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A COMPREHENSIVE REVIEW ON SODIUM ALGINATE BEADS FOR PEPTIC ULCER MANAGEMENT

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ABSTRACT

Peptic ulcer disease (PUD) is a prevalent gastrointestinal disorder characterized by mucosal injury due to excessive gastric acid and pepsin secretion. Affecting approximately 10% of the global population, PUD is a chronic condition with significant morbidity. Helicobacter pylori (H. pylori) infection and nonsteroidal anti-inflammatory drugs (NSAIDs) are the primary causes of PUD. However, eradicating H. pylori infections poses significant challenges, including poor drug accessibility and short gastric residence times. Sodium alginate beads have emerged as a promising drug delivery system, offering regulated release and improved bioavailability. This review provides a comprehensive overview of recent advancements in sodium alginate bead formulation and evaluation methodologies. The integration of innovative formulation strategies and precise evaluation techniques has significantly enhanced the performance and functionality of sodium alginate beads, paving the way for their diverse applications in drug delivery and tissue engineering.

KEYWORDS: Peptic Ulcer, Helicobacter pylori, Sodium Alginate Beads.

1. INTRODUCTION

Peptic ulcer disease is a gastrointestinal issue that causes mucosal injury as a result of pepsin and stomach acid secretions. It typically occurs in the stomach and proximal duodenum, but it

can also occur in the lower oesophagus, distal duodenum, or jejunum in unopposed hypersecretory states like Zollinger-Ellison syndrome, hiatal hernias (Cameron ulcers), or ectopic gastric mucosa (e.g., Meckel's diverticulum).[1]

Peptic ulcer disease is caused by an imbalance between substances that protect and destroy the mucosa of the stomach and duodenum. Patients with gastric and duodenal ulcers present similarly. Patients may experience epigastric or retrosternal pain, early satiety, nausea, bloating, burp, or postprandial misery. These symptoms are nonspecific and may be difficult to distinguish clinically from functional dyspepsia.[2]

Helicobacter pylori, a Gram-negative bacterium, lives between the mucous layer and the gastric epithelium and is specifically suited to survive in the stomach's harsh environment. Helicobacter pylorus is initially found in the antrum, but it eventually migrates to the stomach's more proximal regions.

Peptic ulcer is one of the world's most common gastrointestinal illnesses, impacting 10% of the global population. Duodenal peptic ulcers account for approximately 19 out of every 20 cases. Every year, an estimated 15000 people die from peptic ulcers. The annual incidence rates of peptic ulcer bleeding and perforation were 19.4-57 and 3.8-14 per 100,000 people, respectively. The average 7-day recurrence of bleeding was 13.9%, while the average long term recurrence of perforation was 12.2%.[3] Ulcer Index Ulcers were scored as follows: no ulcer = 0, superficial ulcer = 1, deep ulcer = 2, and perforation = 3. The mean ulcer score for each animal will be reported as an ulcer index.[4]

TYPES OF PEPTIC ULCER

1) Gastric Ulcer

Gastric" is a word that means "related to the stomach.

Gastric ulcers, as the name implies, are found in the stomach. They can occur when stomach acid used to digest food irritates the stomach lining, resulting in an open sore.

2) Duodenal Ulcer

After leaving the stomach and passing through the digestive tract, food enters the duodenum. This is the first section of the small intestine, which continues the digestion process. Peptic ulcers that form on the lining of the duodenum are known as duodenal ulcers.[5]

EPIDERMIOLOGY

The lifetime risk of getting a peptic ulcer range from 5% to 10%, with a rate of 0.1% to 0.3% each year. Peptic ulcers caused 301,000 deaths in 2013, a decrease from 327,000 in 1990. In Western countries, the percentage of people infected with H. pylori roughly corresponds to their age. Prevalence is highest in third-world nations, where it is estimated to be 70% of the Population, whereas wealthy countries have a maximum ratio of 40%.

Overall, H. pylori infections are decreasing worldwide, especially in wealthy countries. Food, contaminated groundwater, or human saliva are all possible routes of transmission.

