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*Article*

# Understanding Stomach Ulcers and Blood Profile Changes

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**Abstract:** Peptic ulcers are a common gastrointestinal condition causing inflammatory damage to the stomach or duodenal mucosa, extending into deeper tissue layers. Symptoms range from mild abdominal pain to severe bleeding and perforation. This article provides a detailed overview of peptic ulcers, including types, symptoms, defense mechanisms of the gastric mucosa, and treatment options. It also examines the impact of peptic ulcers on blood composition, revealing significant changes. The study highlights a knowledge gap in understanding the full systemic effects of peptic ulcers. The research aims to elucidate these effects using a comprehensive review and analysis. Results indicate that peptic ulcers significantly affect blood and gland physiology, suggesting the need for preventative measures and targeted treatments to manage this complex condition. Implications of these findings underscore the importance of avoiding contributing factors to prevent associated illnesses and improve treatment efficacy..

**Keywords:** Peptic Ulcers, Gastric Defense, Blood Changes, Stomach Mucosa, Treatment Options.

## 1. Introduction

Peptic ulcer disease (PUD), commonly known as gastric ulcers or peptic ulcers, is a condition in which the mucosa of the stomach seems to rupture due to the caustic effects of pepsin and acid in the lumen. A histological breakdown of the gastrointestinal tract (GI) lining caused by the production of pepsin or stomach acid is what is known as peptic ulcer disease. It reaches the gastric epithelium's muscular layer of the propria. [1] Usually, it affects the proximal duodenum and stomach. The distal duodenum, the jejunum, or the lower esophagus could all be affected. Origin Mucosal necrosis resulting in lesions measuring at least 0.5 cm (1/5 inch) is known as peptic ulcers. It is the most prevalent ulcer in a region of the digestive tract that is quite painful and typically acidic. One of the most frequent causes of peptic ulcers is Helicobacter pylori. Moreover, pharmaceuticals like a ibuprofen , spirin, and other non-steroidal anti-inflammatory drugs (NSAIDs) can induce or exacerbate ulcers.[1]

In the duodenum, there are four times as many peptic ulcers. Since metastases account for about 4% of stomach ulcers, many biopsies are necessary to rule out malignancy. In most cases, duodenal ulcers are harmless. The ulcer's appearance might be convex, like a colon polyp, concave, pit-like (the picture that most people carry), or classic erosive ulcer. Broadly speaking, the convex form is typically seen in the duodenum and pylori, while the erosive concave type is typically found in the right stomach. These convex growths are shaped differently, but they are always elevated over the surrounding tissues. These growths are characterized by a prolonged lack of surface breaks in the mucosal tissues, and even at bigger sizes, at first they do not visually differentiate from adjacent tissues. This kind

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of ulcer can grow over extended periods of time without experiencing the discomfort that comes with a hole ulcer because of its surface integrity. [2]

Interestingly, convex sores do not have a superficial crater or mucous membrane breach, even at advanced growth phases other than crater. Although these convex growths have some tumor-like characteristics, they are not infectious organisms but rather an aberrant protrusion of stomach tissue that includes the muscles, serous layers, submucosa, and mucous membrane.But even small changes, like crater-shaped sores, could encourage the spread of diseases.[3]

Symptoms

- Burning stomach pain.
- heartburn.
- Intolerance to fatty foods.
- Feeling full, bloated, or burping.
- nausea.

