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From the Authors:

We showed that elevated high-sensitivity cardiac troponin T (hs-cTnT) at the time of hospital admission predicts mortality in patients admitted with community-acquired pneumonia (CAP).[1]

Dr Berk Takir *et al.* argue that presence of sepsis or septic shock instead of (or due to) CAP may have influenced our findings by referring to evidence showing that the degree of sepsis-induced cardiac dysfunction is an important prognostic factor in critically ill patients.[2]

Our study was conducted in a subset (n = 295) of a cohort of (n = 304) patients hospitalized with CAP in a non-academic teaching hospital.[1] Direct intensive care unit (ICU) admission was an exclusion criterion. Sixteen patients (5%) were admitted to the ICU later during admission. Forty-five patients (15%) presented with a pneumonia severity index (PSI) class V,[3] of whom only four patients were later admitted to the ICU. We acknowledge the assumption that secondary ICU admission might act as a surrogate of septic shock in patients with secondary ICU admission. However, given the low rate of these events, it seems unlikely that this has influenced our results. We have therefore conducted a sensitivity analysis in which we excluded patients admitted to the ICU later during admission. Both Kaplan–Meier and logistic regression analyses showed very similar findings compared with the original analyses. Thus, secondary ICU admission (as a surrogate of sepsis) was not an important confounder.

In addition, Dr Berk Takir *et al.* suggest that hs-cTnT might have functioned as a surrogate marker of hypoxaemia (oxygen supply–demand mismatch), rather than an indication of myocardial ischaemia in our study. This hypothesis was mentioned in our manuscript.[1] As stated, we believe that hs-cTnT level elevation during CAP admission might unveil clinically unrecognized coronary artery disease, which can be one explanation of the high long-term mortality rates we described. Other studies have shown evidence supporting this theory.[4,5] Furthermore, we do acknowledge that hs-cTnT levels were influenced by co-morbid disease (i.e. renal failure and heart disease). In our cohort, of all patients with elevated hs-cTnT (≥14 ng/L), 18.9% had chronic renal failure, 29.5% had chronic heart failure and 6.8% had both. Other proposed mechanisms potentially playing a role are inflammation-induced platelet activation[6] and indirect or direct damage caused by the *Streptococcus pneumonia* bacterium. The latter was shown in a recent study in non-human primates, demonstrating that *S. pneumonia* has the ability to invade the myocardium, resulting in cardiomyocyte death.[7]

More detailed studies would be needed to differentiate between possible underlying mechanisms of hs-cTnT elevation in patients admitted with CAP.

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Disclosure statement

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