Respiratory Medicine Case Reports 20 (2017) 89–91

Contents lists available at ScienceDirect

Respiratory Medicine Case Reports

journal homepage: www.elsevier.com/locate/rmcr

Case report

Acute respiratory distress syndrome related to *Mycoplasma pneumoniae* infection

Nouha Chaabane [a, *], Elisabeth Coupez [b], Matthieu Buscot [c], Bertrand Souweine [b]

[a] Pulmonary and Allergology Department, University Hospital, Clermont-Ferrand, France
[b] Intensive Care Department, University Hospital, Clermont-Ferrand, France
[c] Service de Pneumologie, CHU Nice, Université Côte d'Azur, France

ARTICLE INFO

Article history:
Received 20 July 2016
Received in revised form
29 November 2016
Accepted 30 November 2016

Keywords:
Acute respiratory failure
Respiratory infection
Mycoplasma pneumonia

ABSTRACT

M. pneumoniae respiratory infection is usually mild and self-limiting. We report a case of acute respiratory distress syndrome (ARDS) due to *M. pneumoniae* infection in a 60 years old woman. Quick diagnosis was established by multiplex PCR assay for detection of pneumonia-causing bacteria. Outcome was favorable. The factors accounting for the severity of pneumonia caused by *M. pneumoniae* are discussed.

1. Introduction

M. pneumoniae a respiratory pathogen transmitted from person to person via respiratory droplets evolves as both endemic and epidemic infection. The incubation period prior to symptom emergence may be short or as long as 3 weeks. *M. pneumoniae* is one of the most common causes of lower respiratory tract infections (LRTI) and accounts for up to 40% of LRTI in the community [1–5]. *M. pneumoniae* infection may be asymptomatic and when symptomatic is usually mild, causing upper and/or lower respiratory tract symptoms, often self-limiting. Therefore, the term "*walking pneumonia*" has been widely used by physicians [3]. *M. pneumoniae* is much less often involved in severe forms of LRTI as a recent report from the Centers for Disease Control and Prevention, estimated only 2% of detectable pathogens in hospitalized community-acquired pneumonia (CAP) adults patients were due to *M. pneumoniae* [6]. We report a genuine ARDS due to *M. pneumoniae* infections whose outcome was favorable.

2. Case report

A 60 years old woman with post anoxic motor infirmity, living in nursing home, was admitted for acute respiratory failure. Few days prior to admission, she presented abdominal pain and high-grade fever with cough. Her relatives reported an outbreak lower respiratory infection in her nursing home in the past weeks. She has no significant past history of respiratory illness. Physical examination showed superficial polypnea (respiratory rate $\geq$ 50/min), supraclavicular drawing, seesaw respiration and profound desaturation (SpO_2 80% with high concentration oxygen mask). Chest radiograph showed bilateral extensive infiltrates (Fig. 1). She deteriorated rapidly and necessitated intubation and mechanical ventilation. The PaO_2/FiO_2 ratio was 65 at 11 cm H_2O positive end-expiratory pressure. Diagnostic work up of this ARDS did not reveal any extra-pulmonary causal disorder. Intravenous broad-spectrum antibiotics (cefotaxime and spiramycin) were immediately started to cover both pneumococcus and atypical pathogens.

Blood investigations showed 4.83/μL white blood cell count, mainly formed of neutrophils (3.09/μL) elevated C-reactive protein (263 mg/L) and procalcitonin (2.7 μg/L), with normocytic anemia (hemoglobin 11.1 g/dL, MGV 92 fl); platelet 70 cells/mm^3, BUN 13.9 mmol/L; serum creatinine 93 μmol/L; ASAT 121 IU/L; LDH 456 IU/L. Tracheo-bronchial aspirates obtained on admission, detected *Mycoplasma pneumonia* by universal polymerase chain reaction (PCR). Blood and urine cultures were negative. Legionella and pneumococcal urinary antigens were negative. According to

Abbreviations: ARDS, Acute Respiratory Distress Syndrome; ARF, Acute Respiratory Failure; CRP, C-reactive protein; PEEP, Positive End Expiratory Pressure; LRTI, Lower respiratory tract infections; CAP, Community-acquired pneumonia; BUN, blood urea nitrogen; ASAT, Aspartate aminotransferase; LDH, lactate dehydrogenase.