Peptic ulcer disease had a significant impact on morbidity and mortality until the late twentieth century, when epidemiological patterns began to indicate an astonishing decrease in its frequency. The introduction of new effective medications and acid suppressants, as well as the reasonable use of nonsteroidal anti-inflammatory drugs (NSAIDs), are likely to have contributed to the decline in peptic ulcer disease rates.[6]

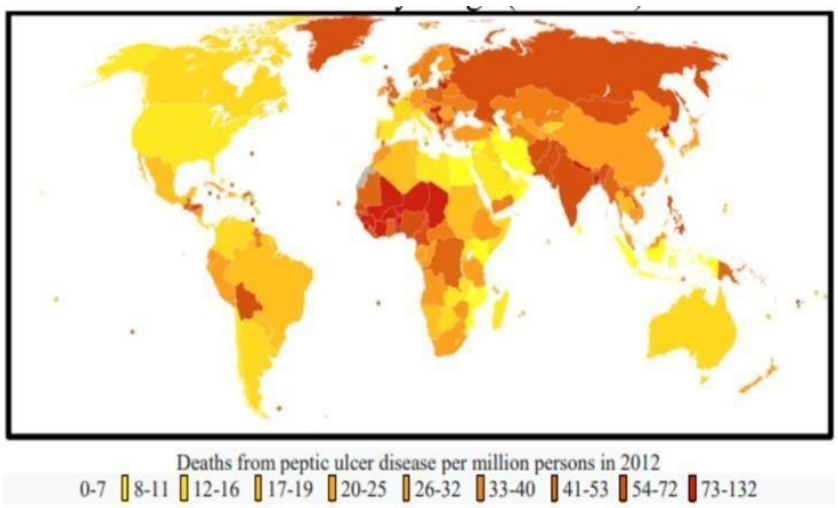

Fig 1: World map shows the area of spread peptic ulcer.[7]

ETIOLOGY

Although previous studies have indicated that seasonal variation did influence the incidence of Perforated Peptic Ulcer (PPU), other studies have failed to prove such a pattern. Patients in developing nations are often young male smokers, whereas patients in developed countries are elderly with several co-morbidities and a history of nonsteroidal anti-inflammatory medications (NSAIDs) or steroids.

NSAIDs, H. pylori, physiological stress, smoking, corticosteroids, and a history of Peptic

ulcer disease (PUD) are all risk factors for PPU. In the presence of risk factors, ulcer recurrence is common, even after first effective treatment. A thorough assessment of 93 research found that the average long-term recurrence of perforation was 12.2%.

- **NSAIDS**

NSAIDs are widely used for its analgesic, anti-inflammatory and anti-pyretic effects. NSAID use is known to increase the risk of PPU.

About a quarter of chronic NSAID users will develop PUD, with 2%-4% bleeding or perforation. Drug interactions with steroids and selective serotonin reuptake inhibitors also raise the risk of PUD. Selective cyclooxygenase-2 inhibitors have a lower risk of developing PUD.

- **H. pylori**

H. pylori remains one of the most frequent infections worldwide. H. pylori prevalence has decreased in developed nations as a result of improved hygiene and reduced transmission during early life. The mean prevalence of H. pylori in PPU patients varies across research due to differences in diagnostic methodologies and geographical location. Recent investigations employing histological methods of H. pylori detection have showed that H. pylori prevalence in individuals with perforated duodenal ulcers ranges between 50% and 80%.

Recurrent PUD is most common in individuals with H. pylori infection, indicating that H. pylori plays a key role in the development of PUD and related consequences. Proton pump inhibitor medication dramatically reduces the incidence of recurrent H. pylori infection, although it has only little efficacy in reducing ulcers among NSAID users.

- **Smoking**

Tobacco is thought to inhibit pancreatic bicarbonate secretion, leading to increased acidity in duodenum.

It also hinders the healing of duodenal ulcers. According to a meta-analysis, smoking may be responsible for 23% of PUD. However, in some studies, there was no difference in tobacco usage between individuals with non-H. pylori, non-NSAID duodenal ulcers and those with H. pylori-related ulcers, implying that smoking plays a limited effect. This is consistent with prior research, which found that smoking did not raise the risk of ulcer recurrence after H. pylori had been eradicated.

