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Guillain–Barré syndrome

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Editor – We read with great interest the article by Sancho-Saldaña *et al*.[1] Firstly, we would like to congratulate the authors who have nicely described a case of Guillain–Barré syndrome (GBS) following a SARS-CoV-2 infection and was found to have a leptomeningeal enhancement in magnetic resonance imaging (MRI) of the spine. Although there is numerous reporting on the neurological involvement of SARS-CoV-2 infection that has been published in the literature, we believe it is still justified to report any extrapulmonary cases of SARS-CoV-2 infection, as we are still learning about the disease. We have written a comprehensive review of literature on all published scientific articles of SARS-CoV-2 infection with neurological involvement and summarised the wide spectrum of presentation which can present with or without respiratory symptoms.[2] However, this article is the first to report leptomeningeal enhancement in addition to the GBS, which warranted further investigation.

Nevertheless, there is some important information that is missing in the article, which is the background history of the patient, especially in terms of her underlying comorbidity and also the list of regular medication that she is taking. We believe this information is vital to determine the aetiology and prognosis of the condition. Apart from that, we believe a follow-up reporting is warranted on the rate and status of recovery after rehabilitation and the presence of other neurological sequelae.

At any rate, we agree with the authors that the causal relationship between GBS and SARS-CoV-2 infection follows the classical para-infectious and post-infectious pattern, as shown in this patient. Moreover, although leptomeningeal enhancement is not uncommon in GBS, we believe that further study and close follow-up is imperative because, apart from being a supplementary diagnostic sign in GBS, it can be linked to the development of a neurological dysfunction, such as multiple sclerosis that can lead to long-term or permanent neurological disabilities.[3] ■

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Lung ultrasound in COVID-19

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Editor – We thank Smallwood *et al* for their timely article about lung ultrasound (US) in COVID-19.[1] We completely agree that US can rule-in COVID-19 and that there is no published data on lung US and screening for COVID-19. We would gladly participate in the pragmatic research trial proposed, and would be happy to help in the set up. We have recently received numerous new US machines, as have many NHS trusts, have participated in a COVID-19 ultrasound database and published our ongoing experience.[2,3] However, the main sticking point to all of this is the number of practitioners who can and/or are 'signed off' to perform a standardised lung US with adequate reporting tools (which hopefully will follow on from the database). I am a respiratory consultant by trade, and very experienced at pleural ultrasound. A few years ago, I had attended a focused acute medicine ultrasound (FAMUS) course with the view to get formally accredited. However, lack of trainers in the north east of England and engagement from radiology colleagues to mentor me locally mean that my colleagues and I are completely self-taught in lung US and know that we are competent and confident. I do not have a set programmed activity for teaching US or any of the governance aspects around it, although we are currently writing up a business case.[3] I am sure that I am not alone in the UK. Furthermore, longitudinal competence programmes for basic point-of-care US do not exist.[4] So, should this pandemic be a time for widespread upskilling of emergency care, acute medicine and respiratory practitioners and not just doctors? Perhaps, but then the governance behind this is mind-boggling, and perhaps hampered by years of underfunding and under-recognition. The recent incorporation of US training into the acute medicine curriculum is welcome but not timely enough.[5] I am afraid there is no easy answer to any of this, and would welcome any further comments from lung US practitioners. ■

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Nosocomial COVID-19 on a green ward

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Editor – We read the recent article on nosocomial spread of COVID-19 on a stroke/neurology ward with interest.[1] We did a similar observational study in our hospital during the peak of the pandemic. The stroke ward was re-designated as a 'green ward', where patients with acute stroke and with non-COVID-19 symptoms were admitted. We wanted to estimate the risk of nosocomial COVID-19 because there was a perception that the risk of COVID-19 was negligible on a green ward.

