Efficacy and Gastrin Levels in Gastroesophageal Reflux Disease Patients Treated with Esomeprazole Following a Potassium: Competitive Acid Blocker

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INTRODUCTION

PPI drastically changed treatment of acid-related diseases. Later, P-Cab was launched in Japan as an acid secretion inhibitor with new MoA, attracting much attention for its more powerful inhibition effect than conventional PPIs. In order to verify the clinical efficacy of P-Cab, controlled clinical trials comparing between P-Cab and PPI were conducted at several medical institutions in Japan [1-4]. Researchers found that there was no significant difference in post-ESD ulcers. As P-Cab inhibits acid secretion more powerfully than conventional PPIs, it was expected that P-Cab would outperform PPI in clinical efficacy in GERD, but just the opposite results were reported one after another, where P-Cab was found comparable or inferior to PPI. In addition, it was found that P-Cab caused a notable increase in gastrin levels along with strong inhibition of acid secretion. The authors and others who conducted the trials decided to determine why GERD symptoms did not improve in spite of adequate acid suppression, and how these symptoms were related to elevated gastrin levels secondary to P-Cab administration. In order to clarify these questions, we decided to investigate how switching from P-Cab to Medium can impact symptom improvement and gastrin levels in patients on P-cab whose symptoms previously did not improve as expected.

METHODS

Esomeprazole 20 mg was orally administered to RE patients who had been treated with Vonoprazan 20 mg once daily for more than 4 weeks. Primary endpoint was changes in the total scores at Week 4 based on the frequency scale for the symptoms of Gastroesophageal Reflux Disease (FSSG) questionnaire. The secondary endpoints were changes in the reflux scores, motility scores, and total scores evaluated based on the FSSG questionnaire, changes in serum gastrin levels, and tolerability.

RESULTS

In 25 patients with mean age of 60.5 years, median FSSG total score demonstrated a significant decrease both at Week 2 (p<0.05) and Week 4 (p<0.01. Median FSSG reflux score demonstrated a significant decrease both at Week 2 (p<0.01) and Week 4 (p<0.01. Median FSSG motility scores showed no statistical significance at any time. Median serum gastrin level significantly decreased from 755 pg/mL at baseline to 309 pg/mL at Week 4 (p<0.01) (Figure 1).

CONCLUSION

The results suggested that acid suppression alone does not produce desired clinical efficacy, and that the gastrin levels before and after the switch had correlations with the patients’ symptoms, offering the possibility that excessive acid suppression may increase gastrin levels, resulting in gastric dysmotility.

I believe that excessive acid suppression as one demonstrated with P-Cab [5] is unnecessary clinically, and that for diseases that require long-term treatment such as GERD and drug-induced ulcers, it makes sense to suppress stomach acid to the minimum in respect of its natural biological balance developed through human evolution.

As a future issue, I believe that clinical trials are needed to thoroughly examine the secondary effects of sustained increase in gastrin levels caused by the use of P-Cab in Japanese subjects.
REFERENCES