## 2. Dsicussion

Stomach burning is the most common indicator of a peptic ulcer. Similar to empty stomach pain, the pain is exacerbated by stomach acid. Many times, symptoms can be reduced by taking medication that lowers acidity or by eating specific foods that lower stomach acid, but the pain may then return. Pain can increase at night and in between meals. Furthermore, a lot of peptic ulcer sufferers don't exhibit any symptoms. Peptic ulcers may sometimes produce severe symptoms, such as:[4]

- vomiting blood may appear red or black
- feeling faint
- breathing difficulties
- Dark blood in the stool, or black or tarry stools
- Nausea or vomiting
- Changes in appetite.
- . Unexplained weight loss

Types of Peptic Ulcer:

Stomach and duodenal ulcers are examples of the digestive tract ulcers covered by the general term "peptic ulcer." In the past, it was thought that eating spicy food and stress caused this kind of ulcer. But as subsequent studies have demonstrated, these are only the exacerbating circumstances. H. pylori infection or an adverse pharmacological response to specific medications, such as non-steroidal anti-inflammatory drugs (NSAIDS), are the causal agents [5]. Pain and discomfit in the abdomen are signs of peptic ulcers. Additional signs and symptoms include bloating, nausea, vomiting, reduced appetite, and weight loss. Some people may also develop dark feces, which are indicative of gastrointestinal bleeding, and blood in their vomit and stools. [6].

Aphthous Ulcers

Sores that develop on the inside of the mouth lining are called mouth ulcers. Mouth ulcers are common and usually arise from dental trauma, including broken teeth, ill-fitting dentures, or fillings. Anemia, measles, viral infections, oral candidiasis, chronic infections, throat cancer, mouth cancer, and vitamin B deficiency are a few prominent causes of mouth

ulcers or sores [7]. One of the most common types of inflammatory oral illnesses, pathos minor, is estimated to affect 15-20% of the global population. It is particularly frequent in North America, where reports of prevalences as high as 50–66% have been made [8] [9]. It has been discovered that smokers had a reduced incidence of aphthous ulcers than non-smokers. [10].

Esophageal Ulcers

Esophageal ulcers are lesions that appear in the esophagus, or food pipe. They are most often generated near the end of the food pipe and are felt as a pain right below the breastbone, in the same area that heartburn feels. Smoking, long-term use of NSAIDS, and acid reflux are all linked to esophageal ulcers. [11].

Stomach ulcer

After food passes from the esophagus into the stomach, it undergoes further digestion to further break down its nutrients before being absorbed in the small intestine. It generates acid and a number of other enzymes that reduce food to simpler forms. A mucous lining lines the inside wall of the stomach, shielding it from the acid and enzymes. An imbalance between the digestive juices the stomach produces and the different components that shield the stomach lining might result in ulcers.[12] One of the signs of ulcers is bleeding. Rarely, the stomach wall may be entirely damaged by an ulcer. Helicobacter pylori is a bacteria that is one of the main cause of stomach ulcers. Antibiotics are typically used in conjunction with drugs to lower stomach acid in order to treat ulcers caused by this particular bacterium.

The initial, inner surface layer of the inner lining is where most ulcers develop. A perforation is a hole in the duodenum or stomach. There is a medical emergency.[13]

Gastric acid secretions:

It is generally accepted that gastric acid is a primary ulcerogenic component in the development of gastric ulcer disease. Studies have shown that pepsin and acid hypersecretors account for nearly half of individuals with gastric ulcers [12]. Acid inhibits their capacity to penetrate the mucosal layer, playing a strict role in mucosal protection [13]. Histamine, acetylcholine, and gastrin are thought to be the three main secretagogues that induce acid secretion. The receptors on the surface of the parietal cells include H2 reacting to histamine produced from specific mast cells, receptors hypersensitive to the muscarinic impacts of acetylcholine produced from the vagus neuron, and maybe receptors sensitive to endogenous circulating gastrin.. [14] Gastrin either directly activates parietal cells or causes ECL cells to produce histamine, which in turn stimulates acid secretion[15][16]. Histamine stimulates acid secretion via a newly discovered histamine receptor called the H; receptor[17]. Furthermore, a number of studies show that histamine and histidine decarboxylase (HDC), the enzyme that synthesizes histamine, are present in the epithelial cells at the base of the pyloric glands. the sole source of acetylcholine (Ach) that has direct effects on the neurological system of the stomach. Without influencing histamine release, the muscarinic-1 agonist McN-A-343 increases acid secretion, indicating that the parietal cell's muscarinic receptor.[18]

