

Letters to the editor

OVERVIEW

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COVID-19: Redeployment through the eyes of an ophthalmologist

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Editor – The COVID-19 outbreak has strained healthcare systems and resources across the world.¹ Strategies to reinforce frontline staff led to the redeployment of sub-specialty doctors.² Among the redeployed ophthalmologists, there was much anxiety due to publicised reports of healthcare professionals being hospitalised.^{3,4} This, coupled with the prospect of being outside the realm of the eye clinic, was indeed a daunting one especially for those who had not worked on medical wards for over 10 years.

My redeployment is best summarised by a single snapshot of the monumental daily effort of the multidisciplinary team on the ward. Physiotherapists, occupational staff and nurses were thinly spread between mobilising patients, organising home support to help increase hospital capacity and keeping up with regular drug dispensing duties. Medical students were utilised on the wards as a vital extra pair of hands. Senior doctors led by example, examining patients by their bedside and astutely investigating for co-pathologies. Added to this were the frequent cameos of other specialties who promptly reviewed patients, epitomising the comradery that had brought the hospital together as one singular team.

How did I fit into this team? As an ophthalmologist, I reviewed adult and paediatric patients on the wards, ambulatory care and intensive care unit, and used this opportunity to teach junior doctors and consultants alike about ophthalmology by the bedside. My redeployed role also ranged from completing ward jobs to phoning patient families. This often meant talking to distraught family members about patients dying and relaying messages of support. Being the emotional bridge between families and patients was toilsome and often left feelings of helplessness.

COVID-19 has raised many questions on the wider socio-political preparedness, but it was also a time for individual healthcare professionals to reflect and improve. I re-evaluated my consultation style and made a conscientious effort to focus more on a patient-centred approach, even during those busy casualty clinics, and included family members in the consultations. Finally, witnessing the resilience, flexibility, bravery and altruistic behaviour of my colleagues reminded me of how proud I should be to be a healthcare professional and work in the NHS. ■

MINAK BHALLA

Specialist registrar in ophthalmology, Royal Free Hospital, London, UK

References

- 1 Gandhi RT, Lynch JB, del Rio C. Mild or Moderate Covid-19. *N Engl J Med* 2020. [Epub ahead of print].
- 2 Academy of Medical Royal Colleges. *Plans regarding trainee redeployment during the COVID-19 pandemic A position statement from the Academy Trainee Doctors' Group*. AoMRC, 2020.
- 3 Iacobucci G. Covid-19: NHS bosses told to assess risk to ethnic minority staff who may be at greater risk. *BMJ* 2020;369:m1820.
- 4 Kirby T. Evidence mounts on the disproportionate effect of COVID-19 on ethnic minorities. *Lancet Respir Med* 2020. [Epub ahead of print].

Vitamin D binding protein in COVID-19

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Editor – With interest, we read the paper of Weir *et al* about the potential association between vitamin D deficiency and COVID-19 severity.¹ The authors focused on the regulation of Tregs and thrombotic pathways by vitamin D. However, we think that vitamin D binding protein (VDBP) may also play a role in severe pulmonary complications of COVID-19.

In patients with COVID-19 pneumonia, a hyperinflammatory syndrome with activation of the complement system may lead to acute respiratory distress syndrome (ARDS), in which the C5a–C5aR axis plays an important role. VDBP has been detected in the bronchoalveolar lavage fluid of ARDS patients. At sites of endothelial injury, VDBP-release augments the chemotactic effect of complement derived C5a and C5a des Arg, promoting monocyte and neutrophil attraction, aggregation and activation to generate an oxidative burst. By competing for the same binding site on VDBP, 25(OH)D₃ and 1,25(OH)₂D₃ may inhibit this chemotaxis, thereby determining disease progression and outcome.²

Autopsy reports from COVID-19 patients have demonstrated severe endothelial injury, widespread vascular thrombosis with microangiopathy, and significant new vessel growth in the lungs. During cell death and lung tissue injury, globular actin (G-actin) is released in the extracellular compartment and polymerises into filamentous actin (F-actin). Sera from patients with ARDS contain F-actin, which may lead to the development of microembolisms, pulmonary vascular angiopathy, and multiple organ dysfunction syndrome. SARS-CoV can induce apoptosis and actin reorganization in mammalian cells under stressed conditions.³ Being members of the extracellular actin scavenger system, VDBP and gelsolin cleave actin and inhibit repolymerisation. However, elevated concentrations and/or prolonged exposure to VDBP-actin complexes may induce endothelial cell injury and death, particularly in the lung microvasculature.⁴

Finally, vitamin D supplementation may increase the low concentration of Tregs in COVID-19 patients.¹ Activated T cells