During the study, we included all patients who stayed for more than 7 days from 15 March 2020 to 30 June 2020. We chose 7 days because, if patients developed COVID-19 after 7 days, we could be certain that they contracted the disease while they were an inpatient rather than delayed diagnosis from admission. Patients were tested for COVID-19 if they developed symptoms, had contact with a symptomatic patient or before transfer to the community. During the study period, staff were advised to use a surgical mask, apron and shield as per public health guidelines.[2]

Four-hundred and forty-three patients were admitted to the green ward during the study period; 138 patients stayed for more than 7 days, of which 59 tested positive either while as an inpatient or within 7 days of discharge and 12 patients died. During the same period 27 out of 47 healthcare professionals contracted COVID-19.

Despite using the recommended PPE, 42% of patients who stayed more than 7 days contracted COVID-19, of whom 21% died. Fifty-seven per cent of healthcare professionals developed COVID-19, of which, one needed respiratory support and all of them recovered.

Our figures are probably an underestimate due to the low sensitivity of the oropharyngeal swab (60%) and as we did not test all patients and staff, we might have missed a few asymptomatic cases.[3]

In summary, the risk of nosocomial COVID-19 is high if patients stayed in for more than 7 days and the risk to healthcare workers is extremely high, even on a green ward. This suggest that the recommended protective measures taken to prevent nosocomial COVID-19 were inadequate. ■

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Vitamin D deficiency and COVID-19

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Editor – Weir and colleagues timidly state that 'If vitamin D does in fact reduce the severity of COVID-19 … [then] supplements would offer a relatively easy option to decrease the impact of the pandemic.'[1] This reticence ignores about 40 years of developing scientific knowledge of the pivotal role of vitamin D, and its many unique extra-endocrine functions, including in the primary immune process and down-regulation of inflammation in all known infections, including ones new to man.[2] It is a vital nutrient, formed uniquely by action of UVB light on 7-dehydrocholesterol in the skin, when the sun is high overhead. In the light of variations in latitude, seasons, weather, skin colour, clothing cover, use of sun blocks and indoor living, the important question is not whether it should be taken, but how much. This can only be determined by measuring blood vitamin D levels, although the mantra of supposed overdose risk is grossly exaggerated.

There are now, to our knowledge, 14 studies that indicate the specific benefit in the COVID-19 pandemic of having a blood level of vitamin D greater than 30 ng/mL (75 nmol/L), and a very significant danger of death from infection if the blood level lies below 10 ng/mL (25 nmol/L).[3] We would not dream of treating diabetes without a knowledge of the blood glucose level. Likewise, with vitamin D deficiency at a time of a serious pandemic, the target blood level is critical, should be checked for and the dose adjusted accordingly. We suggest a target level of not less than 30 ng/mL and not more than 60 ng/mL. In most adults, this will be achieved by a mean D3 supplement of 4,000 IU daily. This is incidentally close to the recommended daily intake, weight-for-weight, for an adult laboratory mouse (~100 IU/kg).[4]

Another neglected fact is that vitamin D, acting via its VDR receptor, forms the working heterodimer not alone but paired with the vitamin A-activated retinol receptor (RXR). Yet National Institute for Health and Care Excellence (NICE) guidelines are dismissive of vitamin D's role in immunity, while generous in that of vitamin A.[5] This is completely illogical as, in their joint functions, the two match each other mol for mol.

NICE is a body that is orientated towards evaluating randomised controlled trials (RCTs) of pharmaceutical products, and so largely discounts observational studies. It does not appear to understand biological systems, such as the case of vitamin D, where sufficiency of an inactive natural storage precursor is crucial for the production of an active one that has many other local roles, in addition to the obvious general control of calcium absorption and bone health.[6]

But to help satisfy NICE skeptics, fortunately we now have an RCT from Córdoba, Spain, into the effect of vitamin D in COVID-19 pneumonia, which appeared after the paper by Weir and colleagues.[7] It demonstrated that, in those treated with oral 25(OH)D, there was a subsequent need for intensive care unit transfer in only 2% (with no deaths), versus 50% and two deaths in the control group. Evidence is cumulative, and is about more than RCTs, as Sir Austin Bradford