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Natural Volatiles & Essential Oils

Evaluation Of Antiulcer Activity Of Sangu Parpam- An Experimental Study

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Abstract:

The Siddha System of Medicine is a traditional South Indian system of medicine. In the Siddha literature, Siddhar classified the diseases into 4448 and also mentioned the treatment for the diseases. Among the diseases that are classified, Gunmam is one of the diseases that is compared with Peptic Ulcer disease. As per the Siddha literature, Sangu Parpam is a unique medicine to treat the disease. Thus, the Sangu Parpam was prepared as per the literature and anti-ulcer activity was carried out by the methods of Pyloric ligation induced ulcer in rats and Ethanol/HCL induced ulcer in rats. The treatment with Sangu parpam shows a reduction in the gastric lesion area and promotes significant regeneration of the gastric mucosa in both methods. Thus, the traditional claim that Sangu Parpam is effective against peptic ulcers was proved. Further evaluation of the medicine is needed for its acceptance worldwide.**Keywords:** Peptic Ulcer Disease, Gunmam, Siddha, Sangu Parpam, Anti Ulcer Activity.

INTRODUCTION:

Traditional medicine has played an important role in meeting the demands of primary health care in many developing countries, and its use has expanded widely in many developed countries[1]. Siddha Medical System (SMS), also known as Indigenous Tamil Medicine, is a unique, significant, and scientific system that has been in use since time immemorial.The Siddha system of medicine deals not only with the external body but also with the internal soul.

The Alma Ata Declaration in 1978 at the WHO international conference on Primary Healthcare (PHC) advocated "the importance of integrating traditional practises as primary health care" and it also ascertained that "health is the state of complete physical, mental, and social well-being, not merely the absence of disease or infirmity".

Nowadays, modern lifestyle habits and fast-moving life have increased the rate of peptic ulcers[2.] Peptic ulcers are present in around 4% of the population. In 2013, nearly 53 million people developed peptic ulcers. 10% of people in the world develop peptic ulcers at some point in their life. In 1990, 327000 deaths were recorded, and in 2013, nearly 30000 deaths were recorded due to peptic ulcers.

Even with the advent of many advanced treatments for peptic ulcer disease, they all possess many side effects like cardiac arrhythmias, hypertension, and nephritis etc., [3]. Though the Siddha literature highly recommends **Sangu Parpam** for Peptic Ulcer Disease, the worldwide usage of this medicine will be on hand if the safety, efficacy, and mode of action of the medicine are established by

standard scientific methods. In this work, **Sangu Parpam,** a herbo-mineral Siddha drug, is taken which is extensively used by traditional medicine practitioners.

Therefore, an attempt has been made to unveil the facts about the herbo-marine Siddha drug **Sangu Parpam**[4]**,** a calcined product conch shell from the literature with anti-ulcer activity.

MATERIALS AND METHODS:

Preparation of Sangu Parpam (SP):

Purification of Sangu [5]:

Sangu was processed in the Thaalithal method (heating process**)** by covering it with Karchunnam (limestone).

Preparation process [4]:

100g of purified Sangu from each purification process was covered up by ground paste of Uthamani (*Pergularia damea*) and kept in the mud lid and closed by another mud lid. Cotton ribbon soaked in wet clay was winded over the rims of both mud lids and let to dry in sun light for 8 hours. Then this set up was subjected to Ganapudam. (100 cow cakes were used). After cooling, the set up was taken out and the calcinated Sangu was taken out, ground well, and stored in an airtight container.

Anti ulcer studies:

Pylorus ligation method [6]:

Albino Wister rats of either sex weighing between 150 to 200gm were divided into six groups of 6 animals each.
Group I: Control (Ghee 5ml/kg)
Group II: Only pylorus ligation
Group III: pylorus ligation + Ranitidine 30 mg/kg body weight, oral.
Group IV: pylorus ligation + **SANGU PARPAM 9.36mg**/200gm
Group V: pylorus ligation + **SANGU PARPAM 46.8mg**/200gm
Group VI: pylorus ligation + **SANGU PARPAM 93.6mg**/200gm

