Aspiration pneumonia in an infant with neurological sequelae – case report

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Abstract

Aspiration pneumonia is a frequent cause of morbidity and mortality in children with neurological deficits. We present the case of a 4-month-old infant from the Foster Care Center, with severe psychomotor retardation, blindness, and associated cardiac malformation, who was admitted to the Pediatrics Clinic of the Emergency County Hospital of Craiova, Romania, presenting aspiration pneumonia and moderate respiratory insufficiency. Under sustained, early instituted treatment, the evolution was towards death. The chest radiography and histopathological examination of the pulmonary tissue confirmed the diagnosis. The neurological impairment was not only a favoring factor for aspiration, through the deglutition disorders, but it was also an aggravating one, through the bacterial colonization of the lungs.

Keywords: aspiration pneumonia, neurological impairment, mortality increase.

Introduction

The incidence of aspiration syndromes associated to anatomical or neurological disorders is unknown. Aspiration pneumonia represents a frequent cause of morbidity and mortality in children with neurological deficits [1–3]. Infants and children, who presented pulmonary impairment secondary to prematurity or respiratory distress syndrome, are more vulnerable than those with healthy, unaffected lungs [4]. Neurological impairment secondary to neonatal hypoxia or the presence of congenital cardiac malformations constitute predisposing factors for pulmonary aspiration [5].

Although, in the last years, progress has been made in understanding the pathogenic mechanisms that are at the basis of dysphagia and the protection reflexes of the airways, and new diagnostic techniques have been introduced, many children are diagnosed and treated for aspiration only after the occurrence of pulmonary impairment [6, 7].

The aim of this paper is to present a case of aspiration pneumonia that affected an institutionalized child with malnutrition, neurological deficit and congenital heart malformation, who was diagnosed and treated in our institution. We discuss the case presentation, clinical, radiological and especially the histological diagnosis, and also the management for this condition.

Case report

The authors present the case of a 4-month-old male infant, who was admitted to the Pediatrics Clinic of the Emergency County Hospital of Craiova, Romania, for inefficient frequent spasmodic cough, cyanosis and respiratory difficulties with an onset of a few hours after feeding. The child came from a teenage mother (15-year-old), he was born prematurely, naturally delivered, and was abandoned in the maternity ward.

We have carried out the paraclinical investigations (complete blood count, glycemia, urea, creatinine, transaminases, acid–base balance, serum electrolytes, serum iron), cultures, heart and chest radiography, heart ultrasound, pneumology and phthisiology examination, ophthalmology and neurological examination, and we have instituted the appropriate therapy.

After death occurred, the material sampled from the lungs was subjected to a histopathological examination through the traditional paraffin inclusion method, followed by Hematoxylin and Eosin (HE) staining, after fixation with 10% formalin.

The patient named T.I.M. presented, upon admission, inefficient frequent spasmodic cough, cyanosis of the peri-oral region and extremities, signs of respiratory insufficiency (grunts, expiratory dyspnea, tachypnea, intercostal and subcostal retraction). The onset of the symptoms had been 5–6 hours before, with frequent spasmodic cough and difficult feeding.

The child came from a teenage mother. The birth began spontaneously at 35 weeks of gestation, with pelvic presentation, and prolonged labor that caused perinatal hypoxia. At birth, the child’s weight was 2150 g, the height was 46 cm, the head circumference was 30 cm, and the Apgar score at one minute and at five minutes was 5. The prolonged perinatal hypoxia caused right lung atelectasis and intraventricular hemorrhage; in the end, it led to significant neurological and motor sequelae (microcephaly, blindness, generalized muscle hypotonia, mental retardation).

Physical examination on admission: moderate general condition, deficient nutritional status, pale skin with diminished elasticity, fever 38.4°C, cyanosis of the peri-
oronasal region and extremities, inefficient frequent spasmodic cough, tachypnea with intercostal retraction, the pulmonary auscultation reveals diminished vesicular murmur in the right hemithorax, rounded abdomen, vomiting, bilateral cryptorchidism, microcephaly, blindness, generalized muscle hypotonia, sleepiness.

The patient was subjected to oxygen therapy, intravenous perfusion for water, electrolyte and acid–base balance, antibiotic (Sulcef), bronchodilator and symptomatic treatment.

