Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Viral etiology of exacerbations of patients with chronic obstructive pulmonary disease during the winter season☆

Etiología viral de las exacerbaciones de los pacientes con enfermedad pulmonar obstructiva crónica en la época invernal

Dear Editor,

Chronic obstructive pulmonary disease (COPD) is a chronic inflammatory process that alters the patient's functional capacity. It is estimated to be the fourth leading cause of death worldwide, affecting 2.4% of the population.[1] COPD exacerbations (COPDE) constitute the main complications of this disease since they deteriorate lung function. These acute processes have multifactorial causes, including viral infections, especially during the winter season.[1,2]

Chronic inflammation caused by viral infection and stimulation of IL-8 and other cytokines appear to be the cause of COPDE and lung lesions.[3] The viral impact in these processes has been underestimated until now due to the detection techniques used; the current use of molecular techniques has demonstrated the leading role of viruses in these episodes. With this technology, different studies have demonstrated viral infection in between 30 and 40% of COPDE cases, primarily involving rhinovirus, influenza and respiratory syncytial virus (RSV).[3,4]

The incorporation of molecular techniques in the diagnosis of viral infections has allowed us to study their participation in acute COPDE cases.

We report a prospective study conducted during the period with the highest prevalence of this type of infection (from October 2018 to February 2019). One hundred and eighty-seven patients (79% men) who came to the emergency department with an exacerbation (cough, expectoration and respiratory distress) of their previously diagnosed COPD were analysed during the study period.

A single throat swab culture was performed in each patient to detect bacteria and viruses. The viruses were studied using a commercial real-time RT-PCR (Allplex Respiratory Assay, Seegen, South Korea) that simultaneously and differentially detected 16 different viruses.

One hundred and four (104) positive samples (55.6%) were detected in the 187 patients, of which 78 (41.7%) corresponded to viruses alone, 19 (10.1%) to bacteria alone, and 7 (3.7%) to viruses and bacteria together. That is, viruses represented 45.4% (85 cases) of all respiratory processes in these patients. Differences in viral aetiology were observed in each of the months studied: with 36.6% in November and 69.7% in December. Similarly, the percentage of viral infections as the sole cause of COPDE has also fluctuated: it was 60% in October and 94.4% in February.

The main viruses detected have been rhinovirus (30.5%), influenza A (H3N2) (28.2%), RSV-B (22.3%), coronavirus (16.4%), adenovirus (1.1%) and metapneumovirus (1.1%). The bacteria detected were mainly *P. aeruginosa* and *H. influenzae* and, of the 7 virus–bacteria associations, rhinovirus represented the majority of them (57%). Five patients (5.8%) with influenza A (H3N2) infection required admission to the ICU; no patient died as a result of viral infection.

A recent meta-analysis on the prevalence of viral infections in COPDE showed that a virus can be detected in up to 50% of cases if molecular techniques are used.[1–4] In our study, positive results for viruses reached a total of 45.4%, a value that is within the range reported. Our study simultaneously analysed the presence of bacteria, although they have represented only 10.1% of the cases. Whether they are simple colonizers or true pathogens cannot be determined.[2,5]

Rhinoviruses have been the most commonly detected viruses, which coincides with other studies that estimate them at up to 58%,[1,2] although in the winter season there is a high circulation of these viruses associated with the common cold. As expected, influenza viruses, in this case circulating A (H3N2), and RSV have been the cause of 50.5% of COPDE cases. Viral aetiology distribution data similar to ours have already been described, although with variations depending on the type of influenza circulating in a given winter epidemic.[1–4]

Rapid identification of the viral aetiology of COPDE cases avoids the unnecessary administration of antibiotics and anticipates a good prognosis. Of the main viruses involved, only influenza could be prevented by systematically vaccinating these patients each season.

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Conflict of interest

The authors have no conflict of interest.

