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.

fever (Figure). Results of real-time PCR for SARS-CoV-2 on a nasopharyngeal swab were negative, but the test was performed 3 weeks after onset of symptoms, at which point sensitivity is approximately 60% to 70%.[13] Although the results of the CT chest scan were consistent with COVID-19 pneumonia, sputum or bronchoalveolar lavage SARS-CoV-2 RT-PCR was not pursued, given the absence of fever or cough. Overall, the temporal evolution of our patient suggests a postinfectious profile in the setting of probable SARS-CoV-2 infection. The results of the CSF SARS-CoV-2 RT-PCR and IgG antibody index were negative, arguing against neuroinvasion, but neither of these tests has been validated, and sensitivity is unknown. On electrodiagnostic testing, our patient had unequivocal demyelinating features, but both axonal and demyelinating variants have been described in association with SARS-CoV-2.[3,4] Although scarce outcome data are available, our patient's motor examination had substantially improved upon dismissal.

Cases of GBS are increasingly reported in the setting of SARS-CoV-2 infection. Given the ubiquity of the virus, coincident disease is a possibility, although the established association between GBS and infection argues against this proposition. Nonetheless, GBS appears to be relatively rare, based on the limited number of cases reported from countries past the peak of the first wave of the pandemic. It is possible, however, that the association was missed early on in the pandemic, either in critically ill patients who died of the illness or in patients with GBS who were not tested for the virus because of mild or no respiratory symptoms. Ongoing surveillance will be needed to confirm and further elucidate the nature of the association.

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Acute Profound Sensorineural Hearing Loss After COVID-19 Pneumonia

To the Editor: We present the case of a 60-year-old previously healthy man who was admitted to the intensive care unit with a confirmed case of coronavirus disease 2019 (COVID-19) pneumonia 3 days after his initial hospitalization and 8 days after the onset of symptoms (fever, cough). The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus was detected in a nasopharyngeal swab and in bronchoalveolar lavage fluid. Reverse transcriptase–polymerase chain reaction did not show any evidence of other concurrent viral infections including influenza, parainfluenza, respiratory syncytial virus, adenovirus, human metapneumovirus, and rhinovirus. An enzyme-linked immunosorbent assay–based antibody test later confirmed the presence of

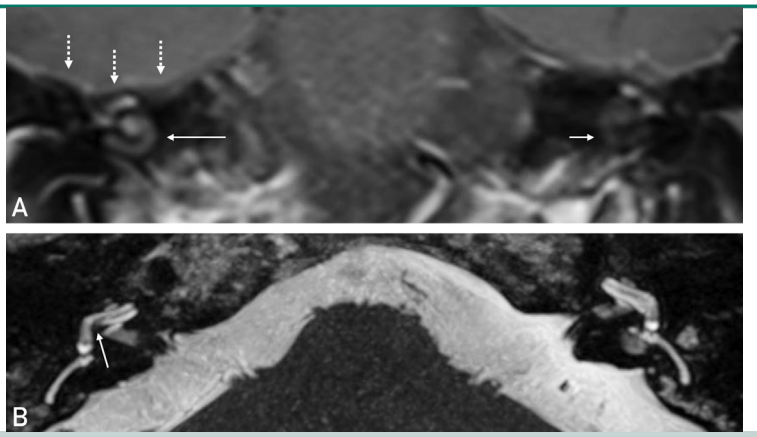

FIGURE. A, Coronal view in the T1 post-contrast sequence. Increased contrast enhancement of the right cochlea (long arrow). The left cochlea shows a normal hypointense pattern (short arrow). Linear contrast enhancement of the meninges at the base of the temporal lobe (dashed arrows). B, Axial view in the T2 sequence. Diminished fluid signal between the scala tympani of the basal cochlear turn and the vestibulum compared to the left side.

immunoglobulin A and G antibodies against SARS-CoV-2. After 13 days of intensive care treatment, the patient was transferred back to a medical floor in a stable cardiopulmonary state, but with hyperactive delirium. After recovery of his mental state, he reported deafness with a loud tinnitus (white noise) bilaterally. The hearing loss was confirmed using acoustically evoked potentials, and the patient was transferred to the ear, nose, and throat service for further diagnostics and treatment.

The patient had experienced no previous episodic or chronic hearing impairment. Audiologic testing revealed complete deafness on the right side and profound sensorineural hearing loss on the left side. A magnetic resonance imaging (MRI) scan showed pronounced contrast enhancement in the right cochlea (Figure A) and a partially decreased fluid signal in the basal turn of the right cochlea (Figure B). Adjacent to the temporal bone, meningeal contrast enhancement was seen at the base of the right temporal lobe (Figure A).

The MRI findings were interpreted as signs of an inflammatory process in the cochlea. Such a process can lead to soft tissue formation or even ossification of the cochlea, making the insertion of a cochlear implant (CI) electrode for hearing rehabilitation more challenging or impossible.[1] Hence the need for urgent CI was given. The patient's condition was still poor because of his recent COVID-19 infection; therefore, CI surgery was performed under local anesthesia with analgosedation instead of general anesthesia. The left ear was treated with three intratympanic triamcinolone injections to avoid the systemic immunosuppressant side effects of intravenous steroids.

