The Effects of Clonidine on Inflammatory Biomarkers in Critically Ill Patients – Old Drug, New Tricks?

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Abstract:

Background: The difficulty in managing the hyper-inflammatory response during sepsis is becoming more prevalent in critical care. Recent evidence has shown that the alpha-2-agonist clonidine, commonly used as a sedative and analgesic, may hasten the resolution of inflammation and could aid in the management of inflammatory conditions. Currently, research has been inconclusive in discovering the exact mechanism of how alpha-2-agonists cause a reduction in inflammation, if at all.

Aims: To retrospectively evaluate whether clonidine was associated with any detectable changes in biochemical markers of inflammation in ICU patients within a critical care environment.

Method: 322 patients were identified as having been administered clonidine during their stay in critical care areas within Nottingham University Hospitals. Basic demographic data was cross-referenced with daily blood results to measure patients' white cell count, platelet and CRP levels. Albumin was also recorded as a marker of liver function. Four measurements were obtained relating to before, during and after patients received clonidine. Analysis, using SPSS v.24, was conducted between these measurements to see if clonidine caused any statistical differences in inflammation.

Results: Clonidine caused a statistically significant ($p=<0.001$) increase of 64mcL in platelet levels. Correlation analysis between the length of clonidine and platelet levels produced a statistically significant ($p=<.001$) r-value of .214 indicating a very weak positive linear association. Clonidine did not cause a significant change in white blood cell ($p=.538$) and albumin levels ($p=.693$). Mann-Whitney U analysis showed clonidine did not significantly decrease ($p=.056$) CRP levels.

Conclusion: Clonidine administration generated a minimal reduction in inflammation when compared with other factors in critical care. The lack of a deleterious effect shows that clonidine did not cause a reversal in inflammation and did not harm patients. This provides a legitimate rationale to continue research into the effects and exact role clonidine has in a critical care setting.