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INSECT AND PANCREAS HISTOLOGICAL CHANGES WERE EXAMINED IN MELOXICAM-TREATED ALBINO RATS

Dr Pradeep Kumar*

Assistant Professor, Department of Physiology, Gouri Devi Institute of Medical Sciences and Hospital, Durgapur, India

ABSTRACT

The upper and lower gastrointestinal tracts are also damaged by NSAIDs, along with the stomach and pancreas. The gastrointestinal tract can become perforated, ulcerated, or bleed when taking meloxicam, like all NSAIDs. Gum acacia hydroalcoholic extract was applied to albino rats treated with Meloxicam and studied on their intestine and pancreas histology. Each group had five rats weighing the same amount. Each group was given 0.2 mg/kg meloxicam per day, while the second group was given 1gm gum acacia per day. A meloxicam-and-gum combination was administered daily to the third group, while a normal diet and water were administered to the fourth group. A 21-day diet was provided to rats. Meloxicam-treated rats consumed gum acacia hydroalcoholic extract and showed considerable protection. As a result of this study, gum acacia protects against meloxicam's damaging effects when used in combination with meloxicam therapy.

Key words: NSAIDs, Gastro-intestinal tract, Hydro alcoholic extract.

INTRODUCTION

A 21-day diet was given to all rats. Rats treated with meloxicam were significantly protected by gum acacia aqueous extract. Animals in the first and third groups showed significant increases in intestinal enzymes such as lipase, amylase, alkaline phosphatase (ALP), and lactate dehydrogenase (LDH), while animals in the second group showed significant decreases (P 0.001). There are few other NSAIDs that are as effective in protecting against the harmful effects of meloxicam therapy as gum acacia [1,2]. It helps reduce appetite, flush out toxins, and treats diabetes in addition. Rats' intestinal and spleen immune systems performed better when gum acacia was administered. The consumption of gum acacia has been documented to lower serum cholesterol levels in recent studies, while other studies have found little to no effect on cholesterol levels [3].

Study objectives were to evaluate pancreatic and intestinal enzyme activity and biochemical changes in rats treated with meloxicam, as well as histopathological changes associated with these enzymes.

MATERIALS AND METHODS

ANIMAL GROUPS

This study examined whether meloxicam affected albino rats' pancreatic juices, intestinal juices, and ileums after consuming it. Each group contained 10 males and females of albino rats weighing 190 grams. Meloxicam was administered in the first group at 0.2 mg per kg of body weight daily, gum acacia was administered in the second group at 1 g/day, and meloxicam was administered in the Third group at 0.2 mg per kg of body weight followed by gum.

There was a 21-day period of drug administration for each group. Control group participants were fed a normal diet and given free access to water. Hematoxylin and eosin was used to examine the general histological structure of the ileum of both control and treated rats. As far as nutrition, temperature, and humidity are concerned, all rats were subjected to the same conditions.

Corresponding Author: - **Dr Pradeep Kumar**

Ultrastructural Microscopic Examination

A 0.1M cocadylate buffer with 3% glutaraldehyde was used for electron microscopy of ileum adjacent to the ileum. A mixture of ethanol and propylene oxide was used to clear the sections after fixation. Lead citrate and uranyl acetate stained ultrathin sections with toluidine blue, while lead citrate stained semithin sections with toluidine blue. Transmission electron microscopy was used to examine the ultrastructure of the tissues [4].

Statistical Analysis

In this study, data management, mean calculation, standard deviation calculation, correlation calculations, and testing for significance will be done using a program that utilizes data management.

RESULTS

CONTROL GROUP

Picture of Histology

In the ileac sections of the control animals, the characteristic layers were demonstrated; serous, muscular submucosa, and mucosa. Several evaginations into the gut lumen were present in the mucosa, which was the most important layer for absorption. Lamina propria connective tissue surrounded each villus and contained capillaries and smooth muscles. It was found that this propria had a few lymphocytes. The primary absorbent cells of these villi were tall columnar cells with oval nuclei

Ultrastructural picture

The uppermost lateral membranes of enterocytes are tightly junctioned at the ultrastructural level. These enterocytes in the supranuclear region were densely packed with parallel finger-like microvilli. Numerous spherical mitochondria and rough endoplasmic reticulum cisternae were present on the sidewalls of enterocytes. The microvillus terminal web is made up of striated filamentous microtubular cores that are connected and form a network.