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LDL cholesterol in one step☆

Colesterol LDL en un paso

Dear Editor:

In line with the joint recommendation of the *European Society of Cardiology* and the *European Atherosclerosis Society* of 2016 on the control of dyslipidemia[1], the 2019 guidelines provide additional data from observational, randomized-clinical, and genetic Mendelian randomization studies that unequivocally show the causal effect of *low density lipoproteins* (LDL) cholesterol in the development of cardiovascular disease of atherosclerotic origin. The therapeutic objectives of the current guidelines point to the concentration of LDL cholesterol as the main therapeutic target and has incorporated a higher level of demand based on new clinical evidence. In this sense, the recommended therapeutic target for very high-risk subjects based on the results of the IMPROVE-IT[2], FOURIER[3] and ODISSEY[4] studies is LDL cholesterol <55 mg/dl and a reduction ≥50% with respect to baseline LDL. Furthermore, this evidence points to switching high-potency statins to high-intensity lipid-lowering therapies. In the therapeutic algorithm for the pharmacological reduction of LDL cholesterol, the guidelines recommend using the statin with the necessary potency to achieve the required reduction based on the target LDL cholesterol, then go to the maximum tolerated dose if the objective is not achieved, then add ezetimibe and, if the goal is still not achieved, add a PCSK9 inhibitor.

All of this implies that in the case of a very high-risk patient with baseline LDL cholesterol >140 mg/dl, it will require the use of

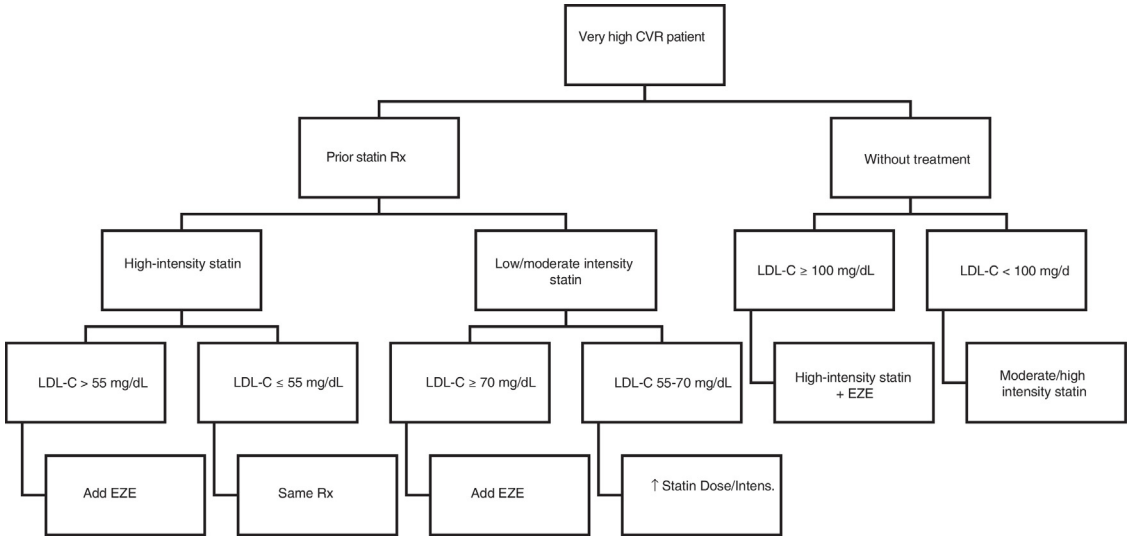

Fig. 1. Therapeutic algorithm to reach the LDL cholesterol goal in one step.
Note: PCSK9 inhibitors are not included as they are only indicated according to the therapeutic positioning report in cases of secondary prevention and/or familial hypercholesterolemia that do not reach the therapeutic objective despite being treated with the maximum tolerated statin dose or being intolerant to statins.
LDL-C: cholesterol linked to *low density lipoproteins*; EZE: ezetimibe; CVR: cardiovascular risk; Rx: treatment, therapy.

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