* Corresponding author. Department: Pneumology, Hospital: Tenon, 4 rue de la Chine, 75020 Paris, France.
E-mail address: nouha.cb@gmail.com (N. Chaabane).

http://dx.doi.org/10.1016/j.rmcr.2016.11.016

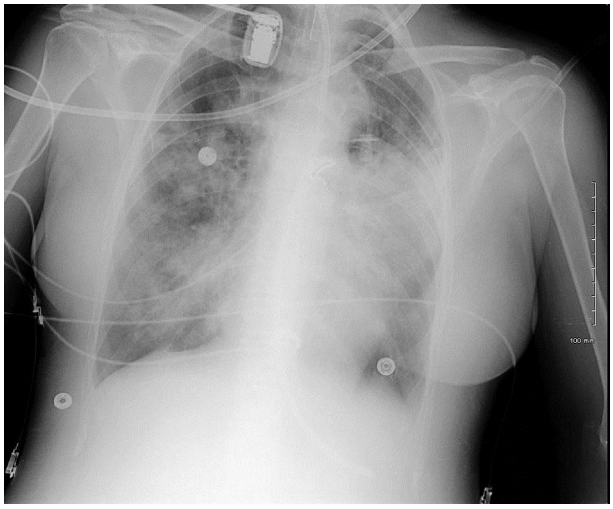

Fig. 1. Chest X-ray immediately showing bilateral extensive infiltrates.

international guidelines, sedation, prone position, inhaled NO and corticosteroids were administered. Outcome was favorable and the patient was weaned from the ventilator on day 9 and discharged from the ICU on day 13 without residual permanent damage. Serologic tests carried out on admission and 3 weeks after discharge showed 4-fold increase antibodies and the presence of anti *M. pneumoniae* IgM antibodies.

3. Discussion

ARDS caused by *M. pneumoniae* has rarely been described. In the present case we could establish a rapid and definite diagnosis of *M. pneumoniae* infection in a patient with ARDS, on the basis of positive PCR together with a negative diagnostic assessment for alternative etiologies.

In 1995 Chan and Welsh reviewed the English-language literature on severe *M. pneumoniae* CAP from 1966 to 1991 and found a total of 46 cases, 13 of which presenting fatal respiratory failure [7]. The average age in this series was 35 years. Miyashita et al. reported a series 227 cases of *M. pneumoniae* CAP, of which 13 presented acute respiratory failure [5]. No mortality was reported. Chaudhry et al. reported a genuine ARDS caused by *M. pneumoniae* and found 10 similar cases in the English literature from 1995 to 2010 [8]. More recently Izumikawa, summarized the Japanese literature from 1979 to 2010 and found a total of 52 cases, 2 of which presenting fatal respiratory failure [9]. As in the previous series, the dominant population was young adults (mean age 42.3 years) without severe underlying diseases. The average duration from onset of infection to the development of respiratory failure was 11.2 days (range, 5–21 days).

In these series as in most other published case reports, *M. pneumoniae* infection was diagnosed by serological antibody tests such as passive agglutination (PA) test, complement fixation (CF) test, or indirect hemagglutinin (IHA) test, either in elevated single titers or elevated paired titers.

One of the reasons for the scarcity of reports on *M. pneumoniae* related ARDS is that ARDS carries a high mortality rate. This indeed does not allow firmly establishing the diagnosis of *M. pneumoniae* infection when the diagnosis relies on paired antibody titers that require several weeks to show seroconversion. Our case as other recent reports suggest that rapid, accurate, and readily available diagnostic test such as multiplex PCR assay for detection of five pneumonia-causing bacteria may improve detection of *M. pneumoniae* in ARDS patients [10,11].

Several factors may account for the severity of pneumonia caused by *M. pneumoniae*. Delayed administration of adequate antibiotics has been suggested to contribute to the severity of *M. pneumoniae* pneumonia [5,9]. Antibiotic resistance although uncommon at least in Europe and northern America [12,13] may be suspected in case of unresponsiveness to macrolides, although delayed response in the absence of resistance has been reported [11]. Possible co-infection with other respiratory pathogens, such as *S. pneumoniae* warrants systematic search for alternative pathogens in severe cases [14]. Hyper-activated cell-mediated immunity may have a strong impact on the course of disease development following *M. pneumoniae* infection and several authors highlighted the need for steroid administration, early in the course of the disease, at least in severe cases in order to reduce the immune-mediated pulmonary injury [5,9].

All these factors argue for the need of antibiotic regimens including *M. pneumoniae* in their spectrum in severe CAP and also for rapid definite etiologic work-up of severe CAP, including rapid diagnostic tools such as multiplex PCR assay for detection of pneumonia-causing Last, the severity of pulmonary disease caused by *M. pneumoniae* can dependent on the capacity of various strains to produce the recently discovered, community-acquired respiratory distress syndrome (CARDS) toxin [15]. Although we could not investigate CARDS toxin production in our case, future epidemiologic investigations regarding CARDS toxin production may be helpful in understanding clinical characteristics of *M. pneumoniae* infections.

Authors' contributions

N.Chaabane collected and analyzed data, and wrote the paper.
M. Buscot wrote the paper.
E. Coupez and B. Souweine collected and analyzed data.