Meloxicam-Treated Group

Picture of Histology

There was atrophy, necrosis, and desquamation of ileal epithelium in rats treated with meloxicam, particularly at villar tips. Intestinal glands apically degenerated, which caused a severe villar fusion. In the lamina propria and muscular layer, there were multiple multinucleated enterocytes with apoptosis [fig 3].

Ultrastructural picture

The ultrastructure of these enterocytes showed severe degeneration of mitochondria and rough endoplasmic reticulum, as well as loss of microvilli. There was a fragmentation and condensing of the nuclei. Apoptotic changes in epithelial cells are associated with degeneration of apical epithelium and irregularities in microvilli.

Figure:1 A close-up of the ileum mucosa in the control group reveals its characteristic layers; the long villi (V), the columner cells (C), the goblet cells (G), the submucosa (Sm) with intestine glands (IG), the muscular layer (M) and the osseous layer (Sr).

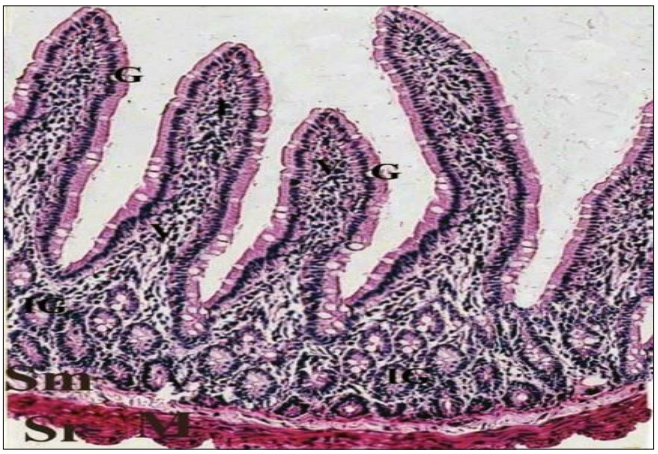


Figure: 2 The photomicrograph shows microvillar brush and goblet cells (Mv and G) in an enterocyte viewed through a transmission electron microscope. Rough endoplasmic reticulum (RER) and mitochondria (M) are present in the cell

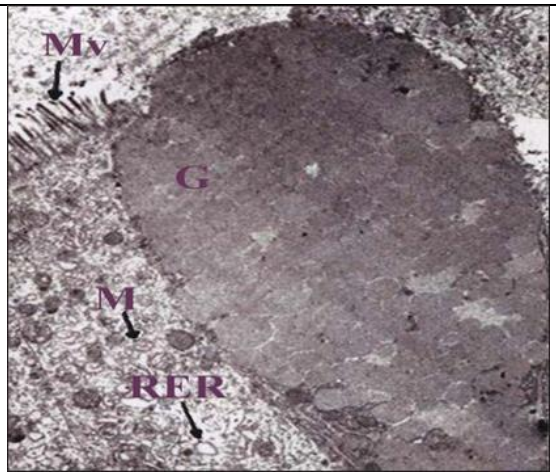


Figure: 3 It consists of an ulcerated epithelial layer of the rat ileum (U) and inflammatory cells infiltrating musculoskeletal lamina propria (I**).**

Figure: 4 The transmission electron photomicrograph illustrates the characteristic changes in the ileum epithelium (N) that are characterized by apoptosis of condensed and

