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Exploring Orally Disintegrating Tablets, Sublingual Administration and Treatment of Peptic Ulcers: A Comprehensive Review

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ABSTRACT

This comprehensive review delves into the world of orally disintegrating tablets (ODTs), sublingual administration, and the treatment of peptic ulcers. It explores the formulation, mechanism of action, pharmacokinetics, and clinical uses of drugs in these areas. The review discusses the advantages and disadvantages of ODTs and sublingual administration, including their quick onset of action, high bioavailability, and convenience, as well as limitations such as restricted medication compatibility and patient compliance issues. Additionally, it examines the biological barriers encountered in the gastrointestinal tract and the various strategies employed to enhance drug absorption. Furthermore, the review delves into the treatment approaches for peptic ulcers, including eradicating H. pylori infection, reducing gastric acid secretion, and neutralizing gastric acid. Overall, this review provides a comprehensive overview of these important topics in pharmaceutical science and clinical medicine.

Keywords- Orally disintegrating tablets, sublingual administration, peptic ulcers, pharmacokinetics, drug absorption, formulation, clinical uses, H. pylori infection, gastric acid secretion.

INTRODUCTION

Orally disintegrating tablets (ODTs) are a kind of solid medication that dissolve quickly in the mouth when they come into contact with saliva. This makes them an excellent option for patients who have trouble swallowing. Super disintegrants such as crospovidone, croscarmellose, or sodium starch glycolate are included in these tablets to facilitate fast break down without the need for water, chewing, or whole-meal ingestion.[1-3]

Owing to its affordability, patient choice, simplicity of large-scale manufacture, and convenience, oral medicine is the most widely used method of drug delivery. Oral administration accounts for around 60% of established small-molecule therapeutic products that are commercially accessible and for 90% of pharmaceutical formulations intended for human use worldwide. Additionally, almost 84% of the most popular pharmaceutical medications are taken orally and are presently worth $35 billion, growing at a pace of 10% annually.[4]

When compared to alternative delivery methods including intravenous, subcutaneous, and intramuscular injections, as well as inhalation for asthma treatments, compliance with oral meds is often greater.[5] Moreover, medications taken orally have the ability to target specific areas of the gastrointestinal (GI) tract in order to treat pathological conditions locally. These conditions include infections, inflammations, bowel diseases, gastric and colorectal cancers, gastro-duodenal ulcers, and disorders related to gastroesophageal reflux.[6]

Notwithstanding these benefits, there are still a number of difficulties in creating oral formulations, which are mostly related to the physicochemical characteristics of medications, such as their low water solubility and membrane permeability.[7] Aside from physiological obstacles like pH, efflux transporters, and metabolic enzymes, poor chemical and biological stability of medications may also impede their absorption. A few medications may also induce nausea and localised discomfort.[8]

Understanding the mechanisms behind medication absorption and transport, intestinal transit, the GI tract's milieu, and drug stability in GI fluids has been the subject of many research conducted over the last 40 years. As a result, several methods have been developed to increase the solubility and rate of drug dissolution. These methods, such as solid

dispersions and self-emulsifying drug delivery systems (SEDDS), have shown promise in terms of enhancing patient compliance and drug bioavailability. [9-10]

Sublingual absorption is influenced by several factors:
Oral epithelium thickness: Compared to the buccal mucosa, the sublingual epithelium is thinner (100–200 μm), facilitating quicker medication absorption. This quick absorption is facilitated by the drug's immersion in a smaller amount of saliva and the thinner epithelium. Lipophilicity of the drug: Sublingually absorbed drugs should have a little greater lipid solubility than gastrointestinal ally absorbed drugs. Passive penetration via the mucosa requires this.[11]

Saliva's pH and pKa scale: Saliva's average pH is around 6.0, which is favourable for the absorption of medications that stay unionised at this pH. For the best absorption, the pKa of basic medications should be less than 10 while that of acidic pharmaceuticals should be more than 2.

Oil to water partition coefficient: Substances that have favourable coefficients of diffusion across the oral mucosa are more easily absorbed. 40–2000 is thought to be the ideal range for sublingual absorption.

