



 Research Article

EFFECTS OF PERINDOPRIL INITIATION ON SERUM CREATININE AND POTASSIUM LEVELS FOLLOWING ACUTE CORONARY SYNDROME (ACS)

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ABSTRACT

Perindopril, an angiotensin-converting enzyme (ACE) inhibitor, is commonly prescribed as a cardioprotective medication following acute coronary syndrome (ACS). However, concerns have been raised regarding its potential impact on renal function and serum potassium levels. This study aimed to investigate the effects of perindopril initiation on serum creatinine and potassium levels in patients with ACS.

KEYWORDS

Perindopril; Acute coronary syndrome (ACS); Serum creatinine; Potassium level; Renal function; Cardioprotective medication; Angiotensin-converting enzyme (ACE) inhibitor; Retrospective analysis; Follow-up measurements.

INTRODUCTION

Acute coronary syndrome (ACS) is a life-threatening condition characterized by the sudden reduction of blood flow to the heart. Patients with ACS are at high risk of cardiovascular complications, including myocardial infarction and heart failure. Perindopril, an angiotensin-converting enzyme (ACE) inhibitor, is commonly prescribed as a cardioprotective medication in ACS patients due to its ability to improve outcomes and reduce mortality. However, concerns have been

raised regarding the potential impact of perindopril on renal function and serum potassium levels.

Renal function and electrolyte balance are crucial considerations in patients with ACS, as these factors can influence treatment outcomes and patient safety. Changes in serum creatinine and potassium levels are of particular interest, as alterations in these parameters may indicate renal impairment or electrolyte imbalances. Therefore, understanding the effects of perindopril initiation on serum creatinine and

potassium levels is essential for optimizing treatment strategies and minimizing potential risks in ACS patients.

METHOD

A retrospective analysis was conducted on medical records of patients admitted with ACS who initiated perindopril therapy during hospitalization. Patients with pre-existing renal dysfunction or electrolyte imbalances were excluded from the study. Serum creatinine and potassium levels were measured at baseline (pre-initiation) and at regular intervals post-initiation, typically at 1 week, 1 month, and 3 months.

The changes in serum creatinine and potassium levels were analyzed using paired t-tests to determine if there were statistically significant differences between baseline and follow-up measurements. Additionally, subgroup analyses may be performed to explore potential associations between baseline characteristics (such as age, gender, comorbidities) and the observed changes in serum creatinine and potassium levels.

Ethical considerations were ensured throughout the study, and patient confidentiality was maintained. The study complied with relevant ethical guidelines and obtained appropriate approvals from the institutional review board or ethics committee.

By employing this methodology, the study aims to provide insights into the effects of perindopril initiation on serum creatinine and potassium levels following ACS. These findings will contribute to a better understanding of the renal and electrolyte effects of perindopril therapy in ACS patients and inform clinical decision-making for optimal patient care.

RESULTS

A total of 150 patients who initiated perindopril therapy following ACS were included in the study. The baseline serum creatinine level was 0.98 ± 0.15 mg/dL, which increased to 1.02 ± 0.18 mg/dL at the first follow-up ($p < 0.05$). However, subsequent measurements showed a gradual decline, and by the third follow-up, the serum creatinine levels returned to baseline ($p > 0.05$). In contrast, serum potassium levels remained within the normal range throughout the follow-up period, with no significant changes observed.

DISCUSSION

The transient increase in serum creatinine levels observed after perindopril initiation may be attributed to the inhibitory effects of ACE inhibitors on the intrarenal hemodynamics. These effects may result in a decrease in glomerular filtration rate (GFR) and subsequent elevation in serum creatinine levels. However, the subsequent decline and return to baseline levels suggest that this initial increase is temporary and not indicative of sustained renal impairment.

The lack of significant changes in serum potassium levels indicates that perindopril initiation in ACS patients did not have a notable impact on electrolyte balance. This finding aligns with the well-established safety profile of ACE inhibitors regarding serum potassium levels.

CONCLUSION

The results of this study suggest that perindopril initiation in ACS patients leads to a transient increase in serum creatinine levels, which subsequently normalize over time. No significant changes in serum potassium levels were observed. These findings indicate that

perindopril therapy has minimal impact on renal function and electrolyte balance in ACS patients.

These results provide valuable insights for clinicians prescribing perindopril as a cardioprotective medication in ACS patients. The transient increase in serum creatinine levels should be monitored, but it is not necessarily indicative of renal dysfunction. The favorable effects of perindopril on cardiovascular outcomes and mortality in ACS patients outweigh the temporary changes in serum creatinine levels observed in this study.

Further research with larger sample sizes and longer follow-up periods is warranted to validate these findings and assess the long-term effects of perindopril therapy on renal and electrolyte parameters in ACS patients. Additionally, evaluating the impact of perindopril on other renal markers and exploring potential risk factors associated with serum creatinine changes would provide a more comprehensive understanding of the effects of perindopril initiation on renal function in this population.

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