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.

favoured by biofilm formation. Therefore, catheter removal is necessary when *Kocuria* involvement is suspected.

To date, no case of native valve endocarditis had ever been described with *K. rhizophila*. Only *K. kristinae* et *K. rosae* have been found to be responsible for IE [8,9].

Identification errors for *Kocuria* species have been reported when using identification methods such as Vitek 2GP, which are liable to misidentify coagulase-negative staphylococci as *Kocuria* species or vice versa [10].

In our case, MALDI-TOF mass spectrometry showed 100% similarity to *K. rhizophila*. Given the increasingly generalized use of this equipment in hospitals and the updating of databases for other identification methods, the prevalence of this type of infection has been changing. Indeed, since 2012–2013 the number of cases reported in the literature has increased significantly.

However, as of now there exist no treatment guidelines or antibiotic susceptibility breakpoints for *Kocuria*. As a result, many publications continue to apply Staphylococcus breakpoints. Based on the staphylococcus-species interpretative criteria for agar disk diffusion assay in the 6th version of EUCAST, the *Kocuria rhizophila* strain isolated from our patient showed sensitivity to cefoxitine, kanamycin, clindamycin and trimethoprim/sulfamethoxazole, but manifested resistance to erythromycin and norfloxacine.

At the same time and according to the E-test® strip, susceptibility testing of the organisms revealed MIC of 16 mg/l to penicillin G (R), 3 mg/l to amoxicillin and amox-acid-clav (I), 1.5 mg/l to levofloxacin (I) and 0.047 g/l to cefotaxim (S).

This type of sensitivity profile seems to implicate the antibiotic resistance mechanisms of streptococci more than those staphylococci. According to the classification rearrangement and the sensitivity profile and in order to avoid clinical failure, we treated this patient based on EUCAST section "PK/PD (non-species related) breakpoints" with a third-generation cephalosporin (3GC).

4. Conclusion

This is the first reported case of *K. rhizophila* causing bacteremia related to central venous catheter use and involving native valve endocarditis. Clinicians should not underestimate the importance of *Kocuria*, especially in cases where the patient has an implanted medical device. Without validated breakpoints for *Kocuria*, sensitivity results should be determined in reference to an antibiotic with minimum inhibitory concentrations (MIC).

Disclosure of interest

The authors declare that they have no competing interest.

Authors' contributions

AP and SZ contributed to the writing. LT, PYR, PG and VG contributed to the review. All authors read and approved the final manuscript.

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Is a COPD patient protected against SARS-CoV-2 virus?

Dear Editor,

We would like to draw attention to the relationship between SARS-CoV-2 and chronic obstructive pulmonary disease (COPD). Considering the mode of transmission via droplets, and the virulence of SARS-Cov-2, we had anticipated that COPD patients would be at increased risk of SARS-CoV-2 infection. However, among 392 patients with documented SARS-Cov-2 infection admitted to two French University Hospitals between the 15 March and the 15 April 2020, only 22 (5.6%) patients had a documented COPD. All COPD patients were former or active smokers. In the entire cohort, 82 (21%) patients were admitted in medical ICU for ventilatory support (i.e., requiring mechanical invasive ventilation in 59 cases and non-invasive ventilation or high-flow oxygen therapy in 23 cases, respectively), including 5 patients (6%) with a documented COPD (GOLD stage I ($n = 2$), II ($n = 1$), III ($n = 1$), unclassified for 1 patient). Two patients required high-flow oxygen therapy only and 3 patients needed non-invasive ventilation. All COPD patients except 1 survived and were discharged from the hospital. Even if this observation is concordant with the low prevalence of 4% in COPD patients diagnosed with SARS-CoV-2 infection in a recent Italian cohort [1], this result is quite surprising since COPD patients admitted in ICU have many SARS-CoV-2 risk factors (i.e., male, overweight, arterial hypertension, chronic cardiopathy and diabetes mellitus). In addition, cigarette smoke and COPD were reported to up-regulate Angiotensin-converting enzyme 2 (ACE-2) expression,

which plays a key role in the pathogenesis of SARS-CoV-2 in lower airways [2].