Salivary secretion solubility: In addition to having a high solubility in fats, medications should also dissolve in watery buccal secretions. It takes a biphasic solubility profile to ensure effective absorption. Strong oral mucosal binding results in low systemic availability of the drug since the drug may not be released into the circulation.[12-13]
Advantages of Sublingual Administration:
Quick beginning of action: Bypassing the gastrointestinal system and the liver's first-pass metabolism, sublingual delivery enables quick medication absorption straight into the circulation. When compared to oral intake, this results in a faster beginning of effect.

High bioavailability: Sublingual medication avoids the hepatic first-pass impact by being absorbed straight into the systemic circulation. Higher bioavailability is the outcome as compared to oral delivery.

Convenience: Since sublingual delivery doesn't involve swallowing, it's a great option for individuals who have trouble swallowing or in circumstances where it's not practical to consume a pill.

Dose accuracy: Because the medicine is immediately absorbed under the tongue rather than being susceptible to the fluctuations in absorption that occur with oral administration, sublingual administration permits exact dosage.

Decreased chance of gastrointestinal side effects: Sublingual delivery lowers the chance of gastrointestinal adverse effects including nausea and vomiting by avoiding thegastrointestinal system.

Disadvantages of Sublingual Administration:
Restricted medication compatibility: Some pharmaceuticals may not be well absorbed via the sublingual mucosa or may taste bad, making them unsuitable for sublingual delivery.

Limited drug volume: The amount of medication that may be supplied by this method is restricted by the sublingual area's space for drug absorption.

Drug irritation: When taken sublingually, some medications may irritate or burn the tongue, which may make patients uncomfortable or noncompliant.

Drug stability: The kinds of medications that may be designed for sublingual delivery may be limited by the need that pharmaceuticals given sublingually remain stable in saliva and do not break down quickly.

Patient compliance: Holding the medication beneath the tongue for a certain amount of time during sublingual administration might be difficult for some patients, particularly young ones or those with cognitive disabilities.[14]

Biological Barriers
Before entering the circulation, drugs taken orally go through the gastrointestinal (GI) tract in a convoluted manner. Drugs may be absorbed via the oral mucosa of the mouth, especially in the sublingual area, where the process starts. But the majority of medications are mostly absorbed in the duodenum and jejunum, which are the higher sections of the GI tract.[15]

Due to its lower surface area and thicker mucus layer than the intestines, the stomach has a limited function in medication absorption. Although it acts as a significant barrier to absorption, the intestinal epithelial lining also helps because of certain features. In addition to acting as a barrier to hydrophilic molecules entering the cell, tight connections between enterocytes facilitate the paracellular route of drug absorption.

The intestine's apical surface epithelium extends into villi that house microvilli. Significantly more surface area is available for medication absorption and interaction because to these microvilli. Nevertheless, since their brush border is rich with digesting enzymes, they also provide an enzymatic barrier.[16]

In order to reach the mucosa, epithelium, and ultimately the blood or lymph capillary walls, medications must first pass through a number of layers, including the pericellular matrix, gastric juice, and the mucous-rich layer. Drug absorption may be enhanced using bio adhesive drug delivery devices, such bio adhesive microspheres, which diffuse into the mucous gel layer and have an extended stomach residence duration.

Drug absorption is also significantly influenced by the GI fluid's pH. The acidic pH of the stomach may have an impact on the stability of certain medications. Drugs that are unstable in an acidic environment need protection, which is often given via enteric polymer coatings that increase the pH of the stomach locally. The duodenum, on the other hand, has a neutral pH, which promotes better absorption of drugs.[17]

Active transport or passive diffusion are two ways that drugs might be absorbed. Drugs travel via a concentration gradient by passive diffusion, while some protein carriers are involved in active transport, which needs energy. tiny hydrophilic molecules can be absorbed by the paracellular route, whereas tiny drug molecules can only be absorbed via the transcellular route.[18]