Disclosures

The authors have no conflict of interest to declare.

References

[1] J.G. Bartlett, L.M. Mundy, Community-acquired pneumonia, N. Engl. J. Med. 333 (1995) 1618–1624.
[2] M.R. Hammerschlag, *Mycoplasma pneumoniae* infections, Curr. Opin. Infect. Dis. 14 (2001) 181–186.
[3] K.B. Waites, D.F. Talkington, *Mycoplasma pneumoniae* and its role as a human pathogen, Clin. Microbiol. Rev. 17 (2004) 697–728.
[4] NIHIDSC, *Mycoplasma pneumoniae* Pneumonia, National Institute of Health Infectious Disease Surveillance Center, Tokyo, Japan, 2007. http://idsc.nih.go.jp/idwr/kanja/weeklygraph/18myco-e.html.
[5] N. Miyashita, Y. Obase, K. Ouchi, K. Kawasaki, Y. Kawai, Y. Kobashi, M. Oka, Clinical features of severe *Mycoplasma pneumoniae* pneumonia in adults admitted to an intensive care unit, J. Med. Microbiol. 56 (2007) 1625–1629.
[6] S. Jain, W.H. Self, R.G. Wunderink, S. Fakhran, R. Balk, A.M. Bramley, C. Reed, C.G. Grijalva, E.J. Anderson, D.M. Courtney, J.D. Chappell, C. Qi, E.M. Hart, F. Carroll, C. Trabue, H.K. Donnelly, D.J. Williams, Y. Zhu, S.R. Arnold, K. Ampofo, G.W. Waterer, M. Levine, S. Lindstrom, J.M. Winchell, J.M. Katz, D. Erdman, E. Schneider, L.A. Hicks, J.A. McCullers, A.T. Pavia, K.M. Edwards, L. Finelli, CDC EPIC Study Team, Community-acquired pneumonia requiring hospitalization among U.S. adults, N. Engl. J. Med. 373 (2015) 415–427.
[7] E.D. Chan, C.H. Welsh, Fulminant *Mycoplasma pneumoniae* pneumonia, West J. Med. 162 (1995) 133–142.
[8] R. Chaudhry, I. Tabassum, L. Kapoor, A. Chhabra, N. Sharma, S. Broor, A fulminant case of acute respiratory distress syndrome associated with *Mycoplasma pneumoniae* infection, Indian J. Pathol. Microbiol. 53 (2010) 555–557.
[9] K. Izumikawa, K. Izumikawa, T. Takazono, K. Kosai, Y. Morinaga, S. Nakamura, S. Kurihara, Y. Imamura, T. Miyazaki, M. Tsukamoto, K. Yanagihara, K. Hara, S. Kohno, Clinical features, risk factors and treatment of fulminant *Mycoplasma pneumoniae* pneumonia: a review of the Japanese literature, J. Infect. Chemother. 20 (2014) 181–185.
[10] G.L. Parrott, T. Kinjo, J. Fujita, A compendium for *Mycoplasma pneumoniae*,

Front. Microbiol. 7 (2016 Apr 12) 513, http://dx.doi.org/10.3389/fmicb.2016.00513.

[11] B. Sztrymf, F. Jacobs, J. Fichet, O. Hamzaoui, D. Prat, A. Avenel, C. Richard, Mycoplasma-related pneumonia: a rare cause of acute respiratory distress syndrome (ARDS) and of potential antibiotic resistance, Rev. Mal. Respir. 30 (2013) 77–80.

[12] O. Peuchant, A. Ménard, H. Renaudin, et al., Increased macrolide resistance of *Mycoplasma pneumoniae* in France directly detected in clinical specimens by real-time PCR and melting curve analysis, J. Antimicrob. Chemother. 64 (2009) 52–58.

[13] M. Yamada, R. Buller, S. Bledsoe, G.A. Storch, Rising rates of macrolides-resistant *Mycoplasma pneumoniae* in the central United States, Pediatr. Infect. Dis. J. 31 (2012) 409–411.

[14] C.Y. Chiu, C.J. Chen, K.S. Wong, M.H. Tsai, C.H. Chiu, Y.C. Huang, Impact of bacterial and viral coinfection on mycoplasmal pneumonia in childhood community-acquired pneumonia, J. Microbiol. Immunol. Infect. 48 (2015) 51–56.

[15] C. Techasaensiri, C. Tagliabue, M. Cagle, P. Iranpour, K. Katz, T.R. Kannan, J.J. Coalson, J.B. Baseman, R.D. Hardy, Variation in colonization, ADP-ribosylating and vacuolating cytotoxin, and pulmonary disease severity among *Mycoplasma pneumoniae* strains, Am. J. Respir. Crit. Care Med. 182 (2010) 797–804.