"According to this method, the Albino Wister Rats were kept under fasting for 24 hours in metabolic cages and were taken care of in order to avoid coprophagy. In the control vehicle, three doses of SANGU PARPAM and the standard drug (Ranitidine 30 mg/kg) were given at different doses for five days orally. At the end of the fifth day, the animals were kept under fasting for 14 hours with water ad libitum. About 30 minutes before the ligation, SANGU PARPAM was administered to the animals. Under light ether anesthesia, the abdomen was opened and the pylorus ligated. Care was taken in order to avoid bleeding or to occlude blood vessels and the abdomen was sutured. The animals were then sacrificed after 6 hours of pyloric ligation under a surplus of ketamine hydrochloride, and the stomach was dissected out. Gastric juice was collected from the sacrificed animal and its volume, pH, free acidity, and total acidity were measured; the ulcer index was also determined. Evaluation of

antioxidant enzymes, SOD, CAT, lipid peroxidation, myeloperoxidation, and histopathological evaluation were done on the excised stomach.

Ethanol/HCL induced ulcer method[7]:

Albino Wister rats were divided into 6 groups of 6 animals each. The animals were of either sex and were of nearly 150-200g in weight.

Group I: Control (Ghee 5 ml/kg)

Group II: Negative Control (HCL/Ethanol mixture containing 0.15 N HCL in 70% v/v Ethanol 1.5 ml) p.o

Group III: HCL/Ethanol+ Ranitidine 30 mg/kg body weight, oral.

Group IV: HCL/Ethanol+ **SANGU PARPAM 9.36mg**/200g

Group V: HCL/Ethanol+ **SANGU PARPAM 46.8mg**/200g

Group VI: HCL/Ethanol+ **SANGU PARPAM 93.6mg**/200g

"The animals were kept under fasting for 24 hours except for drinking water ad libitum until 2 hours before the start of the experiment. Gastric injury was induced with an acidified ethanol solution (150mMHCL/absolute ethanol) 40:60 v/v, (HCL/ethanol solution), as per a modification of the method. Ghee was administered orally to the normal control groups and normal saline was administered to the ulcer control groups. For the Reference group, 20mg/kg omeprazole was orally administered and for the experimental groups, oral administration of Sangu parpam 9.36 mg, 46.8 mg, 93.6 mg/200g was given. After one hour of this pretreatment, ghee and normal saline were orally administered to the normal control group and the ulcer control group, respectively. Except for the normal control group, all the experimental groups were administered with HCL/ethanol solution (5ml/kg) orally for inducing gastric ulcers. With an excess of xylazine and ketamine anesthesia, the rats were euthanized 60 minutes after the treatment. Their stomach was immediately excised and the ulcer index determined. The anti-oxidant enzymes SOD, CAT, GPX, lipid peroxidation, and MPO were analyzed. **[8]**.

RESULTS:

TABLE 1-EFFECT OF SANGU PARPAM ON FREE ACIDITY AND TOTAL ACIDITY IN PYLORIC LIGATION METHOD

Group	Control	Only pylorus	Pylorus+ Ranitidine 30mg/kg	Pylorus+ S.P(II) 9.36mg/200g	pylorus+ S.P(II) 46.8mg/200g	pylorus+ S.P(II) 93.6mg/200g
FREE ACIDITY	36.12±1.1	54.67±1.43#	39.50±1. 3*	40.72±1. 6	40.13±1.02	40.16±1.12*
TOTAL ACIDITY	58.14±1.43	84.32±1.47#	59.10±1.5*	59.20±1.	58.38±1.09	58.18±1.31*

				5		

TABLE 2 -EFFECT OF SANGU PARPAM ON GASTRIC pH AND GASTRIC VOLUME IN PYLORIC LIGATION METHOD

Group	Control	Only pylorus	Pylorus+ Ranitidine 30mg/kg	Pylorus + S.P(II) 9.36mg/200g	pylorus+ S.P(II) 46.8mg/200g	pylorus+ S.P(II) 93.6mg/200g
GASTRIC PH	2.3±0.20	1.23±0.16#	2.58±0.06**	2.35±0.12*	1.93±0.1*	2.1±0.2*
GASTRIC VOLUME	0.68±0.11	4.83±0.4#	2.27±0.12**	2.48±0.33*	2.86±0.14*	2.39±0.32 *

Values are expressed as the mean ± S.D: Control vs. Negative Control # P<0.05, Negative Control vs. Treatment * P<0.05 Std ** P<0.01

TABLE 3 - EFFECT OF SANGU PARPAM ON ULCER SCORE AND ULCER INDEX IN PYLORIC LIGATION METHOD

Values are expressed as the mean ± S.D; Control vs Negative Control ## P<0.01 Negative Control vs SPpy 93.6 ** P<0.01 Std ** P<0.01

