Introduction

*Kp* community-acquired pneumonia is rarely encountered. Its prevalence is higher in Asia, where a higher strain virulence was demonstrated [1,2]. It’s a pyo-pneumothorax, which usually occurs on debilitated or immuno-compromised patients. It is a severe disease associated with high mortality. Here, we report the observation of a *Kp* community-acquired pneumonia occurring in a genetic disorder context.

Observation

L.L., 17-year-old, is hospitalized in the ICU for an acute febrile respiratory distress, ongoing for the past 24 hours, which appeared in a generalized tonic-clonic convulsion post-critical phase. As part of the medical history, there are some genetic defects, including a mosaic Trisomy 13 and duplication of the Xq28 region involving the MECP2 gene. As a result, there is a dysmorphic syndrome and a psychomotor retardation since birth, and febrile seizures since the age of 4. His last stay in hospital dates back to 4 years for a bilateral tonsillectomy. Furthermore, the oral exam found quite regular antibiotic consumption on medical prescription, for recurrent infections, without hospitalization.

The initial clinical examination found a superficial polypnea of 35 cycles per minute, an oxygen saturation of 89% in ambient air, wheezes in both lungs, tachycardia of 160 beats per minute, a blood pressure of 102/55 mm hg blood. A chest x-ray shows an opacity in the right lower lobe. Blood gas is not available. The biological results shows a leukocytosis of 22,500/mm3 with a polynuclear neutrophil predominance, a C reactive protein positive at 501 mg/l, a procalcitonin of 23.3 ng/ml. The immediate evolution is marked by an opacity extended to all the right lung. This observation requires a transfer in ICU where the support consisted in the realization of non-invasive ventilation (NIV) sessions, left lateral decubitus position and of probabilistic antibiotic therapy (amoxicillin-clavulanic acid and ciprofloxacin). A few hours after, there is a worsening of her respiratory status, motivating the realization of an orotracheal intubation with mechanical ventilation. Thoracic CT scan is in favour of a broncho-pleuropneumonia (Figure 1). A bronchoscopy for airway clearance and bronchoalveolar lavage (BAL) is performed. BAL bacteriological analysis helped identify a *Klebsiella pneumoniae*, confirmed by two positive blood cultures. With regard to the blood culture: 10 microliters of whole blood collected by venipuncture and stocked in 2 blood vials (one aerobic bottle and another one anaerobic) then incubated at + 37°C in the PLC BD BACTEC 9240 System of Becton Dickinson. After positivity, a direct examination by Gram stain was directed, then isolation and culture by transplanting on agar medium (example: BCP or TSH) with incubation for 24 hours at + 37°C. The identification of *Klebsiella pneumoniae* is made by the characters morphological and biochemical (API 20E bioMérieux, Marcy l’Etoile Gallery). Antibiotic susceptibility tests were performed on Mueller-Hinton (MH) medium at 37°C with an inoculum of 0.5 McFarland in the agar diffusion technique. Result interpretations were made after 24 hours of incubation in accordance with the sensitivity committee recommendations of the French society of Microbiology-EUCAST 2015. The presence

**Figure 1: Lung image at admission.**
of an ESBL was confirmed by the synergy method between a disc of cephalosporins (cefotaxime-ceftazidime-cefeplime) and a disc of amoxicillin + clavulanic acid remote of 3cm. This germ was resistant to Amoxicillin, Amoxicillin-Clavulanic acid, Ticarcillin, Nalidixic acid, and Cephalosporins except for Cephamicins (Cefoxitin) and Carbapenems (Imipenem). Amikacin and piperacillin-tazobactam worked with respective inhibitory minimal concentrations (IMC) of 0.5 and 1ug/mL. Antibiotherapy treatment lasted for 15 days. Sensitivity results therefore lead to the initiation of an antibiotherapy associating piperacillin-tazobactam (12 g - 1, 5 g/day) and Amikacin (15 mg/Kg/day).

The patient’s clinical condition improvement allowed his extubation after 9 days of hospitalization and the realization of physiotherapy sessions because of persistent bronchial congestion. Control chest CT scan shows significant improvement of the pulmonary images (Figure 2). Her transfert in peripheral service is possible after 17 days of hospitalization in ICU.

Comment

*Klebsiella pneumoniae* is a commensal digestive tract and airway bacterium in human. Contamination takes place through oropharyngeal way or hand contact. These germs can cause pneumonias that can evolve towards abscessation. Often found on debilitated patients, they are rarely community-acquired in the USA and in Europe. However, they are a major cause of community-acquired pneumonia in Asia and in Africa, with a prevalence of 62% in Taiwan and 29% in South Africa [1,2]. In our context, *Klebsiella pneumoniae* is frequently isolated in hospitals. On the other hand, the absence of bacterial ecology data in hospital environment or outside, on asymptomatic carriage of this germ in the general population, are boundaries to the diagnosis of community-acquired *Klebsiella pneumoniae* infection. Consequently, history time line between hospitalization, previous consultations and the beginning of the symptoms, allows to confirm the community-acquired character, reinforced by definitions proposed in the expert consensus [3]. *Klebsiella pneumoniae* community-acquired pneumonias are associated with the following risk factors: advanced age, precarious socio-economic conditions, smoking, underlying chronic diseases (bronchopulmonary, hepatobiliary, renal, solid cancers and malignant hemopathies), immunosuppression (malnutrition, ethylism, diabetes mellitus, HIV infection, various immuno-deficiencies) [2,4,5]. In our observation, identification of a trisomic background appears as a risk factor. Infection susceptibility and their frequent occurrence would be an immunosuppression proof. Germ found in this patient is not a wild-type strain, because it is not only aminopenicillin-resistant, but also cephalosporin and quinolone-resistant. In addition, the possibility of a *Kp* beta-lactamase extended spectrum (BSLE +) is not really possible given the germ sensitivity to the tazobactam – piperacillin association. It is a multi-resistant bacillus but not BSLE +. Prescription of broad-spectrum antibiotics, and invasive instrumentation (bladder probes, catheters, and intubation) promote *Klebsiella pneumoniae* strain mutations in hospital. It seems that some factors also facilitate this germ mutation in the community. Those factors are: age (sixty years old or older), diabetes, antibiotic city treatment especially if it was used three months before the beginning of the symptoms [2,6]. In our case, there is a regular antibiotic intake as a main factor for the surfacing of this resistant strain. The discovery of BSLE+ strains since 1980 and of those now resistant to carbapenems [7], requires a comprehensive sensitivity with minimum inhibitory concentrations assays. Hence the interest of a fast microbiological diagnosis for the start-up of an active antibiotic on the germ.

Conclusion

This observation demonstrates a serious *Kp* community-acquired pneumonia, with support and immediate control of organ failure. It is a diagnostic to consider, especially on debilitated patients. Consequently, microbiological diagnosis must be fast in order to start an appropriate antibiotic therapy. This observation raises the need for a rational use of antibiotic city treatment and the monitoring of the level of resistance of some community-acquired germs.

References


Figure 2: Pulmonary image at D9 of hospitalization.