During his treatment for COVID-19 pneumonia the patient had received two medications with reported ototoxic effects: azithromycin and furosemide. Independent of the individual toxicity profiles of these medications, a toxic effect is unlikely to manifest in MRI, as seen in this case. Furthermore, ototoxicity affects both ears symmetrically. Severe ototoxicity mediated by the above-mentioned drugs is therefore unlikely to be the cause of hearing loss in this patient.

Sensorineural hearing loss is a known complication of a number of viral infections. There is a plausible mechanism that may have caused virus-related hearing loss in the present case. A recent report of a series of 58 patients suggests an association between acute respiratory distress syndrome due to SARS-CoV-2 infection and encephalopathy, with 8 of 13 scanned patients showing leptomeningeal contrast enhancement,[2] as seen in the patient discussed here. Hearing loss is a known possible complication of bacterial or viral meningitis[3] and occurs to varying degrees in approximately 7% of cases.[4] In the present case, MRI signs of inflammation of the meninges and the right cochlea were present and the patient showed clinical manifestations in the form of delirium and hearing loss. Hence, there may have been virus-triggered inflammation of the meninges with subsequent spread to the cochlea, leading to acute hearing loss. Virus-triggered, immune-mediated inflammation seems likely, considering that severe cases of COVID-19 have been associated with a dysregulation of the immune system. In these severe cases, an increased neutrophil-to-lymphocyte ratio and elevated inflammatory cytokines such as interleukin 6 were observed,[5] features also seen in this case.

This stands in contrast to other sensory manifestations of the coronavirus infection such as anosmia, which can occur in otherwise asymptomatic patients. Gene expression databases have shown that the SARS-CoV-2 receptors ACE2 and TMPRSS2

are present in olfactory epithelium. With the virus replicating in the nose and nasopharynx, this represents a mechanism for direct damage of olfactory epithelium by the virus in the frame of a mild infection.

In conclusion, the case presented in this report highlights the importance of urgent audiologic and radiologic diagnostics in COVID-19 patients who report hearing loss, especially if neurologic symptoms are present.

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Glucose-6-Phosphate Dehydrogenase Deficiency and COVID-19 Infection

To the Editor: One unsettling aspect of the coronavirus disease 2019 (COVID-19) (severe acute respiratory syndrome coronavirus 2 [SARS-CoV-2]) pandemic is the variable susceptibility to infection. Some people who are exposed remain asymptomatic, others experience mild to moderate symptoms, while still others become severely ill and die. Hospitalization rates increase with age and approximately 90% of hospitalized patients have underlying medical conditions.[1] However, this does not account for a number of otherwise healthy or younger patients who are severely affected. Other suggested factors include genetic determinants.

A condition that should be considered is glucose-6-phosphate dehydrogenase (G6PD) deficiency. This X-linked recessive disorder with numerous allelic variants affects some 400 million people worldwide, with higher prevalence in Africa, the Mediterranean region, and Asia. Decreased production of G6PD results in deficient levels of nicotinamide adenine dinucleotide phosphate and reduced glutathione, causing oxidative stress and red blood cell destruction. Although frequently asymptomatic, patients may develop hemolytic anemia triggered by certain infectious agents and medications.[2] There is evidence to suggest an association between G6PD deficiency and increased susceptibility to, and severity of illness with, COVID-19 infection.

Wu et al[3] found that G6PD deficiency enhanced infection of cells with human coronavirus 229E (HCoV 229E). Using G6PD-deficient fibroblasts and G6PD-knockdown cells derived from human lung epithelial cells subjected to viral inoculum in vitro, they found that viral gene expression was higher in these cells compared with control cells. Production of viral particles in the deficient cells was also higher over time, indicating that G6PD activity modulates this production. Further, the G6PD-deficient cells were more susceptible to HCoV 229E—mediated cell death. SARS-CoV-2 may have a similar effect on cells in G6PD-deficient patients.

Spain and Italy have been particularly affected by the COVID-19 pandemic, with case fatality rates of 12.0% and 14.2%, respectively, as of this writing.[4] G6PD deficiency is more common in the Mediterranean region. On the Italian island of Sardinia alone, the prevalence ranges from 10% to 15%.[5] The allelic variants of G6PD deficiency in the Mediterranean region tend to manifest more significant phenotypic presentations. Although other factors may account for severity of illness in these countries, G6PD deficiency should be considered.

Reports from the United Kingdom and the United States show increased numbers of COVID-19 infection among members of minority groups. In the United Kingdom, 63% of the first 106 health care and social workers who died from the virus were black, Asian, or minority ethnic (BAME). BAME individuals make up 34% of patients admitted to UK intensive care units, although they only account for 17% of the population in the United Kingdom.[6] In the United States, African Americans have been more significantly affected than other races. Yancy[7] noted the infection rate in 131 predominantly black counties is 137.5 per 100,000 and the death rate is 6.3 per 100,000. These rates are three times and six times higher, respectively, than what are found in mostly white counties. Pre-existing medical conditions and adverse socioeconomic determinants of health may explain some of this disparity. However, G6PD deficiency is common among blacks and Asians. In a study of 63,302 US military personnel, 2.5% of males and 1.6% of females overall were deficient. Prevalence was higher among African American males (12.2%) and females (4.1%), as well as Asian males (4.3%).[8]