related to a long consultation period or inappropriate antibiotic therapy. This is evidenced by pleural drainage performed in two-thirds. The rate of mixed pleural effusion in our series is also higher compared to Oulai’s results. It may be related to the rupture of bubbles or to the introduction of air during drainage. Gas pleural effusion syndrome was less common in the N’goran study.

**Correlations**

- **Radio-epidemiological**
- **Radiological damage and age**

According to Martinot [17], the average age of onset of pleuropulmonary infections in children is 9 months. In our series, lung lesions predominated in the 0–30-month age group with a single newborn and the mean age was 37.5 months. In an Ivorian study on infants, 41.2% were aged 13 to 18 months [11]. Indeed, if newborns are protected by maternal antibodies, the preferential occurrence of bacterial pneumonia in infants is certainly related to a still immature immune system. The prevalence can exceed 80% as in the Ilham and Zeyneb series in Algeria [18].

Regarding the types of lesions, isolated alveolar syndrome predominated in children and adolescents. Cavity, bronchoalveolar and broncho-alveolar-cavity syndromes, alone or associated with pulmonary hyperventilation, predominated in infants (71.43% and 90.9% respectively). The importance of the

**Table 8: Distribution of germs according to age.**

<table>
<thead>
<tr>
<th>Age Group</th>
<th>NFGNB</th>
<th>E. COLI</th>
<th>KB. P</th>
<th>STO.P</th>
<th>STA.A</th>
<th>STA.SA</th>
<th>STO.SP</th>
<th>STA.SP</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-30 months</td>
<td>2</td>
<td>6,25%</td>
<td>1</td>
<td>3,13%</td>
<td>7</td>
<td>21,88%</td>
<td>17</td>
<td>53,13%</td>
<td>1</td>
</tr>
<tr>
<td>30-72 months</td>
<td>1</td>
<td>9,09%</td>
<td>0</td>
<td>0%</td>
<td>8</td>
<td>72,73%</td>
<td>1</td>
<td>9,09%</td>
<td>0</td>
</tr>
<tr>
<td>72-108 months</td>
<td>0</td>
<td>0%</td>
<td>0</td>
<td>0%</td>
<td>4</td>
<td>57,14%</td>
<td>2</td>
<td>28,57%</td>
<td>0</td>
</tr>
<tr>
<td>108-180 months</td>
<td>1</td>
<td>25%</td>
<td>0</td>
<td>0%</td>
<td>2</td>
<td>50%</td>
<td>0</td>
<td>0%</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>4</td>
<td>7,41%</td>
<td>1</td>
<td>1,85%</td>
<td>21</td>
<td>38,89%</td>
<td>20</td>
<td>37,04%</td>
<td>2</td>
</tr>
</tbody>
</table>

**Staphylococcus Auréus**
**Streptococcus Pneumoniae**
**Non-Frementing Gram Negative Bacillus**
**Klebsiella Pneumoniae**
**Echzrichia Coli**
**Staphylococcus Saprophyticus**
**Staphylococcus Sp**
**Streptococcus Sp**

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bronchial component in toddlers is explained by certain anatomo-physiological peculiarities such as the increase in resistance of the peripheral airways promoting inflammation, the easy occurrence of mucous plugs with edema and occlusion of the peripheral bronchi; less developed collateral ventilation with lack of support and frequency of peripheral ventilatory disorders; the presence of an epithelium rich in mucous glands promoting congestion phenomena; soft and depressible airways because they are poor in cartilage, forced expiration, hypervascularization and edema accentuating the decrease in caliber and finally a short Eustachian tube.

In our study, there is a relationship, but statistically of little significance, between the distribution of lung lesions and age with chi-square equal to 0.0217 (<0.05). This statistical link between pulmonary radiological syndromes and age group was not mentioned in the above studies.

**Radiological Damage and Sex**

As in our study, acute pneumonia in children most often affects boys for many authors, [12,19]. In Ivory Coast [11], the sex ratio was slightly in favor of boys as well as in Togo [20] where the sex ratio was 1.3. N’goran’s result in Ivory Coast is like ours. The predominance of infectious pathologies in boys is commonly accepted.

**Radiological damage and vaccination**

Until the early 1990s, Staphylococcus Aureus and Haemophilus Influenzae were the most common pathogenic species [21]. In Senegal, the incidence of Haemophilus Influenza b (Hib) infections fell sharply one year after the introduction of the Hib conjugate vaccine into the EPV in July 2005 [22-24]. Thus, in our study no case was found. Unlike certain series [3,4] which seem to relegate Staphylococcus Aureus to the background, pleuro-pulmonary staphylococcal disease remains a concern in Senegal because it is responsible in our series for 37% of infections.

Despite the introduction in October 2013 of the anti-pneumococcal vaccine (Prenavar 13) in the EPV, making it possible to cover the 10 serotypes involved in most invasive pneumococcal infections and representing strains particularly resistant to antibiotics, (Pneumococcus with reduced sensitivity) STO.P leads our series with 39%. Among our vaccinated patients, 66.7% presented severe forms with a table of pleuropneumonia against 86.7% for unvaccinated patients. The high percentage of complicated pneumonia poses the problem of vaccination coverage of certain serotypes in vaccinated children.

**Radiological Damage and Signs**

As pneumonia may appear in children with atypical symptoms such as abdominal pain, especially in children under 3 years of age, food intake difficulties sometimes associated with vomiting. Any febrile abdominal pain in children without a clinical call point for a digestive aetiology should seek pneumonia [25]. In our series, digestive signs represent 57.3% and are close to the results noted for the lower lobe locations (63.1%). Abdominal pain has not been reported due to the young age of the patients. The statistical link between digestive signs and lower lobe locations could not be proven.