Therefore, how can one explain that COPD patients are underrepresented in patients with SARS-CoV-2 infection?

We could hypothesize that the low prevalence of COPD patients in intensive care settings could be the consequence of pre-existing poor prognosis and decisions to limit the treatment to palliative care. Furthermore, patients with chronic respiratory diseases are likely to be more aware of viral epidemics and are more sensitive to containment and barrier actions than the general population. Another explication could come from COVID-19 pathophysiology.

Cell entry of SARS-CoV-2 is a multi-step process in which ACE-2 and the transmembrane protease serine 2 (TMPRSS2) for S protein priming play a crucial role. Considering a significant inverse relationship between ACE-2 gene expression and the severity of COPD [2], one hypothesis could be that COPD protects against SARS-CoV-2 through a TMPRSS2 inhibitor activity or by an a downregulation of the inflammatory pathway limiting severe forms of infection.

Interestingly, as reported by Halpin et al., inhaled corticosteroids, used by COPD patients can reduce the risk of viral infection. Indeed, budesonide was reported as a promising antiviral and anti-inflammatory drug candidate for the treatment of human rhinovirus infection [3]. *In vitro* experiments showed that inhaled formoterol alone or in combination with bronchodilators (i.e., glycopyrronium and budesonide) inhibited HCoV-229E replication partly by inhibiting receptor expression and/or endosomal function and modulated infection-induced inflammation. Inhaled corticosteroids also reduced the expression of transmembrane serine protease TMPRSS4 and TMPRSS11, which facilitates viral entry and proliferation in human bronchial epithelial cells *in vitro*. Therefore, inhaled corticosteroids in combination with bronchodilators could limit expression or activity of transmenbrane serine protease TMPRSS2 which facilitates SARS-CoV-2 entry in cells.

Surprisingly current smokers (i.e., a large part of COPD patients) could be protected against SARS-CoV-2 infection [4]. *In vivo* models support that chronic cigarette smoke exposure could downregulate ACE 2 expression in lungs via a mechanism dependent of Angiotensine II and Angiotensine II type 1 Receptor [5]. This result suggests a potential protector effect of nicotine against SARS-CoV-2 cell entry via a possible role of nicotinic acetylcholine receptors [4]. Whether cigarette smoking or nicotine do or do not provide a benefit effect warrants further studies.

Understanding why and how remains a challenge for researchers.

Ethical Approval

All procedures performed in studies involving human participants were in accordance with the 1964 Helsinki declaration and its later amendments.

Disclosure of interest

The authors declare that they have no competing interest.

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First case of mild encephalopathy with reversible splenial lesion in SARS-CoV-2 infection

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Central nervous system damage has previously been described for coronaviruses [1]. Regarding SARS-CoV-2, various neurological manifestations have been reported in the literature involving both the central and the peripheral nervous systems [2,3]. A recent systematic review underlined the high rate of central nervous system involvement and neurological manifestations in SARS-CoV-2 infections [4], particularly in those involving severe infection.

Herein, we report the case of a patient infected by SARS-CoV-2 who developed an original neurological presentation defined as mild encephalopathy with reversible splenial lesion (MERS), which to our knowledge had never previously been described as occurring over the course of this infection.

A 47-year-old man from French-speaking Africa with no medical history or daily treatment was driven to our emergency department on April 8, 2020 due to onset of confusion over the previous 48 hours. He also reported that a febrile dry cough and headache had occurred during the previous 15 days. On admission, he presented normal temperature (37.5 °C), tachycardia (heart rate 107) and tachypnea (35 breaths per minute), slightly elevated blood pressure (150/84 mm Hg) and oxygen saturation upon arrival of