
CARTAS AL EDITOR

Pathogens and acute respiratory distress syndrome

Dear editor: Acute respiratory distress syndrome (ARDS), represented mainly by the common cold, pharyngitis, nasopharyngitis, pharyngotonsillitis, laryngitis, otitis media, sinusitis, bronchitis, bronchopneumonia, and pneumonia,¹ is the most common reason for seeking medical attention and the fourth cause of mortality in Mexico.² The principal agents associated with this syndrome are viral;³ however, bacterial agents are associated with increased mortality, and the most common microorganisms are *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis* and *Streptococcus pyogenes*.⁴ In our community there is an increase in failures of common treatments, presumably provoked by an increase in resistant microorganisms or by the presence of uncommon ones.

In order to determine the pathogens most frequently associated with ARDS, their prevalence, and resistance patterns to common antimicrobials, we conducted a clinical survey of 194 students with acute respiratory infection who had not previously received treatment. The students were selected from five high schools belonging to the Universidad Autónoma del Estado de México (UAEM). A clinical diagnosis and appropriate bacteriological culture from the affected sites was conducted for each case.

The clinical distribution of ARDS was: pharyngitis (60.8%), pharyngotonsillitis (34.5%), nasopharyngitis (4.1%) and rinitis (0.5%). The agents associated with these were; *S. pyogenes* (23%), *M. catarrhalis* (55.1%) and *S. aureus* (49.4%). In addition, no bacterial pathogen could be isolated in 27 of the cultures. A high bacterial resistance to common antimicrobials was found: *S. pyogenes* showed a resistance pattern to pefloxacin (86.7%) and trimethoprim-sulfamethoxazole (51.1%), whereas the resistance of *M. catarrhalis* to ampicillin, trimethoprim-sulfamethoxazole, and carbenicillin was higher than 60% and lower than 21% to gentamicin, metilmicin, and nitrofurantoin. The microbial resistance of *S. aureus* to cefotaxime, ampicillin, penicillin, dicloxacilin, and cefatazidime was higher than 80% and lower than 21% for trimethoprim-sulfamethoxazole, gentamicin, cefalotina, and erytromicina. Strains *S. pyogenes* producing β -lactamase were not found.

ARDS is well recognized as a serious public health problem among specific age groups.^{4, 5} Free access to antibiotics and self-medication in most cases, regardless of etiology,⁶ have favored an increase in the rate of bacterial resistance in the three most common pathogens: *S. pneumoniae*, *H. influenzae* and *M. catarrhalis*.⁷ It has been suggested that the use of microbiologic tests, such as cultures of the affected sites, can improve diagnostic and therapeutic accuracy and avoid the emergence of resistant strains.⁸

S. pyogenes was the most common pathogen isolated in a single form; however, the identification of *M. catarrhalis* in all clinical diagnoses, with the exception of nasopharyngitis, was not expected in this population. Currently, it is accepted that *M. catarrhalis* is the third most common pathogen agent in children⁹ and in adults with immunologic compromise⁹ or chronic obstructive pulmonary disease.¹⁰ Its role as an etiology agent in healthy adolescents, however, has not been reported.¹¹ Only a low prevalence rate in carriers of *M. catarrhalis* has been reported in this age group.¹¹⁻¹³

This finding merits some consideration. First, the current rate in carriers of *M. catarrhalis* must be established in this age group, and specifically, in those who present with ARDS, in order to discard its pathogenic role. Second, even though the rates of colonization were naturally elevated and not associated with disease, *M. catharrhalis* is associated with a high betalactamase production index. This can favor the persistence of strains sensitive to betalactamic antibiotics through a synergetic effect with non-producing strains (as is the case for *S. pyogenes*),¹⁴ prolong the clinical course of the disease, and force a change in the selection of the antibiotic in order to avoid the appearance of resistant strains.

The study had some limitations. We were not able to dismiss an etiologic role of *M. catarrhalis* because all subjects were symptomatic. Although we were not

able to test the production of the BRO¹⁵ β -lactamase enzyme by *M. catarrhalis*, it was the second pathogen most commonly isolated. We also cannot dismiss the role of those pathogens previously described as commensals in the etiology of ARDS. This needs further investigation. Recently, *S. aureus* has been recognized as an invasive pathogen of the upper tract respiratory¹⁶ and also has been documented in the familiar transmission of disease¹⁷ and as a responsible of recurrent disease for drug resistant.¹⁸

The most important fact in this study was the high resistance of *S. pyogenes* to trimethoprim-sulfamethoxazole, which explains the high degree of failure of antibiotic treatment in our community. Cultures and office visits, despite the costs, should be considered as a strategy before the use of antibiotics.

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