Group	Control	Only pylorus	Pylorus+ Ranitidine 30 mg/kg	Pylorus + S.P(II) 9.36mg/200g	pylorus+ S.P(II) 46.8mg/200 g	pylorus+ S.P(II) 93.6mg/200g
ULCER SCORE	0±0	7.91±0.19##	3.95±0.22**	6.10±0.14*	4.78±0.14*	4.78±0.18**
ULCER INDEX	0±0	8.02±0.39##	6.13±0.16**	7.66±0.22*	5.13±0.09*	3.93±0.10**

TABLE 4 -EFFECT OF SANGU PARPAM ON TOTAL PROTEIN IN PYLORIC LIGATION METHOD

Group	Control	Only pylorus	Pylorus+Ranitidine 30 mg/kg	Pylorus+ S.P(II) 9.36mg/200g	pylorus+ S.P(II) 46.8mg/200g	pylorus+ S.P(II) 93.6mg/200g
TOTAL PROTEIN (g/dl)	0.76±0.00	0.72±0.00*	0.47±0.00**	0.82±0.00	0.78±0.00*	0.71±0.00

Values are expressed as the mean ± S.D; Control vs Negative Control * P<0.05 Negative Control vs Std ** P< 0.01 Negative Control vs SP II * P<0.05

TABLE 5 -EFFECT OF SANGU PARPAM ON ANTIOXIDANT PARAMETERS IN PYLORIC LIGATION METHOD

Group	Control	Only pylorus	Pylorus+ Ranitidine 30mg/kg	Pylorus+ S.P(II) 9.36mg/200g	pylorus+ S.P(II) 46.8mg/200g	pylorus+ S.P(II) 93.6mg/200g
SOD (Unit/min/mg protein)	0.65±0.01	0.33±0.00#	0.55±0.01*	0.48±0.00	0.52±0.00	0.54±0.00*
CAT (µmol of H202 consumed /min/mgprotein)	0.90±0.00	0.61±0.00#	0.81±0.00*	0.76±0.00	0.80±0.00	0.82±0.00*
GPX (µmoles of glutathione oxidized/min/mg protein)	0.69±0.00	0.47±0.00#	0.59±0.00*	0.51±0.00	0.53±0.00	0.54±0.00*

Values are expressed as the mean ± S.D; Control vs Negative Control ## P <0.01 Negative Control vs Std -Non Significant SP II * P<0.05

TABLE 6 - EFFECT OF SANGU PARPAM ON LIPIDPEROXIDATION IN PYLORIC LIGATION METHOD

Group	Control	Only pylorus	Pylorus+ Ranitidine 30 mg/kg	Pylorus+ S.P(II) 9.36mg/200g	Pylorus+ S.P(II) 46.8mg/200g	Pylorus+ S.P(II) 93.6mg/200g
LPO (nmol of MDA/mg protein)	0.69±0.02	0.83±0.00##	0.67±0.00ns	0.53±0.00	0.52±0.00	0.51±0.00*

Values are expressed as the mean ± S.D; Control vs Negative Control # P<0.05 Negative Control vs Std * P< 0.05 SP II * P< 0.05

TABLE 7 -EFFECT OF SANGU PARPAM ON MYELOPEROXIDATION IN PYLORIC LIGATION METHOD

Group	Control	Only pylorus	Pylorus+ Ranitidine 30 mg/kg	Pylorus+ SP 9.36mg/200g	Pylorus + SP 46.8mg/200 g	Pylorus + SP 93.6mg/200 g
MPO(µmol/mi n/mg tissue)	0.87±0.00	1.06±0.08#	0.75±0.02*	0.75±0.02	0.75±0.03	0.77±0.02*

Values are expressed as the mean ± S.D; Control vs Negative Control # P<0.05 Negative Control vs Standard * P<0.05 SP II * P<0.05

TABLE 8 - EFFECT OF SANGU PARPAM ON ULCER SCORE AND ULCER INDEX IN HCL/ETHANOL INDUCED ULCER MODEL

Group	Control	Only HCL/ Ethanol	HCL/Ethanol+ Ranitidine 30 mg/kg	HCL/Ethanol + SP 9.36mg/200g	HCL/Ethanol + SP 46.8mg/200g	HCL/Ethanol + SP 93.6mg/200g
ULCER SCORE	0±0	11±0.36##	2.33±0.21**	4.33±0.42	4.33±0.56**	3.33±0.42*
ULCER INDEX	0±0	15±0.36 ##	3±0.36 **	7.33± 0.42**	5.33±0.56 *	4.03±0.42 *