- **Others**

Alcohol use is known to damage the stomach mucosa and increase gastrin release. Despite the immediate effects, there is no evidence that drinking causes PUD. Zollinger-Ellison syndrome (ZES) is caused by a pancreatic gastrin-secreting tumour that induces parietal cells in the stomach to produce more acid, resulting in gastrointestinal mucosal ulceration. Over 90% of individuals with ZES develop peptic ulcers, which are often resistant to proton pump inhibitor therapy. ZES should be considered in individuals who have multiple or refractory peptic ulcers, jejunal ulcers, a family history of PUD, and concomitant diarrhoea. All ZES patients should be examined for Multiple Endocrine Neoplasia 1 (MEN1) syndrome.[8]

SYMPTOMS

Many people with peptic ulcers don't have symptoms. If there are symptoms, they may include.

- Dull or burning stomach pain. For some people, pain may be worse between meals and at night. For others, it may be worse after eating.
- Feeling of fullness or bloating.
- Belching.
- Heartburn.
- Nausea.

Peptic ulcers can cause bleeding from the ulcer. Then symptoms might include:

- Vomiting blood, which may appear red or black.
- Having dark blood in stools, or stools that are black or tarry.
- Feeling dizzy or fainting.[9]

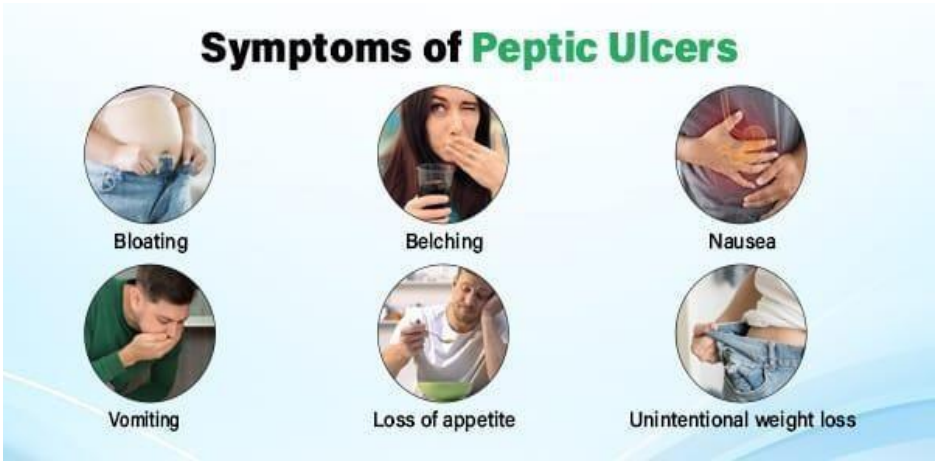

Fig 2: Symptoms of Peptic Ulcers.[10]

RISK FACTORS

- Helicobacter pylori
- NSAIDs
- Past gastric ulcer and family history
- Stress and diet
- Smoking
- Alcohol[11]

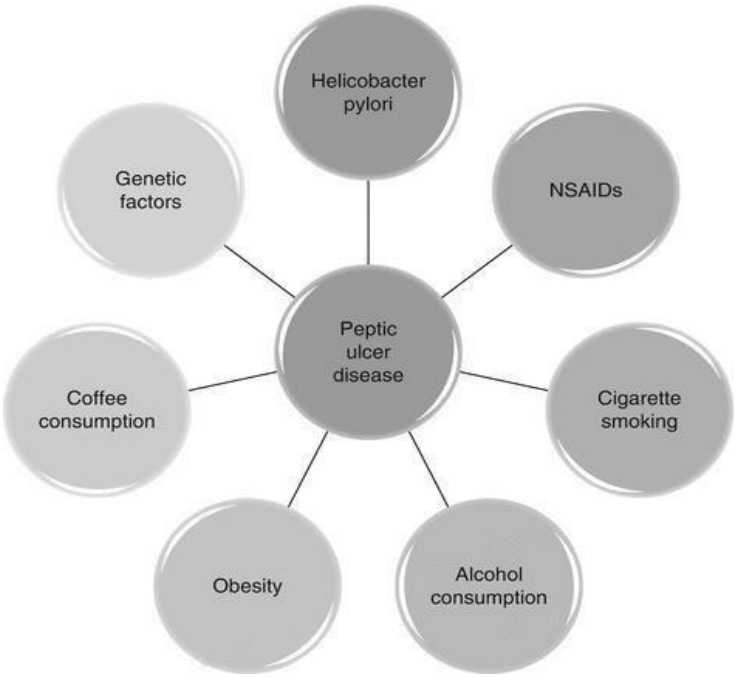

Fig 3: Risk factors of Peptic Ulcer.[12]