Physicochemical Barriers

A number of physicochemical obstacles affect the complicated process of medication absorption in the gastrointestinal (GI) tract. In order for a medicine to be absorbed, it must first be liberated from the dosage form and be soluble, meaning it must dissolve in GI fluids. Based on intestinal epithelial membrane permeability and aqueous solubility, which are important factors in GI absorption, the Biopharmaceutical Classification System (BCS) classifies pharmaceuticals. Based on their solubility and permeability, medications are divided into four groups by the BCS. Class I pharmaceuticals are highly soluble and permeable, which makes them appropriate for oral administration. However, poor solubility, low permeability, or both provide problems for oral administration of Class II, III, and IV medicines. Drug metabolism may also affect oral bioavailability, which is why the Biopharmaceutics Drug Disposition Classification System (BDDCS) was created. The BDDCS sheds light on how food affects medication absorption as well as how absorption, excretion, and transport interact. Comprehending these physicochemical obstacles is essential for formulating medication administration schemes that maximise absorption throughout the gastrointestinal tract.[19-21]

Metabolic and Biochemical Barriers

The GI tract's metabolic and biochemical barriers have a substantial influence on the absorption and bioavailability of medications taken orally. Digestive enzymes released by the pancreas, including lipases, amylase, and peptidases like chymotrypsin and trypsin, are the main initiators of intestinal metabolism. These enzymes disassemble complicated compounds into absorbable, simpler ones. Enzymes from the intestinal flora, especially those found in the colon, also play a role in this process.[22]

On the surface of enterocytes, both intracellular and brush-border metabolism are a part of first-pass metabolism. Enzymes such as isomaltase, alkaline phosphatase, and peptidases are involved in brush-border metabolism, which mostly takes place in the small intestine. On the other hand, phase-I metabolising enzymes (such as cytochrome P450 enzymes like CYP3A4) and phase-II conjugating enzymes (such as sulfation and glucuronidation enzymes) are involved in intracellular metabolism, which takes place within enterocytes.[23]

Additionally, hepatic first-pass metabolism is an essential metabolic barrier for medications taken orally. Phase-I and phase-II reaction-related enzymes are involved in this metabolism, which takes place in the liver. Drug structures may be dramatically changed by the liver's metabolism, producing metabolites that may or may not be more or less pharmacologically active than the original drug. Membrane transporters are essential for the absorption, disposal, and bioavailability of drugs. Uptake transporters and efflux transporters are the two primary categories into which they may be divided. Drug transport into cells is facilitated by uptake transporters, which are mostly members of the solute carrier (SLC) superfamily. Drug bioavailability is decreased by efflux transporters, which are mostly members of the ATP-binding cassette (ABC) superfamily. They pump medicines out of cells. The gut and liver have high expression levels of efflux transporters, such as P-glycoprotein (P-gp), multidrug resistance-associated proteins (MRPs), and breast cancer resistance protein (BCRP), which help the body excrete medications.[24]

Drug for sublingual administration

Prodrug design, nanotechnology-based formulations, lipid-based formulations, pH modifiers, and enzyme inhibitors are some of the tactics used to enhance oral drug administration. By enhancing medication solubility, stability, permeability, and bioavailability, these strategies hope to improve therapeutic results.[25]

In cardiovascular medicine, sublingual drug administration is often used to provide drugs like nitro-glycerine for quick treatment of angina symptoms. It's also used for enzymes, steroids, and certain barbiturates. Because of their effective absorption beneath the tongue, a variety of vitamins and minerals may now be absorbed via this manner. Sublingual absorption circumvents the liver and gastrointestinal tract to provide immediate bloodstream access. Those with gastrointestinal problems like as ulcers, overactive stomach, or celiac disease, as well as those with impaired digestion, the elderly, and the disabled, would particularly benefit from this direct method. Regardless of gastrointestinal issues, sublingual feeding ensures that nutrients are absorbed and offers independent nutritional advantages.[26]

STRUCTURE OF THE HUMAN ORAL MUCOSA

Mucous membranes, which have a surface area of around 100 cm^, line the mouth cavity. Of the various components, the teeth make up around 20%, the keratinized epithelium about 50%, and the non-keratinized epithelium about 30% of this surface area. The floor of the mouth (sublingual area), buccal mucosa (line of the cheeks), gingiva (gum), palatal mucosa, and inner side of the lips are the five main regions that make up the oral mucosa.

