Dear Editor

A recent article on 150 COVID-19 patients from Wuhan, China, was a comprehensive analysis of clinical predictors of mortality [1]. Age, cardiovascular comorbidities, total leucocyte counts, lymphocyte count, platelet count, liver and kidney functions, IL-6, C-reactive peptide (CRP) and cardiac biomarkers were significantly associated with increased mortality. Fulminant myocarditis was stressed as a poor prognostic marker. However, an equally important parameter is the percentage of lymphocytes. In the same trial, total leucocyte count was $10.62 \times 10^9$ cells/litre vs $6.76 \times 10^9$ cells/litre in the dead vs survival group, respectively. On the other hand, lymphocytopenia was more profound; $0.662 \times 10^9$ cells/litre in the dead group vs $1.4262 \times 10^9$ cells/litre in the survival group. Both these values were statistically significant (p value < 0.05) [1].

Similarly, in an initial compilation of data of 51 COVID-19 patients at the authors’ current centre from India, a trend supporting all the above observations is becoming increasingly noticeable. The mean leucocyte count was $5.7 \times 10^9$ cells/litre with a mean lymphocyte percent of 40.6% (Figure 1, 2). As the mean leucocyte count falls in the normal range, this is a relative lymphocytosis, defined as increased lymphocyte percent to 40% or more [2]. The mean haemoglobin was 16 g/dL and platelet count was $260 \times 10^9$ per litre. All the patients had stable vitals, preserved organ functions and required only symptomatic treatment for fever with or without cough; thereby being classified as having mild upper respiratory tract infection [3].

Lymphopenia has been found to be very common (85%) in critically ill COVID-19 patients [4]. Another study tried to develop predictive models,
where lymphocyte count > 20% at day 10 of illness has been found to be characteristic of the moderate group with favourable prognosis [5]. In concordance with that, as our mean lymphocyte count was 40.6%, all patients were predicted to have a recovery.

Multiple mechanisms have been proposed for lymphopenia in severe COVID-19. Direct lymphocyte inhibition, lymph node destruction, inflammatory cytokines, lactic acidosis suppressing lymphocytes and coronavirus attaching to the angiotensin-converting enzyme 2 (ACE2) receptor on lymphocyte are few plausible explanations [4]. However, relative lymphocytosis defies all these pathophysologies and hence may be an indication of a favourable prognosis. Acute viral illnesses like varicella, influenza, infectious mononucleosis are also associated with relative lymphocytosis; most of these eventually develop a spontaneous recovery [6]. The mechanism as to why some people respond favourably needs further research.

This is a very significant observation as a leucocyte count with differential count is done in all patients at admission. This, along with other predictors like age and comorbidities, can be used to make a quick, early decision on the further priority and triage, thereby assisting in efficient resource allocation.

**Conflict of interest**

None declared.

**References**