STAGES OF PEPTIC ULCER

1. Acute Stage

Acute peptic ulcer disease symptoms are characterized by fast onset, evident manifestation, and rapid progression. At this point, if diagnosed and treated correctly, the condition can be totally healed. However, most people frequently disregard the symptoms and do not go to the doctor, making the disease more complicated.

2. Chronic Stage

If acute peptic ulcer disease is not treated, it will cause inflammation and swelling for a long period and may eventually progress to chronic form. In the chronic stage, the lesions spread, the condition becomes more difficult to cure, and can potentially result in hazardous complications such as atrophic inflammation, intestinal metaplasia, pyloric stenosis, bleeding,

perforation, and gastric cancer, as well as infection of surrounding organs.[13]

DIAGNOSTIC TEST FOR PEPTIC ULCER

Blood test Doctors may utilize blood tests to look for evidence of H. pylori infection or peptic ulcer complications. For an NIH blood test, a health care provider will collect a blood sample from you and send it to a lab.

- **Urea breath test**

To diagnose H. pylori infection, doctors may do a urea breath test. You will swallow a capsule, drink, or pudding containing urea that has been "labelled" with a particular carbon atom for the test. If H. pylori is present, the bacteria will convert urea to CO2. After a few minutes, you'll breathe into a container and exhale CO2. A health care specialist will evaluate your exhaled breath. If the test finds the labelled carbon atoms, your doctor will diagnose an H. pylori infection in your digestive tract.

- **Stool test Doctors**

Stool tests may be used to diagnose H. pylori infection. Your doctor will give you a container to collect and store a stool sample. You will receive instructions on where to submit or bring the kit for testing.

- **Upper gastrointestinal**

Upper GI endoscopy and biopsy: A doctor uses an endoscope, a flexible tube with a camera, to examine the lining of your upper GI tract, which includes your oesophagus, stomach, and duodenum. During upper GI endoscopy, a clinician takes biopsies by putting a tool through the endoscope and extracting small pieces of tissue from the stomach lining. A pathologist will analyse the tissue using a microscope.[6]

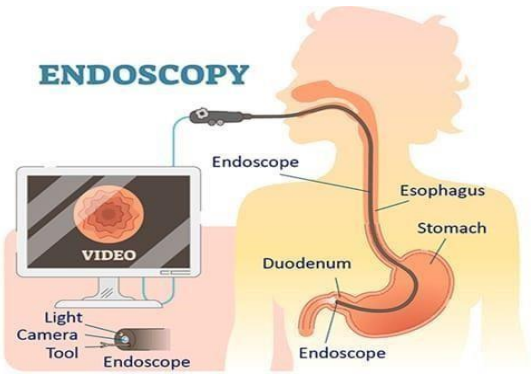

Fig 4: Upper GI endoscopy.[14]

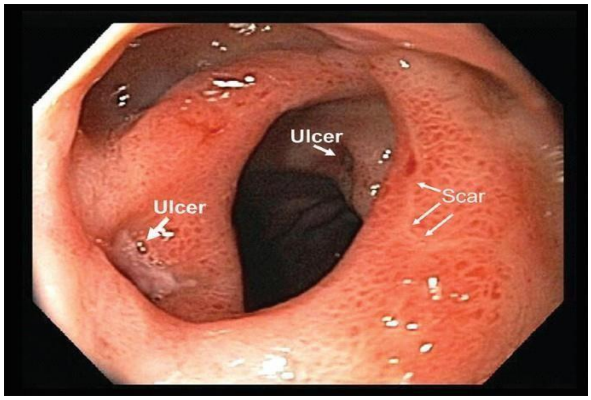

Fig 5: Photograph of peptic ulcer taken during an upper endoscopy.[15]

COMPLICATIONS

1. Obstruction

Pyloric stenosis is caused by the formation of a fibrous scar at or near the pylorus, while duodenal stenosis is caused by healed duodenal ulcers, and fibrosis and contraction from healed gastric ulcers along the smaller curvatures can induce a 'hourglass' deformity.