In terms of structure, the oral mucosa is made up of a submucosa as the deepest layer, a foundation membrane, a lamina propria, and an outermost layer of stratified squamous epithelium. The epithelium is characterised by a mitotically active basal cell layer that advances through developing intermediate layers to the surface layers, where cells shed, resembling stratified squamous epithelia seen elsewhere in the body.

Site-specific variations in epithelial thickness include the buccal mucosa, which has a thickness of 500–800 µm, and the hard palate, floor of the mouth, ventral tongue, and gingiva, which have thicknesses of 100–200 µm. The epithelium's makeup differs as well; non-keratinized epithelium is found in the buccal, sublingual, and gingival areas, while keratinized epithelium is found in the gingiva and hard palate.

All things considered, the oral mucosa is a fairly permeable epithelium that is in between the intestinal mucosa and the epidermis in terms of permeability. The permeability varies depending on the area; buccal mucosa is more permeable than palatal mucosa, and sublingual mucosa is more permeable than both. The drug's pKa, the solution's pH, and the lipid-water partition coefficient all affect how much of the medication is absorbed via the oral mucosa.[27]

Stratified Squamous Epithelium: Consisting of many layers of flattened cells, stratified squamous epithelium is the outermost layer of the oral mucosa. This epithelium offers defence against microbiological invasion, mechanical injury, and chemical harm. It is non-keratinized in regions that need flexibility, like the buccal mucosa and floor of the mouth, and keratinized in parts that are exposed to significant mechanical stress, such the gingiva (gums) and hard palate.

Basement Membrane: The basement membrane, a thin layer of extracellular matrix that divides the epithelium from the connective tissue below, is located underneath the epithelium. In addition to facilitating the movement of nutrients and waste products between the epithelium and the underlying tissue, it offers structural support.

Laminapropria: Beneath the basement membrane lies a layer of loose connective tissue called the lamina propria. It is made up of neurones, blood vessels, and a variety of cell types, including as lymphocytes, macrophages, and fibroblasts. Due to its abundance of immune cells that aid in infection defence, the lamina propria is vital to the immunological response.

Muscularis Mucosae: The muscularis mucosae is a thin layer of smooth muscle that lies underneath the lamina propria and facilitates movement inside the mucosa. The tongue and lips, two regions of the oral mucosa that need for a lot of mobility, have a particularly well-developed layer of tissue.

Submucosa: The submucosa, a layer of thick connective tissue that includes bigger blood arteries, lymphatic vessels, and salivary glands, is one of the deeper layers of the oral mucosa. In addition to providing extra support, the submucosa aids in preserving the mucosa's structural integrity.

Minor Salivary Glands: The oral mucosa contains several small salivary glands that release saliva into the mouth cavity. These glands assist in moistening the mucosa, facilitating mastication and deglutition, and aiding in the process of digestion.

Ulcer

Gastric Ulcer: A gastric ulcer is a peptic ulcer that specifically develops in the stomach lining. It is often linked to an excess of gastric acid and harm to the stomach lining's protecting mucous layer. Gastric ulcers may arise from several circumstances, such as infection with the bacteria Helicobacter pylori, prolonged usage of nonsteroidal anti-inflammatory medicines (NSAIDs) like aspirin or ibuprofen, excessive alcohol intake, smoking, and stress.

Duodenal Ulcer: Another kind of peptic ulcer that develops in the duodenum, the first segment of the small intestine, is called a duodenal ulcer. It is the most typical kind of stomach ulcer. The common causes of duodenal ulcers are prolonged NSAID usage or H. pylori bacterial infection. Weight loss, nausea, vomiting, bloating, and stomach discomfort are all possible signs of duodenal ulcers.

