Editorial

Pediatric Pneumonia in China: New State of Alarm or Foreseeable Situation?

Paolo Sossai1, Barbara Hugonin Rao1, Enrico Bernini Carri1

1 MEDIS International School of Maxiemergency and Disasters, Modena, Italy.
*Corresponding email: paolo.sossai[.]unicam.it

The international community has been alarmed by the report of an epidemic of respiratory viral infections (many of which have evolved into pneumonia) in Chinese schools: the Chinese National Health Commission sounded the alarm on November 13, 2023, hypothesizing that Mycoplasma Pneumoniae was involved in the genesis of the pneumonia cases [1]. Are we facing a new epidemic caused by an unknown virus or can this situation have logical explanations? On November 22, 2023, WHO (World Health Organization) asked the Chinese authorities for more details on this noted increase in respiratory diseases in children [1].

The Chinese authorities affirm that no new virus has been identified but that these are infections attributable to normal respiratory viruses, which are common for the season (Respiratory Syncytial Virus RSV, Influenza, Rhinovirus and Adenovirus) sometimes associated with Mycoplasma Pneumoniae. Mycoplasma pneumoniae is a bacterium without a cell wall (therefore resistant to penicillins), and as a result a Gram stain cannot be used. In 1938, Reimann described for the first time seven cases of pneumonia, which he defined as atypical due to its torpid evolution, and in 1944 Monroe Eaton discovered that the agent of these “atypical” pneumoniae was a germ later called Mycoplasma Pneumoniae [2]. In about 30% of Mycoplasma Pneumoniae infections in children, the pathogenic viruses are present [3]. A fact to consider is the increase in necrotizing pneumoniae deriving from Mycoplasma Pneumoniae, Streptococcus pneumoniae and Staphilococcus aureus. A French study by Lemaitre C et al found that in the period between 2009-2011 the cases of necrotizing pneumonia doubled, reaching 9% of pneumonia cases [4].

One of the possible causes of this increase would be antibiotic resistance. As far as Mycoplasma pneumoniae strains are concerned, resistance to macrolides may reach up to 18% in Asian countries [5]. In these cases, the careful use of tetracyclines, doxycycline and quinolones are recommended for bone and joint risk in children. Let’s not forget that the presence of Mycoplasma Pneumoniae infections can be complicated by the onset of cold agglutinins syndrome [2]. So, what could be the causes of this “alert” that has shaken public opinion and the scientific community? It is possible that greater isolation and much more restrictive regulations in China than in other countries, during the SARS-CoV-2 pandemic, may have contributed to
immunity “debt” (from fewer contacts) preventing the development of effective defenses against viruses commonly circulating in school communities during this period of time, and above all among younger children [6].

A greater immaturity of this immune defensive system may have been the basis of a rapid and widespread contagion of common respiratory viruses that have taken root more significantly in organisms that are therefore “less defended” and more exposed. One of the hypotheses that we consider most credible to explain the sharp reduction of other viruses in the SARS-CoV-2 pandemic phase, is that of the fundamental role of “viral interference”. It is known that the immune system “learns” to defend itself thanks to continuous infections, not only by producing antibodies (B lymphocytes) but also by maturing a gradual cell-mediated “Immune memory” (T lymphocytes); in children, this defensive system, not yet mature, is replaced by “innate immunity” that provides immediate defense, as well as the gradual production of the different types of Interferon that is stimulated by infectious insults, and that “interferes” with the possibility of other viral infections attacking the body [7].

If this hypothesis can be confirmed, it will reaffirm the importance of interferons in common infectious processes and will confirm one of the hypotheses of why Sars-Cov2, in children and adolescents, has a less serious clinical course due to the high amounts of interferon, such as type 1 (alpha and beta) for example, produced by plasmacytoid dendritic cells [8]. The other hypothesis formulated to explain the lower spread of other viruses during the SARS-CoV-2 pandemic, is that hygiene measures would have slowed down the spread of all infectious agents, but this should have also involved SARS-CoV-2 itself, which instead has remained prevalent over all other viruses. In any case, we must remember that periodically the world has been and will be plagued by pandemics which, with current facilitated transportation, will spread increasingly faster. For example, immediately after the SARS-CoV-2 pandemic WHO raised the alarm for the spread of monkey pox (monkeypox), even if this news was more subtle [9]. We thought we only had to deal with chronic diseases but the SARS-CoV-2 pandemic has reminded us that we must not let our guard down against infectious agents, just like Carlo Urbani taught us with SARS, and both humanity and nations must be aware of having to close borders quickly once the first infectious outbreaks occur. In addition, strengthening and maintaining an adequate infectious surveillance network, as well as adequate hospital facilities with adequately trained and informed personnel, is a necessity.

References