Causes of Peptic Ulcer Disease

The pathophysiology and etiology of H. pylori ulcers

The two Australian scientists initially discovered that the primary cause of stomach ulcers is Helicobacter pylori in 1982. Gram negative H. Pylori is a spiral shaped, motile, microaerophilic, flagellated bacterium [14]. The effector protein cytotoxin associated gene A (cagA) is encoded by type I strains of Helicobacter pylori, which are known to have pathogenic properties. Gastric carcinomas and ulcers are caused by cagA, which alters cell

shape, increases motility, and disrupts cell junctional activity after translocation into the host cell [19]. In gastritis, H. pylori stimulates the cytokine expression such TNF-a. Additionally, polymorphonuclear leukocytes, lymphocytes, monocytes, and plasma cells in the lamina propria, as well as severe intraepithelial neutrophil infiltration [21], are overexpressed in pylori-infected stomach tissue [20]. With the right antibiotic regimens, the infection can be completely eliminated, resulting in little mucosal irritation and a low risk of ulcer recurrence. The typical treatment for H. pylori infection consists of triple therapy regimens that include two antibiotics (clithromycin and amoxicillin) plus a proton pump inhibitor (ranitidine bismuth citrate). [14] .

NSAIDS (Non-steroidal anti-inflammatory drugs)

NSAIDS are useful medications that have anti-inflammatory, analgesic, and antipyretic properties. They are applied to a broad range of clinical diseases, such as musculoskeletal disorders and arthritis. Regrettably, the fact that they cause stomach ulcers has restricted their use. Approximately 25% of long-term users of these medications have stomach ulcers.[22].

By inhibiting the production of COX, which has been shown to prevent the conversion of AA to PGs, NSAIDS aid in the advancement of ulceration by compromising the mucosal barrier, which corrodes pepsin and leads to the development of peptic ulcers. [23] [24]. Furthermore, endothelin-1 (ET-1) is a powerful vasoconstrictor that induces mucosal damage when COX-1 is inhibited by NSAIDS. By preventing prostaglandins from being synthesized, NSAIDS cause stomach damage by activating neutrophils and releasing reactive oxygen species (ROS) locally. [25] The pathophysiology of ulceration is further caused by more NSAIDs, which also significantly diminish mucosal blood flow, mucus-bicarbonate discharges, impaired platelet aggregation, increased leukocyte adhesion and reduced epithelial cell renewal. [26]. stomach acid ulcers. Methods for triple therapy consisting of On the other hand, stomach defense occurs. It is the first defense against the colonization of bacteria and receptors. Additionally, a number of studies suggest that the parietal cell originates from the postganglionic fibers as 22 Studies have demonstrated that the enzyme cyclo-oxygenase action exacerbates the effects of non-steroidal anti-inflammatory drugs (NSAIDs) through causing superficial lesions to deepen, interfering with platelet aggregation, and slowing ulcer healing. [27][28]

Medications

In addition to NSAIDs, the etiology of PUD has been linked to corticosteroids, bisphosphonates, potassium chloride, and fluorouracil.

There seems to be a nonlinear link between smoking and duodenal ulcers. Alcohol can cause acidity and irritate the stomach mucosa.

A hypersecretory environment can arise under the subsequent circumstances.