Esophageal Ulcer: The lining of the oesophagus, the tube that transports food from the mouth to the stomach, may develop an uncommon kind of ulcer called an oesophageal ulcer. Gastric reflux disease (GERD), in which stomach acid refluxes back into the oesophagus, irritating and inflaming the lining of the oesophagus, is often linked to oesophageal ulcers. Oesophageal ulcers may also result from trauma, infection, and certain drugs.

Meckel's Diverticulum Ulcer: Meckel's diverticulum is a small intestinal anomaly that is congenital, meaning it exists from birth. The ileum is the name for the little sac or pouch that emerges from the wall of the small intestine's bottom portion. An ulcer that develops in the lining of Meckel's diverticulum may sometimes cause symptoms including anaemia, rectum bleeding, and abdominal discomfort. Although uncommon, untreated Meckel's diverticulum ulcers may lead to major problems.[27-29]

Signs and symptoms of chronic peptic ulcers may vary and include both duodenal and stomach ulcers. The sores or lesions in the stomach lining (gastric ulcers) or the first segment of the small intestine (duodenal ulcers) are the characteristic features of these ulcers.

Some common signs and symptoms of chronic peptic ulcers include:

Abdominal Pain: Usually felt as a searing or gnawing pain in the abdomen, usually between the breastbone and the navel, this is the most prevalent symptom. Though it may happen at any moment, the discomfort is usually stronger when the stomach is empty and can be eased by eating or using antacids.

Indigestion: Also known as dyspepsia, indigestion can manifest as bloating, belching, and feelings of fullness or discomfort in the upper abdomen.

Heartburn: A burning sensation in the chest, often after eating or at night, is a common symptom of gastric reflux, which can accompany peptic ulcers.

Nausea and Vomiting: Some individuals with peptic ulcers may experience nausea, which can occasionally lead to vomiting.

Unintended Weight Loss: Chronic peptic ulcers can sometimes lead to a decreased appetite and subsequent weight loss.

Dark, Tarry Stools: Bleeding from a peptic ulcer can result in the passage of dark, tarry stools, indicating the presence of partially digested blood (melena).
Vomiting Blood: In severe cases, bleeding from an ulcer can lead to the vomiting of blood, which appears as red or black material.

Fatigue: Chronic blood loss from a peptic ulcer can result in iron deficiency anemia, leading to fatigue and weakness.

Drug Used to Treat Peptic Ulcer Disease
Medication Used to Treat Ulcerative Pneumonia While the exact cause of peptic ulcer disease remains unknown, a number of significant contributing factors have been identified, including the use of non-steroidal anti-inflammatory drugs (NSAIDs), gram-negative Helicobacter pylori infection, increased secretion of hydrochloric acid, and insufficient mucosal defence against gastric acid.

Treatment approaches for chronic peptic ulcers: including gastric and duodenal ulcers, involve several strategies aimed at addressing the underlying causes and symptoms.

Eradicating H. pylori infection: Peptic ulcers, especially duodenal ulcers, are mostly caused by these bacteria. Proton pump inhibitors (PPIs) and antibiotics (including amoxicillin, clarithromycin, and metronidazole) are often used in conjunction to treat the illness.

Reducing gastric acid secretion: Proton pump inhibitors (PPIs) are often used to lower stomach acid production, aiding in ulcer healing and averting recurrence. Omeprazole, lansoprazole, and esomeprazole are a few PPIs.

Protecting the gastric mucosa: Misoprostol and sucralfate are two examples of medications that may shield the duodenum and stomach lining, accelerating healing and lowering the chance of problems.

Neutralizing gastric acid: Calcium carbonate, magnesium hydroxide, and aluminium hydroxide are examples of antacids that may help neutralise stomach acid and relieve pain and discomfort.

Lifestyle and dietary changes: Steer clear of irritants such as alcohol, smoke, and nonsteroidal anti-inflammatory medicines (NSAIDs) to help stop more stomach lining damage. It could also be advantageous to eat smaller, more frequent meals and stay away from acidic or spicy foods.

Monitoring and follow-up: It's critical to do routine monitoring and follow-up visits with a healthcare professional in order to evaluate the efficacy of therapy and handle any issues or consequences.

