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Opinion Article

Role of Levothyroxine and its Effect on Stomach

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DESCRIPTION

Levothyroxine sodium monotherapy is commonly used as a replacement treatment for hypothyroid individuals all over the world. The need for a dose that is individualized to each individual has been firmly emphasized. However, a considerable number of patients fail to exhibit a biochemical and/or clinical response, necessitating higher thyroxin dosages to achieve the desired serum TSH values. Long-term inadequate therapies have a negative impact on body balance. In these patients, frequent dose modifications and repeated diagnostic procedures have been linked to increased health costs.

Recently, the causes of an elevated thyroxin need were examined. The significance of alteration in stomach physiology on subsequent intestinal T4 absorption has been frequently highlighted among these. The method by which thyroxin intestinal absorption is hindered in individuals with gastric diseases is unknown, but it appears to be related to the chemical and physical features of both naive and salificated thyroxin molecules. The levo-isomer of thyroxin, levothyroxine, is insoluble in water and other common organic solvents.

The salification process by a saturating excess of sodium hydroxide results in the creation of sodium salt, which is the chemical used in all pharmaceutical preparations of thyroxin. Because of safety and patient desire, oral administration is the preferred mode of administration.

Oral levothyroxine absorption is inefficient, with reported percentages, approximately 70% of the prescribed dose. The jejune-ileal tract represents the true location of absorption, while only a small portion of oral thyroxin is absorbed in the duodenum. Humans, unlike rats, have no evidence of extensive bowel absorption. Furthermore, the analysis of the lag time between thyroxin consumption and its presence in plasma rules out the likelihood of stomach absorption. Several clinical trials, however, have demonstrated that changes in gastric physiology may have a significant impact on oral thyroxin absorption, resulting in an increased demand for the medicine. The goal of this study was to review what was known and what was unknown about the effect of the stomach environment in the absorption of oral thyroxin.

The majority of medications are absorbed at the gut level. This idea is based on the mucosal membrane's enormous surface area and the existence of several transporters. The integrity, motility, mucus composition, and resident microbial population of the duodenum all influence absorption. Drug absorption from the stomach, on the other hand, is commonly assumed to be insignificant, despite the fact that passive diffusion through the gastric wall has been theorized and demonstrated for chemicals such as ethanol and tiny neutral molecules. The ionization status of the medication appears to be related to gastric absorption, which in turn depends on the gastric juice pH: in fact, in an acidic environment, acidic pharmaceuticals are primarily present and absorbed in a unionized form, while this process is minimal for basic drugs.

However, due to the scarcity of papers on this subject, additional research is required. In any case, the stomach environment has a significant impact on drug behavior and pharmacokinetics. In fact, numerous processes must be considered when examining the so-called "gastric phase," which is a critical precondition for intestinal medication absorption. The medicine undergoes disintegration, dissolution, and sometimes precipitation once it reaches the stomach; additionally, the active ingredient must reach the exact site of absorption.

The active substance is released from the solid state as a result of disintegration. The duration of this stage is heavily influenced by the formulation type and excipients utilized (tablets, capsules, immediate-release formulations), fasting or feeding condition, stomach residence time, and gastric motor activity. At the same time, the medication is dissolving. The dissolving process involves the movement of solute molecules from the solid phase to the liquid phase, which is represented by gastric juice. Again, physicochemical properties of the medication (e.g., particle size and polymorphisms) and physicochemical circumstances (e.g., gastric juice pH and viscosity) may influence this process.

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