- Syndrome of Zollinger Ellison.
- Mastocytosis systemic.
- Fibrosis of the cysts.
- The hyperparathyroid state.
- Hyperplasia of antral G cells.[28]

Ethanol

There are several different mechanisms underlying ethanol-induced gastrointestinal lesions, such as mucosal cell injury, impaired mucosal blood flow, and reduction of stomach mucus content. Research has shown that ethanol administration through the stomach to the epithelium results in increased vascular permeability, edema formation, and a rapid and

time-dependent secrete of endothelin-1 into the blood stream, which occurs prior to the development of hemorrhagic mucosal erosions due to vasoconstriction. Furthermore, ethanol causes the necrotic lesions in the stomach mucosa by reducing the release of biocarbonate (HCO) and mucus formation. Furthermore, it has been documented that ethanol activates mitogen-activated protein kinases (MAPK) and TNF-a. Furthermore, ethanol has initiated apoptosis, which results in cell death.[27]. Additionally, it has been observed that the metabolism of ethanol results in the generation of superoxide anion and hydroperoxy free radicals, which affect the lifting's healing process. Increases in oxygen-derived free radicals and lipid peroxide content induce significant alterations in levels of cells, damage to membranes, death of cells, exfoliation, and erosion of epithelium. [22]

Mechanisms of gastric ulcer healing:

Mucosal tissue necrosis, mostly brought on by ischemia, causes a gastric ulcer by stopping the flow of nutrients and producing reactive oxygen species. Tissue necrosis and the production of arachidonic acid derivatives from wounded cells, like leukotrienes B, attract leukocytes and macrophages to necrotic tissue. In an attempt to repair the necrotic tissue, these cells phagocytize it and produce pro-inflammatory cytokines, that in turn activate nearby endothelial cells, fibroblasts, and epithelial cells. [29] Two components make up the morphology of a stomach ulcer: the base, which is formed of granulation tissues, a connective tissue rich in fibroblasts, macrophages, and proliferating microvessels, and the edge, which is surrounded by neighboring non-necrotic mucosa. The intricate process of ulcer healing involves the tissue trying to restore its integrity through self-healing following an injury.[30] It has been suggested that the phases of this process—haemostasis, inflammation, proliferation, and remodeling—can be identified in a sequential order with some overlap. The stages and duration of ulcer recovering can be characterized as follow: The ulcer development phase occurs within the third day of the injury and is indicated by inflammatory infiltration, tissue necrosis, the creation of the ulcer margin, and the development of granulation tissue; the ulcer recovering phase occurs between three and ten days of the injury and is divided into two phases: an early healing phase marked by rapid movement of epithelium cells and contraction of the ulcer base followed by a slow healing phase marked by angiogenesis in the ulcer bed, transforming of granulation tissue, and full regrowth of epithelium; the rebuilding phase occurs between twenty and forty days after the ulceration and consists of the reforming of glands, muscular mucosae, and muscular propria; the maturation phase occurs between forty and 150 days after the injury. The primary elements participated in the healing of gastric ulcers are defined by the maturation and differentiation of specialized cells and include:1) Protooncogenes, the first primary response genes 2) Angiogenesis and factors that promote angiogenesis.3. Platelets.4) Proteins that shock the body. 5) Annexin-1. 6) Remodeling of tissue and extracellular matrix.[31]

Cytokines

Cytokines are crucial for mucosal defense because they regulate the mucosal immune system in a fundamental way. Tumor necrosis factor (TNF)-a, interleukin (IL)-1B, IL-2, IL-6, IL-8, and other pro-inflammatory cytokines are involved in the pathophysiology of peptic ulcers. 32

An inflammatory response of the stomach mucosa causes infiltration, which in turn triggers transcription and produces a number of proinflammatory cytokines [33]. It has been demonstrated that IL-1 decreases the degree of gastric damage and increases injury resistance. [34] [35]. Although the exact mechanism of IL-1's protective effects is unknown, it has been discovered that IL-1 lessens damage by paradoxically inhibiting leukocyte adhesion. Moreover, IL-1 has been implicated in the suppression of stomach acid output

[36].[37][38] Additionally, IL-I increases prostaglandin and NO release, maybe through promoting the expression of COX-2 and NOS, protecting the 48-hour-old gastroduodenal neutrophils and mononuclear cells. Moreover, it has been demonstrated that IL-1 inhibits the mucosa's produce of other mediators that pruduce ulcers, such as PAF and histamine come from mast cells. [39][40]

VEGF (Vascular endothelial growth factor)

The most powerful inducer of angiogenesis is VEGF, a 46-kDa homodimeric glycoprotein that is produced by several cell types such as smooth muscle cells, fibroblasts, mega-karyocytes, macrophages, and neoplastic cells [41][42].