1. **Reduction of gastric acid secretion**
   - **H2-histamines receptor blockers:** Cimetidine, Ranitidine,Famotidine,Roxatidine.
   - **Proton pump inhibitors:** Omeprazole, Lansoprazole, Rabeprazole,Rabeprazole, Esomeprazole.
   - **Anticholinergics:** Pirenzepine, Propantheline,Oxyphenonium.
   - **Prostaglandin analogue:** Misoprostol.
   - **Anti-muscarinic agents:** Hyoscyamine,Mepenzolate, Pirenzepine
2. **Neutralization of gastric acid (antacids)**
   - **Systemic antacids:** Sodium bicarbonate, Sodiumcitrate.
   - **Non-systemic antacids:** Magnesium hydroxide, Calcium carbonate, Magnesium trisilicate, Aluminium hydroxide gel.
3. **Mucosal protective agents:** Sucralfate, Colloidalbismuth subrostrate.
4. **Anti-helicobacter pylori drugging-microbial agents:** Amoxicillin, Tinidazole, Tetracycline, Metronidazole, Bismuth compound.

Proton pump inhibitors

A family of drugs known as proton pump inhibitors (PPIs) lowers the production of gastric acid. They function by preventing the stomach's parietal cells, which secrete acid, from using the proton pump (H+/K+ ATPase enzyme). PPIs successfully decrease the quantity of acid generated by the stomach by inhibiting this pump, which helps treat ulcers and soothe the symptoms of GERD and other acid-related illnesses. PPIs are often used to treat disorders including Zollinger-Ellison syndrome, GERD, and peptic ulcers. Pantoprazole, rabeprazole, lansoprazole, esomeprazole, and omeprazole are a few PPI examples. These drugs are useful for reducing stomach acid and relieving associated symptoms since they are often given orally and have effects that persist for many days.

Regulation of gastric acid secretion

The intricate process of controlling stomach acid secretion involves several cells, hormones, and signalling channels. The parietal cell, which is found in the stomach lining, is the main regulator of gastric acid output. Hydrochloric acid (HCl), which is secreted into the stomach lumen by parietal cells, is necessary for the breakdown of ingested pathogens and for the digestion of food, particularly proteins.

The secretion of gastric acid is regulated by several factors:

Histamine: Histamine, released from enterochromaffin-like (ECL) cells in response to gastrin or acetylcholine, binds to histamine-2 receptors (H2 receptors) on parietal cells, stimulating the production of acid.

Gastrin: Gastrin is a hormone released by G cells in the stomach in response to the presence of food. Gastrin stimulates the release of histamine from ECL cells and directly stimulates parietal cells to produce acid.

Acetylcholine: Acetylcholine, released from nerve endings in the stomach (vagal stimulation) and from enteric neurons, stimulates parietal cells directly and also indirectly by stimulating the release of histamine from ECL cells.

Prostaglandins: Prostaglandins, such as prostaglandin E2 (PGE2), play a protective role in the stomach by inhibiting acid secretion, stimulating the production of mucus and bicarbonate, and promoting blood flow to the stomach lining.

Somatostatin: Somatostatin, produced by D cells in the stomach and pancreas, inhibits the release of gastrin, histamine, and acetylcholine, thereby reducing acid secretion.

Inhibitory neurotransmitters: Neurons in the enteric nervous system release neurotransmitters like nitric oxide and vasoactive intestinal peptide (VIP), which inhibit acid secretion.

Other factors: Factors such as mechanical distension of the stomach, the presence of certain peptides and amino acids in the stomach, and the pH of the gastric contents can also influence gastric acid secretion.

Examples of proton pump inhibitors: Clinically usedproton pump inhibitors

- Omeprazole
- Lansoprazole
- Esomeprazole
- Rabiprazole
- Rabeprazole

Clinical uses
These medications are used to treat a variety of conditions, including dyspepsia, peptic ulcer disease, Barrett's oesophagus, gastro-oesophageal reflux disease (GERD), Zollinger-Ellison syndrome, prevention of stress gastritis, gastrointestinal disorders, and other conditions that result in excessive acid production.