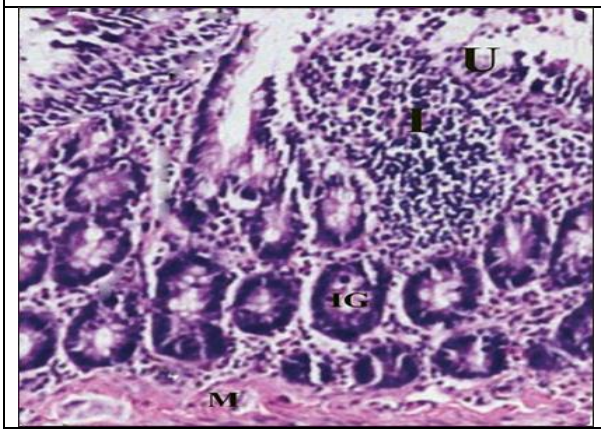
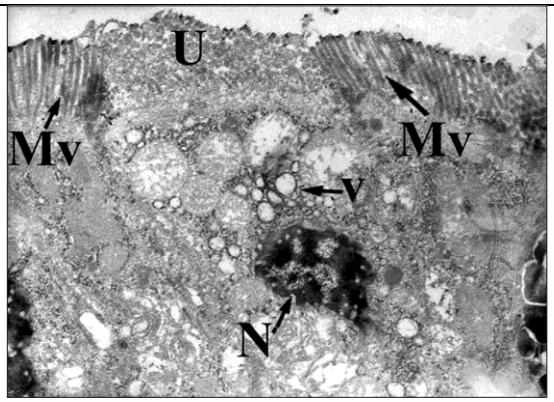
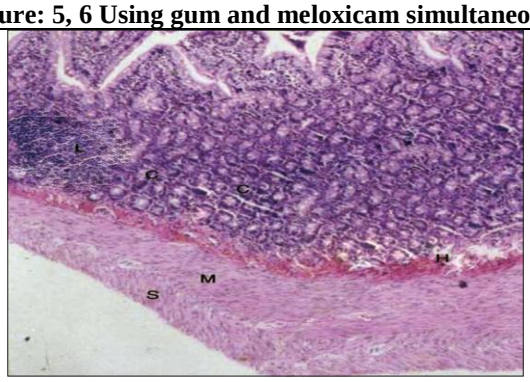
	fragmented nuclei, increased vacuolization (v), degeneration of the apical surface epithelium (U), and irregularities in microvilli.
	

Figure: 5, 6 Using gum and meloxicam simultaneously, photomicrograph the mucosa of a rat's ileum



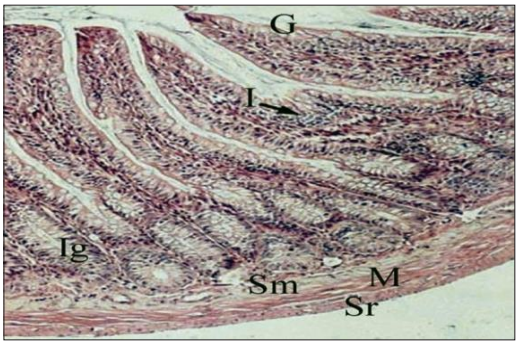
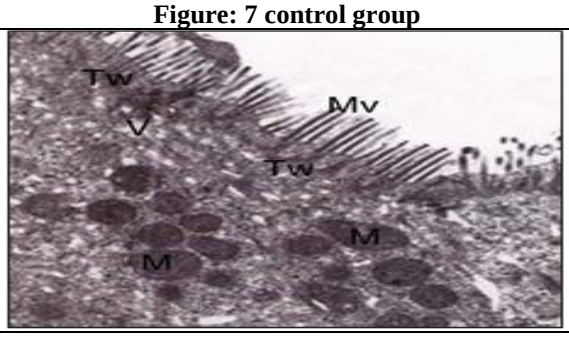


Figure: 7 control group



A Group Treated with Gum Acacia and Meloxicam Picture of Histology

Approximately 90% of gum-treated animals displayed significant improvement, but their normal histological architecture could not be completely restored. Submucosal hyalinization with thickened serosa and muscula were observed as well as vilar fusion, epithelial cell proliferation, and crypt proliferation. It was still possible to observe lymphocyte nodules [Fig 5 and 6]. In the control group, goblet cells were also found in a greater number [Figure 7]