Values are expressed as the mean ± S.D; Control vs Negative Control ## P<0.01 Negative control vs Standard ** P<0.01 SP II *

TABLE 9 -EFFECT OF SANGU PARPAM ON TOTAL PROTEIN LEVEL IN HCL/ETHANOL INDUCED ULCER MODEL

Group	Control	Only HCL/ Ethanol	HCL/Ethanol+ Ranitidine 30 mg/kg	HCL/Ethanol + SP 9.36mg/200g	HCL/Ethanol + SP 46.8mg/200g	HCL/Ethanol + SP 93.6mg/200g
TOTAL PROTEIN (g/dl)	50.67±3.6	74±9.89#	67±1.67ns	48.67±2.56ns	44.67±2.56ns	34.33±2.08ns

TABLE 10 -EFFECT OF SANGU PARPAM ON ANTI OXIDANTS ENZYMES IN HCL/ETHANOL INDUCED ULCER MODEL

Values are expressed as the mean ± S.D; Control vs Negative Control #P<0.05 No Significant changes in Negative Control vs Standard Negative control vs SP II

Group	Control	Only HCL/ Ethanol	HCL/Ethanol+ Ranitidine 30 mg/kg	HCL/Ethanol + SP 9.36mg/200g	HCL/Ethanol + SP 46.8mg/200g	HCL/Ethanol + SP 93.6mg/200g
SOD (Unit/min/mg protein)	0.4±0.07	0.14±0.01##	0.49±0.08	0.39±0.07 ns	0.38±0.02 ns	0.44±0.01 ns
CAT (µmol of H2O2 consumed/min/ m g /protein)	5.31±0.34	2.59±0.19##	4.20±0.22*	3.39±0.15*	4.09±0.05	4.59±0.22

Values are expressed as the mean ± S.D; SOD: Control vs Negative Control ## P<0.01 Negative Control vs SP II Non Significant CAT: Control vs Negative Control ## P<0.01 Negative Control vs Standard * P<0.05 SP II * P<0.05 GPX : Control vs Negative control## P<0.01Negative control vs Standard * P<0.01 SP II * P<0.05

GPX (µmoles of glutathione oxidized /min/mg protein)	7.2±0.06	3.49±0.10##	6.15 ±0.11*	5.18±0.09ns	5.38±0.90	5.66±0.27

TABLE 11 -EFFECT OF SANGU PARPAM II ON LIPID PEROXIDATION LEVEL IN HCL/ETHANOL INDUCED ULCER MODEL

Group	Control	Only HCL/ Ethanol	HCL/Ethanol+ Ranitidine 30 mg/kg	HCL/Ethanol + SP 9.36mg/200g	HCL/Ethanol + SP 46.8mg/200g	HCL/Ethanol + SP 93.6mg/200g
LPO (nmol of MDA/mg protein)	4.49±0.21	13.63±0.36##	5.03±0.48ns	5.03±0.13ns	4.67±0.63ns	5.15±0.11ns

Values are expressed as the mean ± S.D; Control vs Negative Control ## P<0.01 No significant changes between Negative control vs Standard and SP II

TABLE 12 -EFFECT OF SANGU PARPAM ON MPO LEVEL IN HCL/ETHANOL INDUCED ULCER MODEL

Group	Control	Only HCL/ Ethanol	HCL/Ethanol+ Ranitidine 30 mg/kg	HCL/Ethanol + SP 9.36mg/200g	HCL/Ethanol + SP 46.8mg/200g	HCL/Ethanol + SP 93.6mg/200g
MPO (µmol/min/mg protein)	0.37±0.061	0.47±0.05$^{\#}$	0.29±0.012*	0.41±0.04	0.35±0.02	0.26±0.05*

Values are expressed as the mean ± S.D; Control vs Negative Control # P<0.05 Negative control vs Standard * P<0.01 SP II * P<0.01