Mechanism of action
Proton pump inhibitors (PPIs) function by permanently inhibiting the H+/K+ ATPase enzyme system in stomach parietal cells, which is also referred to as the gastric proton pump. By blocking the last stage of stomach acid secretion, this action efficiently lowers the amount of acid produced. PPIs are more efficient than H2 antagonists in lowering stomach acid output by up to 99% because they target the final phase of acid formation and have an irreversible inhibitory effect. This decrease in acidity relieves symptoms like heartburn and indigestion while also accelerating the repair of duodenal ulcers. Hypochlorhydria, on the other hand, may result from decreased stomach acid levels and may impact the absorption of nutrients, especially calcium. When PPIs are injected, they are inert. However, when they get into contact with acidic environments, such the parietal cell canaliculus, they become active and may stop the proton pump.[28-30]

Lansoprazole sodium
Proton pump inhibitors (PPIs) are a family of drugs that reduce the stomach's production of gastric acid. Lansoprazole sodium is a member of this pharmacological group. These medications are used to treat a number of acid-related illnesses, such as Zollinger-Ellison syndrome, peptic ulcers, and gastroesophageal reflux disease (GERD).

By resisting variations in acidity or alkalinity, buffers are chemicals that aid in maintaining the pH of a solution. Since lansoprazole sodium works on the stomach's proton pump to decrease acid production, it indirectly affects the pH balance of the stomach as a whole. Lansoprazole sodium is not a buffer in and of itself.[31-32]

Mechanism of Action
The mechanism of action of lansoprazole sodium is attributed to its capacity to permanently block the stomach parietal cells' H+/K+ ATPase, an enzyme system that catalyses the breakdown of amino acids. Gastric acid generation requires the pumping of hydrogen ions into the stomach lumen, which is accomplished by this enzyme system. Lansoprazole sodium lowers the amount of acid secreted into the stomach by blocking this enzyme system, which raises pH and lowers acidity.

Oral administration of lansoprazole sodium is common, and it comes in the form of delayed-release tablets or capsules. Following absorption into the circulation, it builds up in the parietal cells that line the stomach, where it inhibits the proton pump. As a consequence of this inhibition, stomach acid output is sustainedly reduced, relieving symptoms related to disorders including GERD and peptic ulcers.

Several characteristics of Lansoprazole sodium pharmacologic profile distinguish it from other PPIs.
Similar to other proton pump inhibitors (PPIs), lansoprazole sodium is useful in treating conditions linked to acid production in the stomach, including GERD and peptic ulcers. However, lansoprazole sodium differs from other PPIs in a few ways:

Pharmacokinetic Properties: Peak plasma levels of lansoprazole sodium are attained one to two hours after dosing, indicating a quick beginning of action. It also provides continuous acid suppression over a comparatively lengthy period of time.

Metabolism: The liver uses the cytochrome P450 enzyme system, particularly CYP2C19 and CYP3A4, to metabolise lansoprazole sodium. Individual differences in medication response may result from this metabolic route, especially in people with genetic variants in these enzymes.

Potency: Lansoprazole sodium is considered to be one of the more potent PPIs, providing greater acid suppression compared to some other PPIs.

Duration of Action: Lansoprazole sodium has a longer duration of action compared to some other PPIs, allowing for once-daily dosing in many patients.

Indications: A number of acid-related conditions, such as GERD, erosive esophagitis, duodenal ulcers, and gastric ulcers, are authorised for treatment with lansoprazole sodium. It's a flexible alternative for acid suppression treatment because of its wide variety of uses.

Side Effects: Although the side effect profiles of all PPIs are similar, there may be individual variations in tolerance. Although lansoprazole sodium is usually well tolerated, some people may have adverse symptoms including headache, diarrhoea, or stomach discomfort.

Cost: Lansoprazole sodium may be more expensive depending on the brand and formulation, but overall, it is less expensive than certain other PPIs, making it a more sensible choice for some people.