2. Haemorrhage

Minor bleeding from the erosion of small blood vessels at the base of an ulcer occurs in all ulcers and can be diagnosed by analysing the stool for occult blood. Chronic blood loss may cause iron deficiency anaemia. Severe bleeding may result in 'coffee ground' vomitus or Melania. A penetrating chronic ulcer can erode a major artery (for example, the left gastric, gastroduodenal, or splenic artery), resulting in extensive and severe haematemesis and, in some cases, death.

3. Perforation

A perforated peptic ulcer is a severe abdominal emergency. Perforation is more common in chronic duodenal ulcers than in chronic gastric ulcers. The following consequences may occur.

i. Upon perforation, the contents leak into the smaller sac or the peritoneal cavity, resulting in acute peritonitis.
ii. Air escapes from the stomach and settles between the liver and the diaphragm, creating the typical radiological picture of air beneath the diaphragm.
iii. Infection can cause a subphrenic abscess between the liver and diaphragm.
iv. Perforation may spread to nearby organs such as the liver and pancreas.

4. Malignant Transformation

Most peptic ulcers follow the maxim that "cancers ulcerate, but ulcers rarely cancerized". A chronic duodenal ulcer never becomes malignant, but less than 1% of chronic gastric ulcers develop into cancer.[16]

TREATMENT

The purpose of peptic ulcer disease treatment is to alleviate symptoms, heal craters, prevent recurrences, and avoid complications.[6]

Table 1: Allopathic Medication for Peptic Ulcer.

Type of peptic ulcer medicine	Name of medicine	Mechanism of action of peptic ulcer medicine	Side effects of medicines
1. Proton- pump inhibitor medicine	Omeprazole, Lansoprazole, Pantoprazole	The mechanism involves inhibiting the H+-K- ATPase channel, which consists of the movement of the ions.	Headache, pain in the abdomen, vomiting, vitamin deficiency, and flatulence
2. Histamine receptor-inhibiting medicines	Cimetidine, Famotidine, Ranitidine	Inhibits the secretion of histamine in the parietal cells lining the digestive system.	The side or adverse effects are anxiety, depression, and dizziness.
3. Antacid treatment medicines	Use of aluminium hydroxide and magnesium hydroxide powder.	Aluminium hydroxide blocks the action of the pepsin enzyme. Magnesium hydroxide causes the retention of osmotic fluid in the body.	The side effects are vomiting, bad mouth taste, cramping in the abdomen, and imbalance of electrolytes.
4. Protective medicines	Misoprostol and Sucralfate	Enhances the secretion of the mucous fluid and augments the development of the lining of the gastrointestinal tract.	The side effects are pain in the head, pain in the body, and pain in the abdomen.

PREVENTION AND CONTROL

Certain lifestyle choices and habits can reduce your risk of developing peptic ulcers. These include.

- Not mixing alcohol with medication.
- Not drinking more than two alcoholic beverages a day.
- Washing your hands frequently to avoid infections.
- Limiting your use of ibuprofen, aspirin, and naproxen.

Maintaining a healthy lifestyle by quitting smoking cigarettes and other tobacco use and eating a balanced diet rich in fruits, vegetables, and whole grains will help you prevent

developing a peptic ulcer.[17]

INTRODUCTION TO SODIUM ALGINATE BEADS

Sodium alginate beads are spherical gel particles made from a natural polymer that are used to encapsulate and control the release of other substance. They are formed when a solution of sodium alginate is dropped into a solution containing calcium ions, causing the ions to cross link the alginate chains and form a gel.[18]

Sodium alginate beads are widely used in pharmaceutical and biological industries due to their regulated drug delivery capabilities and diverse uses. These sodium alginate beads, derived from brown algae, are biocompatible, biodegradable, and may create hydrogels with divalent cations, notably calcium ions. The gel matrix encapsulates pharmaceuticals or bioactive chemicals and allows for controlled release over time.[19]