**Radiological Damage and Germs**

**Lung Damage and Germs**

For some authors, the contribution of radiography in the etiologic diagnosis of pneumonia is questionable. The appearance of the X-ray cannot suggest the causative organism [26]. On the other hand, it is accepted that alveolar syndrome is characteristic of Pneumococcus [27,28]. It is also classic to suggest STA.A bronchopneumonia in the presence of confluent and extensive alveolar opacities, excavations or pneumatoceles.

In our study, the isolated alveolar syndrome was mainly caused by STO.P. Alveolar syndrome associated with bronchial syndrome was caused by STO.P in half of the cases and pulmonary hyperventilation in more than half of the cases. Broncho-alveolar syndrome with pulmonary hyperventilation was also due to STA. SA, STA.SP and STO.SP. Broncho-alveolar cavity syndrome was in two thirds of cases due to STA.A and in one third to STA. SA and STO.SP. Associated with hyperventilation, it was due to STA.A. The isolated cavity syndrome was caused equally by STA.A and NFBGN. Thus, the unexcavated lesions were mainly due to STO.P, while the excavated lesions were more related to STA.A and NFGNB. NFGNB seems to possess a necrotizing power as important as that of STA.A.

In our series, there was a statistically significant association between pulmonary parenchymal involvement and the pathogens STO.P and STA.A with chi-square equal to 0.0002 (<0.05). Apart from cavity syndrome, the radiological lesions caused by STA.SA and STA.SP were like those of STA.A.

Even if there was no statistical link between the distribution of germs and the presence of pulmonary and digestive hyperventilation (chi-square equal to 0.19 therefore> 0.05), the hyperventilation appears to us to be a sign of great diagnostic value because it was present for all Staphylococci and was constant whenever STA.A was involved.

**Pleural involvement and germs**

According to Martinot [17], the existence of a liquid or gaseous pleural effusion or bullous images constitute the keys to the diagnosis of pleuro-pulmonary staphylococcal disease in infants and makes it possible to start immediately, a first-line antibiotic therapy for staphylococci methicilino-resistant. For Sardet [21] and Gaudelus [22], pneumococcus is the germ most often responsible for bacterial pleuro-pneumonia in children.

In our series, pleural effusion syndrome, whether free or encysted, was observed mainly with STO.P and STA.A. The gas pleural effusion syndrome was due to STA.A and NFGNB in identical proportions. Mixed pleural effusion syndrome was linked to the presence of Staphylococci and NFGNB. The mixed pleural effusion syndrome in STO.P infections was certainly iatrogenic,
the chest tube allowing the accidental introduction of air.

If no statistical link was demonstrated between the pleural radiological signs and the pathogenic agents (chi-square equal to 0.571 therefore >>> 0.05), the comparative analysis of the images of patients infected with STO.P and STA.A concluded that apart from drainage, gas pleural effusion syndrome and mixed pleural effusion syndrome were characteristic of STA.A and NFBGN.

Epidemiological and Bacteriological Correlations
Age and germs
Several African studies have objected to a particular distribution of pathogens according to age [3,11,29].

In the study by S. Oulai, which only involved infants, STA.A represented 35% of the germs found against 20% for STO.P [16].

In F. Tinsa's study, which concerned children aged 9 to 132 months, whose most representative age groups were those of 1-3 years, and 3-5 years, STO.P constituted 41% of the germs isolated, while no case of STA.A has been objectified [12].

In our study, STA.A was of interest to newborns and infants. STO.P was predominant in children (72.73% of small children and 57.14% of older children), with no statistical link between age and the pathogen (chi-square = 0.189 so> 0.05).

Terrain, comorbidities, and pathogens
In infants who are not exclusively breastfed, malnutrition promotes the development of infections. Conditions such as symptomatic HIV infection or measles, too [2]. A study by Sow in Senegal reported 79.55% of lung infections in malnourished children [30]. For Orega in Côte d'Ivoire, lung infection is the main infection encountered in infected malnourished children with 65.45% of cases [31].

In our study, 60% of malnourished patients presented with Staphylococcus infection, 88.9% of which were infants aged 7 to 14 months. Only one patient was 84 months old (11.1%). Of the 26.66% of malnourished who presented with STO.P infection, 75% were infants, and only one child was 90 months old (25%). Malnutrition questions the distribution of pathogens. It should be sought systematically in the event of staphylococcal disease in a child or pneumococcal infection in an infant. As the number of patients with sickle cell disease or HIV infection is too low, we cannot confirm their influence on this distribution. Co-morbidities are consequences of diagnostic delay and treatment failure, antibiotic therapy being probabilistic. According to there is a link between severe IRAB and low social status [32].

In our series, 81.5% of the patients came from a disadvantaged background. Pericarditis and meningitis were due to STO.P. The abscessed collections were due to STA.A.

The relationship between the pathogen, the site, the comorbidities and the age were not specified in the above series.

Conclusion
As the prognosis of bacterial pneumonia in children can be very severe, it is imperative to make a diagnosis early. Unfortunately, not very specific clinical signs, late and random bacteriological results do not allow a good etiological orientation. The detailed study of radiological syndromes, compared with epidemiological and clinical data, has the merit of orienting probabilistic antibiotic therapy and avoiding complications.

References
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