Many repair processes, including the healing of gastric ulcers produce by an imbalance between substances that harm the gastric mucosa barrier and those that have a safety effect, depend heavily on angiogenesis and VEGF. There is evidence from multiple studies that VEGF has a part in the healing of stomach ulcers. [43]

NO (Nitric oxide) is produced from L-arginine by the NO synthases (NOS), which are NO enzymes that catalyze the process. NO has been shown to be involved in both the pathophysiology of mucosal harm and GI mucosal defense. [44] Moreover, NO may have an impact on exocrine and endocrine secretion, as well as muscular tone. Because inhibition of these enzymes can cause abnormalities in GlI motility, blood flow, secretion, etc., CNOS, NNOS, and ENOS are crucial to the GI tract's normal function [45].which contributes to mucosal damage and dysfunction under some clinical circumstances by producing comparatively large quantities of NO [46][47]. The stomach mucosa is more vulnerable to damage when NO production is suppressed. Neutrophils are not recruited to inflammatory areas when NO is present. Infiltration of neutrophils into the GI tract mucosa is decreased by NO [48]. Nitric oxide has been shown to have gastroprotective properties, which include promoting angiogenesis and reducing acid secretion [49][50]. Nitric oxide's fast reactivity with different oxygen molecules may be the cause of its gastroprotective properties. 56. Conversely, inducible NOS species inside the biological system [51].Additionally, by preventing the produce of histamine from enterochromaffin like cells, nitric oxide suppresses stomach secretion.[52][53]

Prostaglandins Prostaglandins are fatty acids with 20 carbons that are made from arachidonic acid by the cyclooxygenase enzyme. According to Hawkey and Rampton's research, prostaglandins promote the release of mucus and bicarbonate, preserve mucosal blood flow, and strengthen epithelial cells' resistance to cytotoxin-induced damage [54]. The GI mucosa is inflamed, and prostaglandins have been shown to decrease leukocyte recruitment, which may be one of these chemicals' positive effects [55]. It has been demonstrated that prostaglandin E2 (PGE2) is a strong inhibitor of PAF, histamine, and TNF-a production from intestinal and peritoneal mucosal cells [56][57]. Additionally, it has been discovered that nitric oxide prevents conditions where dins prevent neutrophils from producing reactive oxygen species. [58].

LTs (Leukotrienes) Through the action of lipoxygenase, arachidonic acid is converted into leukotrienes, which are thought to be significant mediators of inflammation and allergic reactions [59]. It has been proposed that there are two primary categories of LTs: peptido-leukotrienes (LTC4, LTD4, and LTE4) and leukotriene B4 Two. Strongly stimulating the produce of reactive oxygen products of neutrophil metabolism, LTB4 is a very effective chemotaxin for neutrophils and plays a major role in tissue damage linked to mucosal inflammation. The function of LTs) on the stomach was explained by Goldberg and Subers. [60] It has been demonstrated that LTs cause the stomach's vascular bed to constrict, which

is followed by macromolecule leakage from the postcapillary venules. Moreover, a number of additional investigations have revealed that LTC4 can cause tissue necrosis and larteriolar venous vasoconstriction [61][62]. LTs may therefore act as a possible proulcerogenic agent. According to reports, the synthesis of gastric mucosal LTC4 and B4 increases in a concentration-dependent manner upon ethanol administration. This suggests that LTs may act as mediators in the harm that ethanol causes to the stomach.[63] The exact method through which ethanol induces the rat stomach mucosa to produce LT is unknown. arteries in the rat submucosa, which might be brought on by the disruption of phospholipase membranes within the cells. This would activate the activity and raise the quantity of arachidonic acid, which would then improve LT synthesis.[64]

Moreover, it has been proposed that LTB4's capacity to encourage leukocyte adhesion to the vascular endothelium plays a role in the pathophysiology of NSAID-induced stomach injury. Moreover, LTB4 might be involved in a similar way in the pathophysiology of ulcers brought on by Helicobacter pylori infection. out of 66 It's interesting to note that patients with gastric H. pylori colonization have much greater gastric juice LTB4 levels than people who do not have Endothelin pylori. [65].