PURPOSE OF THE STUDY

Bead formulation is crucial for the optimal performance of sodium alginate-based drug delivery systems. The formulation process determines key characteristics such as bead size, shape, drug loading capacity, and release kinetics, which strongly impact drug delivery system performance and effectiveness. Customizing formulation characteristics can enhance release patterns, stability, bioavailability, and lessen potential negative effects. Fine-grained control over bead properties allows drug delivery systems to be adjusted to specific therapeutic demands, leading to improved patient compliance and treatment outcomes.[20]

OVERVIEW OF SODIUM ALGINATE BEADS

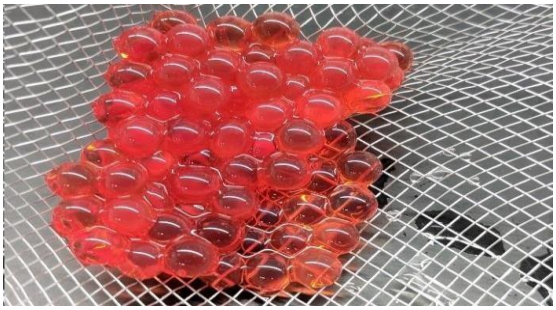

Fig 6: Sodium Alginate Beads.[21]

Sodium alginate beads are created by crosslinking sodium alginate polymer chains, resulting in spherical or irregular shapes. The technique creates a porous structure in the beads, enabling the trapping of medicines or medicinal substances. Sodium alginate beads are

versatile due to their unable features, allowing for controlled medication release, protection against degradation, and targeted administration to specific body regions.[15]

CHEMICAL STRUCTURE

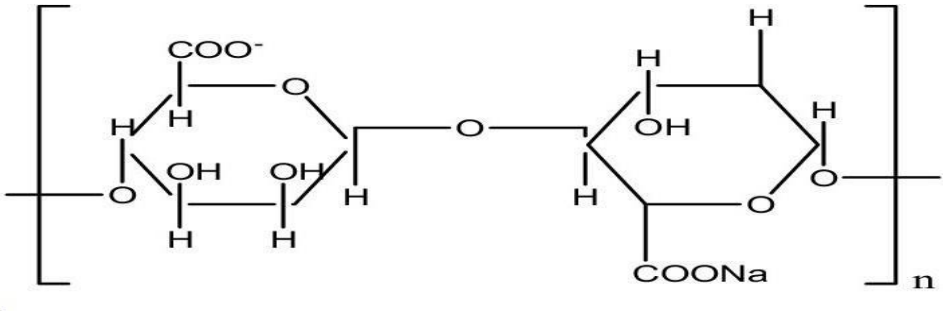

Fig 7: Chemical structure of the sodium alginate molecule.

Sodium alginate is a linear polysaccharide made up of repeated units of β-D-mannuronic acid (M) and α-L-guluronic acid (G), linked by 1,4-glycosidic bonds. The distribution of M to G residues in polymer chains changes based on extraction source and processing circumstances, leading to distinct physical and chemical characteristics.

PROPERTIES OF SODIUM ALGINATE

Sodium alginate is biocompatible, biodegradable, and mucoadhesive, making it ideal for biomedical applications. The capacity to create hydrogels in the presence of divalent cations, including calcium ions, enables the production of sodium alginate beads and controlled drug delivery systems.[19]

HOW SODIUM ALGINATE BEADS WORK

1. Formation of a Protective Gel Layer

- Sodium alginate is a natural polysaccharide that reacts with gastric acid (HCl) to form a viscous gel.
- This gel floats on the surface of the gastric contents, creating a mechanical barrier that protects the ulcerated mucosa from further acid attack and irritation**.**

2. Neutralization of Acid

- When combined with antacids like sodium bicarbonate, sodium alginate can neutralize excess stomach acid, reducing acidity and relieving pain.

3. Prolonged Drug Retention

- If sodium alginate is used as a drug carrier (in bead form), it can provide controlled and sustained drug release at the ulcer site, enhancing the therapeutic effect.

