



## Clinical importance of serum procalcitonin in ulcerative colitis patients

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### Abstract

We have read with interest the recently published article entitled "Clinical significance of serum procalcitonin in patients with ulcerative colitis" by Koido *et al*. They aimed to investigate the association of procalcitonin with ulcerative colitis (UC) activity. They concluded that elevated procalcitonin levels were significantly correlated with UC activity. We would like to thank the authors for their comprehensive contribution.

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**Key words:** Procalcitonin; Ulcerative colitis; Clinical significance; Disease activity; Remission phase

**Core tip:** In a recently published article entitled "Clinical significance of serum procalcitonin in patients with ulcerative colitis", Koido *et al* investigated the association of procalcitonin with ulcerative colitis (UC) activity. They concluded that elevated procalcitonin levels were significantly correlated with UC activity. We would like to thank the authors for their comprehensive contribution.

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### TO THE EDITOR

We have read with interest the recently published article entitled "Clinical significance of serum procalcitonin in patients with ulcerative colitis" by Koido *et al*<sup>[1]</sup>. They aimed to investigate the association of procalcitonin (PCT) with ulcerative colitis (UC) activity. They concluded that elevated procalcitonin levels were significantly correlated with UC activity. We would like to thank the authors for their comprehensive contribution.

PCT under normal conditions is the 116-amino acid peptide precursor of the biologically active hormone calcitonin secreted by the parafollicular C-cells of the thyroid gland and involved in calcium homeostasis<sup>[2]</sup>. Their levels increase in inflammatory states following bacterial or fungal infections, tumors, trauma and surgery<sup>[3]</sup>. In addition, PCT can be a useful tool for diagnosis of sepsis and septic shock. Additionally, it can be used as a guide for antibiotic therapy in individual patients as a surrogate biomarker<sup>[4]</sup>.

Currently, only a few studies examining the relationship between PCT and UC exist in the literature. Oruç *et al*<sup>[2]</sup> reported that PCT has no diagnostic value in determining disease activity after PCT levels in UC patients failed to show any statistical significance when compared with that of healthy control subjects.

Clinically, it is not easy to differentiate relapses of UC from an existing enteric infection. Several microorganisms including *Clostridium difficile*, *Campylobacter*, *Salmonella* and *Shigella* species, *Escherichia coli*, *Entamoeba histolytica*, and cytomegalovirus can precipitate exacerbation of UC. A

recent study by Nitzan *et al*<sup>[5]</sup> concluded that high rates of *Clostridium difficile* infection are associated with severe inflammatory bowel diseases related to prior antibiotic use.

In the study of Koido *et al*<sup>[1]</sup>, they declared that stool cultures were performed to exclude all possible infective etiologies. We, however, did not find this method to be adequate since some of these microorganisms cannot be cultivated *via* the classic stool culture method. Routine stool examination, parasitic examination and *Clostridium difficile* toxin A/B antigen tests are vital, albeit, they are simple methods. Since the infective pathogens couldn't be cultivated in stool cultures, these undetected bacterial, viral or parasitic microorganisms might have triggered the exacerbation of UC and led to the elevation of PCT levels. It would have been better if the authors had performed detailed microbiological assessments. In addition, the authors didn't clarify whether any of the patients with severe UC was given anti-microbial therapy prior to UC activity. It would have been more accurate, if the authors had mentioned these conditions as limitations. Either nonspecific elevations or false negativity in PCT levels can be seen in some situations<sup>[3]</sup>. In this context, repeated measurements of PCT could have been performed to minimize or rule out false negativity that may arise as a result of single measurements. And finally, it would have been more useful if the authors mentioned the level of the procalcitonin in the remission phase.

In conclusion, further studies are required to determine the relationship between PCT and UC activity. It is vital that PCT be evaluated with other independent variables and markers (*e.g.*, C-reactive protein, erythrocyte sedimentation rate) to provide the required information about the inflammatory status of the patient. Also, the crucial role of detailed microbiological assessment should be critically appraised.

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