Endothelin : is a peptide with 21 amino acids that is obtained from vascular endothelial cells. It has been proposed that this peptide plays a pathophysiological role in cases when there is vascular Additionally, endothelin may function as a feature regulator of vascular tone and an endogenous that opposes the effects of EDRF. prostacyclin, a calming agent generated from endothelium (PGI2). The characteristic of stomach ulcers brought on by drugs like aspirin and ethanol is vascular congestion [66]. The gastrointestinal mucosa is affected by the ulcerogenic properties of a number of lipid mediators, which may have an impact on the vascular smooth muscle and/or endothelium.[67] Endothelin increased the mucosa's susceptibility to ethanol-induced injury. At a concentration that the gastric mucosa could tolerate, endothelin also increased the damage that hydrochloric acid caused to the stomach. Moreover, endothelin caused a noticeable vasoconstriction and increase in stomach vascular perfusion. It is quite possible that endothelin's vasoconstrictor effects in the stomach are the cause of the proulcerogenic pressure, which is most likely a reflection of its actions. It seems that the preservation of stomach mucosal integrity requires a balance between endothelial constricting and relaxing forces, especially when a necrotizing agent is present. [68][69]

Neurohormonal mechanisms: Mechanisms that are at least partially activated by the central nervous system and hormonal variables support the gastric mucosal defense. Experiments have shown that central vagal stimulation raises intracellular pH in the stomach's surface epithelial cells and induces mucus secretion. Furthermore, even though the CRF pathway plays a role in the body's hormonal reactions to stress [70]. Furthermore, peripheral CRF plays a major role in controlling the mechanisms of gastric defense. Specifically, the CRF2 receptor is recognized to have anti-apoptotic properties in gastric epithelium cells and to impede the motility and emptying of the stomach. [68] The control of gastric protective mechanisms is significantly influenced by other hormone mediators, such as gastrin-17, cholecystokinin, thyrotropin-releasing hormone (TRH), and neurokinin A. Blocking the CGRP receptor, suppressing NO synthase, and ablating afferent nerves can all attenuate the effects of these mediators. [71][72]

P/D1 cells in humans and gastric A-like cells found in rodents generate the hormone peptide ghrelin, which is implicated in the control of growth hormone release and stimulation of appetite. Additionally, it has the capacity to have important protective effects at the stomach level, such as improving mucosal blood flow by stimulating the release of CGRP and NO from sensory afferent neurons. [73][74]

Impact of Proton Pump Inhibitors (PPIs) on the healing of ulcers and protection of the stomach mucosa:

PPIs are very useful in helping to heal stomach damage caused by NSAIDs, even when NSAID use is ongoing. This is because they activate both acid-dependent and -independent pathways. This has been shown by a number of lines of evidence that are clinical and preclinical. PPIs are benzimidazole derivatives that have been substituted and have strong inhibitory effects on the release of stomach acid.[75]

Innovative therapeutic approaches for stomach ulcer prevention and management

The promotion of ulcer healing is largely dependent on controlling gastric acid secretion, but there is growing interest in characterizing the mechanisms underlying ulcer healing and exploring the possibility of pharmacologically modifying the rate and caliber of ulcer treatment. [76]