4. Mucoadhesion for Enhanced Protection

- The alginate gel adheres to the gastric mucosa, forming a sticky protective layer over the ulcer, promoting healing and reducing irritation.[22][23]

METHODOLOGY

FORMULATION TECHNIQUES OF SODIUM ALGINATE

A. Ionotropic gelation method

Ionotropic gelation is a commonly used process for producing sodium alginate beads. This process involves extruding sodium alginate into a solution containing divalent cations, often calcium chloride. Calcium ions crosslink alginate strands, forming hydrogel beads. In traditional ionotropic gelation processes, only the alginate solution is submerged in the crosslinking agent solution. Controlled release is a recent discovery in ionotropic gelation processes.[20]

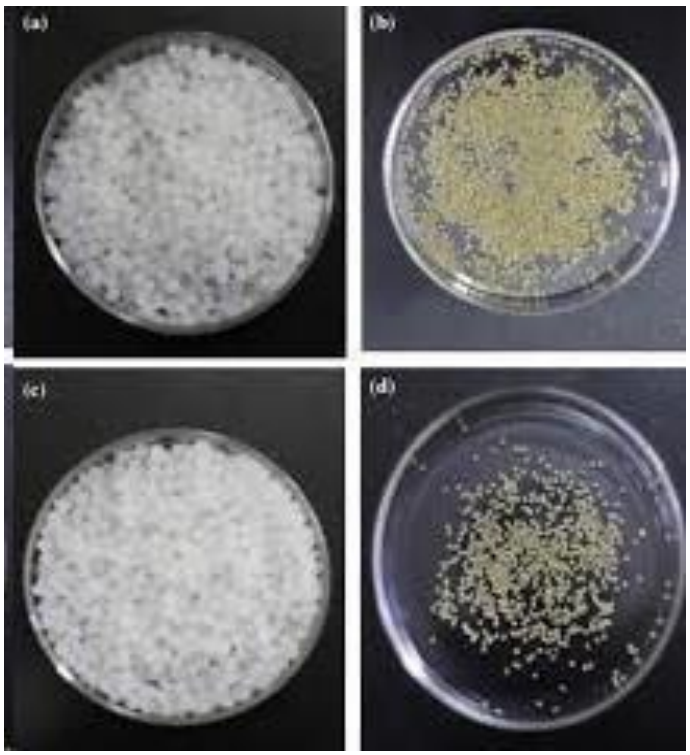

Fig 8: Sodium Alginate Beads.[24]

B. Emulsification and Solvent Evaporation Technique

The emulsification and solvent evaporation approach involve dispersing a sodium alginate solution in an organic solvent, then emulsifying in an aqueous phase with a crosslinking agent. The organic solvent evaporates, forming sodium alginate beads. This approach can encapsulate hydrophobic medicines within an alginate matrix. However, it may need

additional procedures for solvent removal and purification, and the use of organic solvents may limit its application in specific drug delivery systems.

a) Preparation of Sodium Alginate Solution

Sodium alginate solution is emulsified into an aqueous phase with a crosslinking agent, commonly calcium chloride (CaCl2), using mechanical or sonication. The technique produces tiny droplets of sodium alginate solution distributed in the aqueous phase.

b) Emulsification

Sodium alginate solution is emulsified into an aqueous phase with a crosslinking agent, commonly calcium chloride (CaCl2), using mechanical or sonication. The technique produces tiny droplets of sodium alginate solution distributed in the aqueous phase.

c) Crosslinking of Alginate Chains After emulsification, the organic solvent evaporates, leaving hardened sodium alginate droplets in the aqueous phase. Calcium ions in the aqueous phase crosslink alginate chains, creating a three-dimensional network structure.

d) Formation of Sodium Alginate Beads

As the solvent evaporates, the sodium alginate droplets solidify and eventually form spherical or irregularly shaped beads. The crosslinked network ensures structural integrity of the beads.

e) Washing and Drying

Beads are typically rinsed with distilled water to remove remaining solvent, crosslinking agent, and unreacted ions. The beads are dried using air or freeze-drying processes to get their final state.

f) Characterization and Analysis

The resulting sodium alginate beads are characterized using various techniques such as scanning electron microscopy (SEM), Fourier-transform infrared spectroscopy (FTIR), and particle size analysis. These analyse provide insights into bead morphology, structure, and properties.