DISCUSSION

After 21 days of meloxicam therapy, either alone or combined with gum, both intestinal mucosa and brush borders were shown to be affectionate as well as a significant increase in brush border enzyme activity (lipase, amylase, alkaline phosphatase, lactate dehydrogenase). This compares well with either a control group or a gum-only treated group. The results reported earlier were in line with these. Cox-1 and Cox-2 inhibition was confirmed to be required to induce intestinal damage in [5,6], and the inhibitors, despite causing hypermotility, invasion of bacteria, and increased expression of meiosis-

related proteins like iNOS and eNOS, upregulated Cox-2, which counteracted subsequent events such as increased myeloperoxidase (MPO) and iNOS activity to maintain mucosal integrity. When each of Cox-1 and Cox-2 is inhibited, intestinal damage occurs only. It has been suggested that NSAIDs act as detergents in the digestive tract, causing disruption of mucus gel and/or the integrity of all membranes. The concentration of acid in NSAIDs may also be increased in the mucosa [7]. A history of stomach ulceration, chronic bleeding, and iron deficiency is associated with the use of NSAIDs, as well as damage to small and large intestines [8, 9]. Anaemia, oxidative DNA damage, cognitive dysfunction and iron deficiency contribute to iron deficiency coupled with compromised immune function [10]. A marked hypermotility in the rat small intestine was observed after an ulcerogenic dose of conventional NSAIDs was given. Intestinal damage and bacterial invasion begin much earlier after this change in motility occurs than within 20–30 minutes after the change in motility occurs. Consequently, intestinal hypermotility may have an important role in the pathogenic mechanism of meloxicam-induced small intestinal lesions because abnormal contraction of the intestinal wall disrupts the unstirred mucus layer over the epithelium, which increases mucosal sensitivity to pathogens and irritants. The intestinal damage could be prevented if intestinal hypermotility and bacterial invasion were inhibited potently [11, 12]. Also, mucosal hypoxia and microvascular damage were caused by intestinal hypermotility caused by the Cox-1 inhibitor [13]. During the above-mentioned gastrointestinal lesion model, peroxynitrites are formed when nitric oxide interacts with oxygen radicals. With 0.5% gum sonicated, most protease activities were reduced, whereas bacteria of *B. gingivalis* and *B. intermedia* showed trypsin-like activity. There was almost always a lower inhibitory effect for gum-soluble fractions when compared to sonicate fractions. Various mucosal offensive and defensive factors make acacia gum an effective anti-ulcer drug. Moreover, this agent's ability to inhibit these periodontal pathogens and their enzymes suggests it may be beneficial in the treatment of these conditions [14].

Some plants produce milky juices that are infused with gum resin. As a result of finely powdering them and rubbing them with water, they form emulsions that are mainly used in medicine [15]. Gums are a high-energy food source made up of water, complex polysaccharides, calcium, and trace minerals. Cations, especially divalent ones, can be bound to Arabic gum. In our study, histology revealed marked changes in the intestinal tract, the main tissue; rats treated with meloxicam developed ulcers and infiltrating the intestinal wall; animals treated with meloxicam and gum were found to have mild pathological changes, superficial ulcers and minor inflammation [16]. However, when gum was used alone, the picture was incomparable to that exhibited by those who were not treated. The former studies also found that gum did not decompose or disintegrate efficiently in the alimentary tract and absorbed large quantities of water, acting as a mechanical laxative [17, 18]. The consumption of gum increases the excretion of faecal nitrogen, does not affect starch digestion, and does not inhibit vitamin A absorption, an important factor in ulcer healing.

CONCLUSION

As an anti-Cox-1 and Cox-2 NSAID, meloxicam therapy is one of the new anti-Cox-1 and Cox-2 drugs utilized in this research. It was concluded that gum acacia protects and defends against the effects of meloxicam therapy.

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