Currently, new pharmacological approaches are being researched to mitigate the negative effects of conventional NSAIDs on the gastrointestinal system. The primary option being actively evaluated are: (i) two inhibitors of cyclooxygenase and 5-lipooxygenase (5-LOX), to prevent mucosal harm caused by enhanced leukotriene biosynthesis resulting from the change of arachidonic acid metabolism towards the leukotriene pathway as a consequence of cyclooxygenase inhibition; (ii) traditional NSAIDs related with phosphatidylcholine, to reduce the destabilizing effect of these medications on the extra-cellular mucosal lining of zwitterionic phospholipids; (iii) NO-donating NSAIDs, known as cyclooxygenase inhibitors /NO donors (CINODs), which aim to prevent the harmful actions of NSAIDs through the gastroprotective activity of exogenous NO (iv) NSAIDs cause the release of H2S, a gaseous mediator that is vital to the preservation of blood flow and the integrity of the gastric mucosa.A few of the medications listed above are during clinical development. Specifically, in phase II or phase III trials controlled with placebo or naproxen, licofelone, a dual cyclooxygenase/5-LOX inhibitor, has been demonstrated to spare the human stomach mucosa when taken for 4–12 weeks to healthy participants or patients with osteoarthritis. [77][78]

In a 4-day study involving healthy volunteers, soy phosphatidylcholine administration significantly decreased the aspirin's gastric-injuring action as determined by endoscopic examination; however, prostaglandin levels found in gastric samples were significantly lower in both treatment groups. A recent study assessing the safety of ibuprofen coupled chemically with phosphatidylcholine in individuals with osteoarthritis found that this combination was more tolerable than ibuprofen by itself.[79] [80]

Blood hematological parameters

Blood makes up around 8% of an adult's body weight. Males have approximately 5–6 liters, and females have about 4-5 liters. The primary cause of this discrepancy is the variations in body sizes between males and females. It is 38 degrees Celsius on average.[81] It is mildly basic (pH less than 7 is regarded as acidic) with a value of 7.35-7.45. The viscosity of whole blood is 4.5–5.5 times that of water, meaning that it has a higher flow resistance than water.[82] Blood's viscosity is essential to its function since too little or too much resistance may strain the cardiac muscle and create serious cardiovascular issues.[83]

Because the arteries contain larger levels of oxygen than the veins, the blood in the artery is a brighter red. [84]

variations in some hematological markers in stomach ulcer patients:

Due to the high prevalence of gastric ulcers in Iraq and the knowledge of how they affect blood profiles, the red blood cell distribution width (RDW), which is based on the width of the RBC volume distribution curve, reflects the heterogeneity of the peripheral

blood erythrocyte volume [85]. An elevated RDW is indicative of both inefficient red cell synthesis and excessive red cell death. [86] RDW is a novel chronic inflammatory marker that is linked to multiple sclerosis, enclosing spondylitis, and skin eruptions but independent to age, gender, or hemoglobin level. [87][88][89] The erythrocytes' functional specialization is in the transportation of oxygen. Depending on the disease or physiological state, their numbers can change. [90, 91]Their readiness serves as a barometer for the animal's overall health. The degeneration of red blood cells and variations in the rate of oxygen-induced red blood cell synthesis are the causes of morbidity. Additionally, both (Hb) (PCV) are the most crucial blood parameters for the diagnosis of cancer, liver disease, and anemia, among other illnesses. The concentration of hemoglobin and the quantity of red blood cells are determined by the size of the cells. Any alteration in their values suggests that one or both of them are damaged. The existence of a toxic component negatively impacts the process of blood production, and the disorder of the percentage indicates that poisons have been removed from the blood [92].Ulcer injury is the cause of the greater drop in concentration in the control group when compared to other groups.[93]

## 3. Conclusion

The pathophysiology of stomach ulcers differs based on the kind of ulcer and can arise from a variety of sources. It significantly affects the blood and gland physiology. It is important to avoid the factors that contribute to its emergence because it can induce other illnesses and has complicated, slowly acting treatment options.

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