The first investigations carried out revealed alterations of the complete blood count (anemia, leukocytosis, and thrombocytopenia), of the serum electrolytes, and hypocalcemia (Table 1).

Table 1 – Paraclinical data

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Initial value</th>
<th>Evolution value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin [g/dL]</td>
<td>10.4</td>
<td>5.2</td>
</tr>
<tr>
<td>Platelets [No./mm$^3$]</td>
<td>125 000</td>
<td>62 000</td>
</tr>
<tr>
<td>Leukocytes [No./mm$^3$]</td>
<td>17 600</td>
<td>27 000</td>
</tr>
<tr>
<td>Serum iron [μg/dL]</td>
<td>25</td>
<td>–</td>
</tr>
<tr>
<td>Na$^+$ [mmol/L]</td>
<td>121</td>
<td>130</td>
</tr>
<tr>
<td>K$^+$ [mmol/L]</td>
<td>5.4</td>
<td>3.2</td>
</tr>
<tr>
<td>Blood calcium [mg/dL]</td>
<td>7.6</td>
<td>–</td>
</tr>
<tr>
<td>Urea [mg/dL]</td>
<td>12</td>
<td>55</td>
</tr>
<tr>
<td>Creatinine [mg/dL]</td>
<td>0.29</td>
<td>1.8</td>
</tr>
<tr>
<td>AST [IU]</td>
<td>28</td>
<td>88</td>
</tr>
<tr>
<td>ALT [IU]</td>
<td>14</td>
<td>76</td>
</tr>
<tr>
<td>TB [mg/dL]</td>
<td>0.17</td>
<td>1</td>
</tr>
</tbody>
</table>

AST: Aspartate transaminase; ALT: Alanine transaminase; TB: Total bilirubin.

The chest X-ray revealed the presence of a homogenous opacity in the middle region of the right lung field and the increase in size of the cardiac silhouette (Figure 1).

The heart ultrasound revealed the presence of an ostium secundum (inter)atrial septal defect and the persistence of the arterial duct.

The pneumology, phthisiology and pediatric examination raised the suspicion of aspiration pneumonia – paramedian and right superior lobe pneumonia with an atelectatic component.

The ophthalmology examination revealed bilateral blindness.

During the hospitalization period, the general conditions deteriorated, fever and the respiratory difficulties persisted, the water, electrolyte and acid–base imbalances were maintained, and there was also registered severe anemia, thrombocytopenia and signs of multi-organ dysfunction. The culture collected from the tracheal and bronchial secretion aspirated revealed the presence of *Klebsiella pneumoniae*.

We continued with the water, electrolyte and acid–base rebalancing, and we instituted repeated transfusions of isogroup isoRh erythrocyte and platelet mass, Aminoven, antibiotic treatment (Ccefot, Zyvoxid, Medaxone). The general conditions of the patient continued to gradually deteriorate, with limited response to the administered therapy; on the 7th day since admission, the patient died.

The histopathological analysis of the lungs indicated massive interstitial stasis, with blood extravasations and intra-alveolar macrophages, as well as areas of edematous alveolitis (Figures 2–4).

We could also observe emphysematous areas, with breaks in the alveolar septa and the appearance of air spaces of variable sizes, as well as the presence of areas of alveolar collapse. In the bronchioles, we discovered focal ulcerations, epithelial scaling and peribronchial focal lymphocyte accumulations (Figure 5).

In the liver, we discovered sinusoid, centrilobular and portal stasis, as well as steatosis loading of the periportal hepatocytes (Figure 6). In the brain, we could observe meningeal and intraparenchymal stasis, cerebral edema and blood extravasations (Figure 7).
Aspiration pneumonia in an institutionalized child who presented a number of risk factors: prematurity, malnutrition, neurological deficit, congenital heart malformation; each of these factors increased the risk of morbidity and mortality.

Aspiration pneumonia is the consequence of food and secretions from the oral cavity, the upper respiratory tract or the stomach making their way into the lungs, and it manifests when the reflex defense mechanisms of the airways disappear, especially when dysphagia is also present [1]. The result of aspiration is the colonization of the lungs by the bacteria present in the upper respiratory tract and in the oral cavity, which leads to atelectasis infiltrates, abscess, empyema, sepsis, shock and, in severe forms, death [8, 9].

