and this led to anticoagulation in nearly double the number of patients in the intervention group.[5]

Interestingly, 9% (15/167) of patients had an intracardiac source of embolus ('heart failure and thrombus' and 'valvular heart disease') and 25.1% (42/167) of patients had no underlying cause. Despite this, only 4.2% (7/167) of patients had transoesophageal echocardiography (TOE). A thrombus located in the left atrium or, more precisely, the left atrial appendage (LAA) is the most prevalent source of intracardiac emboli and is typically associated with AF. TOE is the imaging modality of choice for the evaluation of LAA.[6,7] Furthermore, in the absence of diagnosed AF, left atrial or LAA abnormalities may be a compelling indication for prolonged ECG monitoring.

In summary, investigation for aetiology of stroke in young patients should involve scrupulous cardiac investigations identifying those patients who would benefit from prolonged ambulatory ECG monitoring and increased utilisation of TOE. ■

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References

1 Bahl R, Kubiak M, Yusoff S *et al*. Proportion of acute ischaemic strokes attributable to a cardiac aetiology in an unselected young patient population: A single centre experience. *Clin Med* 2020;20:174–7.
2 Kamel H, Okin PM, Elkind MS *et al*. Atrial fibrillation and mechanisms of stroke: time for a new model. *Stroke* 2016;47:895–900.
3 Freedman B, Camm J, Calkins H *et al*. Screening for atrial fibrillation: A report of the AF-SCREEN International Collaboration. *Circulation*. 2017;135:1851–67.
4 National Institute for Health and Care Excellence. *AliveCor Heart Monitor and AliveECG app (Kardia Mobile) for detecting atrial fibrillation (MIB35)*. NICE, 2015. www.nice.org.uk/advice/mib35
5 Gladstone DJ, Spring M, Dorian P *et al*. Atrial fibrillation in patients with cryptogenic stroke. *N Engl J Med* 2014;370:2467–77.
6 Manning EJ, Weintraub RM, Waksmonski CA *et al* Accuracy of transesophageal echocardiography for identifying left atrial thrombi. A prospective, intraoperative study. *Ann Intern Med* 1995;123:817–22.
7 Veinot JP, Harrity PJ, Gentile F *et al*. Anatomy of the normal left atrial appendage. *Circulation* 1997;96:3112–5.

Procalcitonin for patient stratification and identification of bacterial co-infection in COVID-19

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Editor – an abundance of biomarkers has been measured in hospitalised patients with COVID-19. Initial reports from China have shown that most patients with COVID-19 did not have elevated procalcitonin (>0.5 μg/L).[1,2] However, elevated levels were found more frequently in severe cases and in patients who died.[2–4]

Variance in procalcitonin levels have previously been proposed to differentiate systemic inflammation of bacterial origin from viral origin in community acquired pneumonia and sepsis, with a significant rise indicating bacterial infection.[5,6] The lack of a procalcitonin rise in viral infections may be due to virus-stimulated production of interferon-γ by macrophages, which inhibits TNF-α in the immune response.[5] The presence of lower procalcitonin levels has been shown to have a 94% negative predictive value for bacterial co-infection in intensive care unit patients with confirmed influenza A(H1N1)pdm09.[7] Therefore, we suggest that raised procalcitonin observed in COVID-19 could be due either to bacterial co-infection, which is itself causing increased severity and driving systemic sepsis, or as a direct marker of a more severe or widespread viral infection.

As such, procalcitonin measurement on admission may be a useful marker to firstly predict patient deterioration in hospital and secondly, non-elevated procalcitonin on admission may be a good predictor of the absence of bacterial co-infection and allow the more targeted use of antimicrobials thus promoting antibiotic stewardship. Further work is needed to correlate the presence of raised procalcitonin and the presence of bacterial co-infection in COVID-19 patients. ■

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References

1 Chen N, Zhou M, Dong X *et al*. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. *Lancet* 2020;395:507–13.
2 Guan WJ, Ni ZY, Hu Y *et al*. Clinical characteristics of coronavirus disease 2019 in China. *N Engl J Med* 2020 [Epub ahead of print].
3 Chen G, Wu D, Guo W *et al*. Clinical and immunologic features in severe and moderate Coronavirus Disease 2019. *J Clin Invest* 2020 [Epub ahead of print].
4 Chen T, Wu D, Chen H *et al*. Clinical characteristics of 113 deceased patients with coronavirus disease 2019: retrospective study. *BMJ* 2020;368:m1295.
5 Müller B, Becker K, Schächinger H *et al*. Calcitonin precursors are reliable markers of sepsis in a medical intensive care unit. *Crit Care Med* 2000;28:977–83.
6 Müller B, Harbarth S, Stolz D *et al*. Diagnostic and prognostic accuracy of clinical and laboratory parameters in community-acquired pneumonia. *BMC Infect Dis* 2007;7:10.
7 Rodríguez AH, Avilés-Jurado FX, Díaz E *et al*. Procalcitonin (PCT) levels for ruling-out bacterial coinfection in ICU patients with influenza: A CHAID decision-tree analysis. *J Infect* 2016;72:143–51.