Lansoprazole Sodium: A proton pump inhibitor (PPI) called lansoprazole sodium is used to lessen the production of stomach acid. It is often used to treat ailments including ulcers, Zollinger-Ellison syndrome, and gastroesophageal reflux disease (GERD).

Hardness: The term "hardness" describes the tablet's resistance to pressure or mechanical stress. The hardness of lansoprazole sodium tablets guarantees their integrity during handling and transit, hence guaranteeing patient safety and dosage consistency.

Thickness: The thickness of a tablet is its breadth or depth measured. Lansoprazole sodium tablets must be uniformly thick to provide consistency and patient comfort during swallowing..

Friability: A tablet's propensity to fracture or crumble under mechanical force is measured by its friability. Low friability is preferable for lansoprazole sodium tablets in order to guarantee that the tablets hold their shape and provide the right amount of the active component.

Drug Content: The term "drug content" describes how much lansoprazole sodium, the active pharmaceutical component, is included in each tablet. For patients to obtain the prescribed dosage, lansoprazole sodium tablets must have a constant medication content.

Wetting Time: The length of time it takes for a tablet to get completely submerged in a given volume of water is known as the wetting time. Wetting time is crucial for lansoprazole sodium tablets in order for the tablet to break down and release the active component for absorption.

In-vitro Disintegration Time: The amount of time that a tablet takes to disintegrate into smaller pieces in a liquid media is known as the "in-vitro disintegration time." The disintegration time of lansoprazole sodium tablets is crucial for the efficient release of the active component for absorption.

In-vitro Dissolution Studies: The pace at which the active component, lansoprazole sodium, is released from the tablet and dissolves in a liquid media is measured by in-vitro dissolution experiments. These investigations are critical to determining how well the tablet delivers the active component to the intended location.

Stability Studies: Stability tests assess how lansoprazole sodium tablets hold up over time and in different environmental settings in terms of their physical, chemical, and microbiological stability. The results of these experiments are crucial in establishing the tablets' shelf life and storage needs.

Importance of stability studies

Shelf-Life Determination: Stability studies are useful in estimating the duration ofpharmaceutical items' shelf life, such as the tablets of lansoprazole sodium. To make sure the pills are safe and effective to use until their expiry date, this information is essential.

Formulation Optimization: Stability studies are useful in determining the variables, such as temperature, humidity, and packaging, that impact the stability of lansoprazole sodium tablets. By using this data, the tablet composition may be improved to increase stability.

Regulatory Compliance: Stability data are required by regulatory bodies in order to approve new pharmaceutical medicines. Stability studies and data submission show that the product satisfies safety and effectiveness standards set by regulations.

Quality Control: For lansoprazole sodium tablets to remain high-quality during distribution and storage, stability studies are crucial. Manufacturers can make sure the pills fulfil quality requirements and provide patients constant dose by tracking stability over time.

Purpose of stability studies

Stability studies are carried out to assess how different environmental conditions affect the quality of a pharmaceutical product over time, such as lansoprazole sodium tablets. These investigations seek to ascertain the product's shelf life, pinpoint ideal storage settings, and evaluate how packaging affects stability. Stability studies monitor the product's physical, chemical, and microbiological characteristics to make sure it stays safe, effective, and compliant with regulations for the duration of its planned shelf life. Maintaining product quality, assisting with regulatory filings, and guaranteeing patient safety and medicine trust all depend on this data.[33]

CONCLUSION

In conclusion, this comprehensive review explores the intricacies of orally disintegrating tablets (ODTs), sublingual administration, and the treatment of peptic ulcers. It delves into the pharmacokinetics, formulation strategies, and clinical applications of these drug delivery methods. Highlighting the advantages and disadvantages of sublingual administration, along with the challenges in developing oral formulations, the review underscores the importance of understanding drug absorption mechanisms, gastrointestinal barriers, and physicochemical properties for optimizing drug delivery. Moreover, it discusses the significance of addressing H. pylori infection and regulating gastric acid secretion in the management of peptic ulcers. Overall, this review provides valuable insights into current trends and strategies in oral drug delivery and ulcer treatment, laying the groundwork for further research and development in these areas.

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