Studies have shown that, from a bacteriological point of view, just as in the case of adults, anaerobic bacteria are more frequently involved in the production of aspiration pneumonia, especially Gram-positive cocci (Peptostreptococcus), fusobacteria and bacteroides fragilis. When aspiration occurs in institutionalized children, the nosocomial germs are usually aerobic or, at times, aerobic ones (alpha-hemolytic streptococcus, Escherichia coli, K. pneumoniae, etc.) [10, 11].

There is no standard diagnosis test for most types of aspiration; therefore, the differential diagnosis in children remains a challenge. The diagnosis for aspiration is clinical and anamnestic, and certain diagnosis-supporting evaluations are added to it [complete blood count, heart and chest radiography, bacterial cultures, CT (computerized tomography) scans, endotracheal aspirate]. The chest radiography is an indicator for the presence of pulmonary disease, with a characteristic distribution of lung aspiration, as well as for the progression or resolution of the process in time. Radiological alterations are present in all children with foreign body aspiration [12]. In the studied case, the diagnosis was suggested by the patient’s medical history (sudden onset in an institutionalized child with neurological deficits and secondary malnutrition) and the physical examination, and it was supported by the paraclinical data, the characteristic pleural and pulmonary radiography and the culture collected from the tracheal and bronchial secretions aspirate.

In a study conducted in Pakistan on 107 children with pulmonary aspiration, neurological disorders were present in 29% of them, 5.6% presented asphyxia at birth and 10% were premature. The most frequent causes of pulmonary aspiration were caused by milk (31.8%) and oral secretions (19.6%); these children were also the ones who needed a longer hospital stay and recorded a higher percentage of mortality [13]. Pulmonary aspiration was the most frequent cause of recurrent/persistent pneumonia in children (17.7%), in a study conducted in Egypt [14].

Eating difficulties and malnutrition are constantly...
present in children with disabilities because of the presence of chewing and swallowing dysfunctions, spasticity, anorexia, vomiting and recurrent infections. Malnutrition causes, in its turn, the alteration of growth and damages to all organs and systems, especially the neurological and immune ones. Therefore, a multidisciplinary team (neurologist, gastroenterologist, and dietician) is needed to determine the presence of eating disorders and motor and neurological deficits, and to constantly reevaluate the feeding [15, 16]. Eating disorders in children with disabilities are associated to an increase in morbidity and mortality secondary to gastroesophageal reflux, dumping syndrome or aspiration [16]. Studies show that approximately 37–40% of children who present eating disorders are premature [17, 18], while the prevalence of eating disorders in children with neurological damage is estimated between 33 and 80% [17, 19].

Patients with massive aspiration have a 25% mortality rate [20]. In a study conducted by Kohda et al. (1994), 2/3 of the patients with neurological disorders present repeated pulmonary aspirations in their history [21], while in Sheikh et al. study approximately 12% of the 112 neurologically normal children with chronic respiratory symptoms presented swallowing dysfunctions and chronic silent aspiration, with no evidence of gastroesophageal reflux [22].

From a histopathological point of view, the most suggestive aspect for aspiration pneumonia is the presence in the lung of multinucleated giant cells, or even the presence of foreign body granulomas or lymphocyte accumulations [23]. At the same time, acute inflammatory aspects can be present both in the alveoli and the bronchioles [24]. This was also revealed in our case, as the histopathological analysis confirmed the suspicion of pulmonary aspiration raised by the patient’s medical history, by the clinical examination and by the chest radiography.

Conclusions

In the case of our patient, prematurity, pelvic presentation and prolonged labor caused perinatal hypoxia, which led to neurological sequelae. Pulmonary aspirations occurred on the background of severe malnutrition secondary to eating disorders characteristic for children with neurological disorders. It is necessary to carefully follow, detect and appropriately treat, as the case may be, the nutritional problems, considering their impact on growth, quality of life and increase in the risk of mortality, especially in very small children (less than one year old). Aspiration pneumonia represents a frequent cause of morbidity and mortality in children who present neurological deficits. The result of aspiration is the colonization of the lungs by bacteria, which leads to atelectasis infiltrates, abscess, sepsis, shock and, in severe forms, death.

Conflict of interests

The authors declare that they have no conflict of interests.

References


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