g) Optional: Drug Loading and Release Studies

Dry beads can be loaded with pharmaceuticals or bioactive compounds through processes like soaking, immersion, or co-precipitation. Drug release studies analyse the kinetics and performance of prepared beads as drug delivery vehicles.[19]

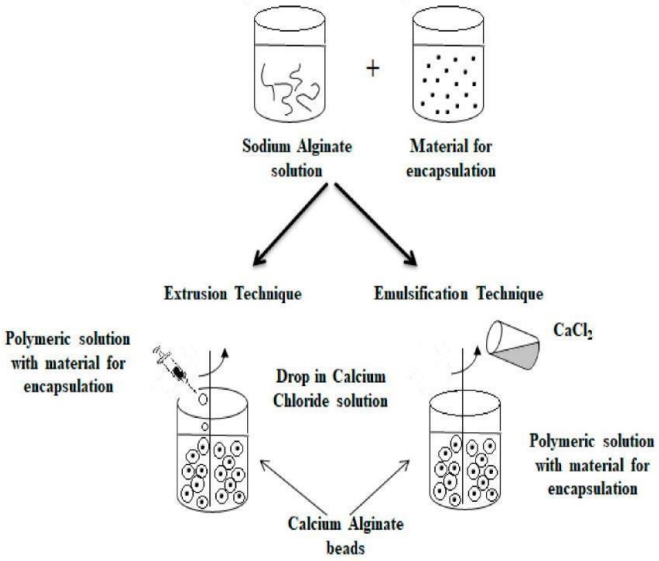

Fig 9: The Encapsulation process by extrusion (on the left) and by emulsification technique (on the right).[25]

EVALUATION METHODS FOR SODIUM ALGINATE BEADS

1. Physical Characterization Techniques

SEM, AFM, and particle size analysis are crucial for determining the size distribution, surface topography, and morphology of sodium alginate beads. SEM and AFM give high-resolution images of bead shape and surface characteristics, allowing for detailed analysis of their structure and integrity. Quantifying bead size distribution with particle size analysis techniques like DLS or laser diffraction ensures formulation uniformity.

2. Chemical Characterization

Chemical characterization techniques such as FTIR, DSC, and XRD are used to analyse the chemical, structural, and thermal properties of sodium alginate beads. FTIR spectroscopy reveals chemical bonds and functional groups in the beads.[16]

3. Biological Evaluation

To evaluate sodium alginate beads, biocompatibility, cytotoxicity, and bioactivity are tested both *in vitro* and *in vivo*. *In vitro* studies use cell culture models to assess the impact of beads on biological processes, including as cell viability, proliferation, and inflammatory response. *In vivo* studies use animal tests to evaluate the biocompatibility, pharmacokinetics, and tissue distribution of beads after delivery. These research offer vital insights into the safety and effectiveness of sodium alginate beads in biomedical applications.

4. Assessment of Drug Release Kinetics and Stability

Assessing drug release kinetics and stability is crucial for evaluating the effectiveness of sodium alginate beads as drug delivery vehicles. In vitro release studies track the release of encapsulated medicines from beads under simulated physiological settings. Several mathematical models, including zero-order, first- order, Higuchi, and Korsmeyer-Peppas, are used to study drug release kinetics and forecast processes. Stability studies evaluate the physical and chemical stability of beads during various storage conditions, such as temperature, humidity, and light exposure, to maintain product quality and shelf-life stability.[19]

CONCLUSION

Peptic ulcer disease (PUD) is a common clinical concern affecting people of all ages, with a significant impact on healthcare delivery, health economics, and patient quality of life. Helicobacter pylori (H. pylori) remains a significant risk factor for PUD development. The clinical prognosis of H. pylori infection is influenced by its predilection site. A comprehensive understanding of PUD pathophysiology can enhance clinicians' awareness of potential complications. Even though there has been a lot of development, further research is necessary to formulate and evaluate sodium alginate beads in a more innovative way. Recent advancements in formulation techniques and evaluation methods for sodium alginate beads have significantly contributed to the field of drug delivery and tissue engineering. Sodium alginate beads are widely utilized in pharmaceutical and biomedical fields for their versatility in controlled drug delivery systems. This review provides an in-depth exploration of recent advancements in formulation techniques and evaluation methods for sodium alginate beads.

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