Group	Control	Only HCL/ Ethanol	HCL/Ethanol+ Ranitidine 30 mg/kg	HCL/Ethanol + SP 9.36mg/200g	HCL/Ethanol + SP 46.8mg/200g	HCL/Ethanol + SP 93.6mg/200g
Mucus weight (g)	0.52±0.02	0.27±0.02$^{\#\#}$	0.39±0.08*	0.42±0.01	0.29±0.01	0.27±0.01*
PGE2 (Pg/ml)	132±1.46	46.67±1.84$^{\#\#}$	87±1.67*	47±2.03*	64.33±1.17	70.67±1.12

Table 13 -EFFECT OF SANGU PARPAM II ON MUCUS WEIGHTAND PGE2 IN HCL/ETHANOL INDUCED ULCER MODEL

Pyloric Ligation Model

The animals treated with all the dose levels did not produce any significant weight variations throughout the study period.

The animals treated with SP at the dose of 9.36, 46.8 and 93.6mg/kg showed a statistically significant

Values are expressed as the mean ± S.D; Control vs Negative control ## P<0.01 Negative control vs Standard *P<0.05 SP II * P<0.05

decrease (P < 0.05) in the free acidity level when compared to the normal control group. (Table 1).

The pyloric ligation group alone showed a marked increase in the total acidity level when compared to the normal control group, which is statistically significant (P 0.05).

In animals treated with Sangu Parpam in different doses, there was a statistically significant variation in gastric pH (P 0.05) and total volume of gastric juice when compared to normal control

animals (P 0.05) (Table 2). The ulcer score as well as the ulcer index of the Sangu Parpam also showed a significant variation (P 0.01) (Table 3) when compared with the control group.

There is no significant variation in the total protein (Table 4) level of the Sangu Parpam treated group compared with the control group. In the ulcer induced group, the anti-oxidant enzymes SOD, CAT, GPX, LPO, and MPO were decreased when compared with the control group. SP and the control group both have an increase in antioxidant enzyme levels, which protects against ulcer formation and has anti-ulcer activity.(Table 5,6,7)

HCL / Ethanol Induced Method

The ulcer score was found to significantly increase in the ethanol induced group of animals when compared with the control group (P 0.01). The ulcer index also showed a significant increase when compared with the control group. (Table 8).

In animals treated with SP in different doses, there was a statistically significant decrease in ulcer score and ulcer index when compared with the ethanol induced ulcer group (P 0.01) (Table 9). The animals treated with Sangu parpam did not produce any significant variation in total protein levels. (Table 9)

The antioxidant enzyme SOD level did not change significantly.But the animals treated with 46.8mg/200g showed a significant increase (P 0.01) in catalase and GPX levels, while the 93.6mg/200g group also showed a significant increase in values (P 0.01). But the LPO and MPO levels did not show any significant variation. (Table 10, 11,12)

The animals treated with Sangu parpam as well as standard drugs showed a significant increase in mucus weight. (Table 13).

DISCUSSION:

The study concluded that Sangu parpam has anti-ulcer activity in rats using the Pyloric Ligation Model. The antiulcer property of Sangu parpam in the pylorus ligation model is evident from its significant reduction in free acidity, total acidity, number of ulcers, and ulcer index[9]. Moreover, this SP significantly suppressed the formation of the ulcers. The significant inhibition of gastric ulcer in rats pre-treated with SP was comparable to that of ranitidine, which is a standard drug used for curing gastric ulcers (Fig.1). Sangu parpam treated animals decreased both the concentration and the pH, and increased the gastric wall mucus and gastric mucosa, so it is suggested that Sangu parpam can suppress gastric damage induced by aggressive factors. As per the study, SP shows significant anti-ulcer activity.

HCL-Ethanol Induced Ulcer Model

Peptic ulcers are caused by an imbalance between the protective and the aggressive mechanisms of the mucosa, and are the result of the association of several endogenous factors and

aggressive exogenous factors that are related to living conditions. Sangu Parpam could significantly protect the gastric mucosa against HCL-Ethanol induced injury. Compared to the control group, the test drug showed a significant increase in protection of the gastric wall mucosa and also in ulcer area by inhibiting oedema and leukocyte infiltration of the submucosal area (Fig.2). The PGE2, SOD, and CAT levels of tissue homogenate reveal increased levels of antioxidant enzymes in the treated group. This study provides complete evidence that the SP possesses an anti-ulcer activity.

Conclusion:

Sangu Parpam was taken for anti-ulcer studies. The studies revealed that Sangu Parpam had a significant anti-ulcer activity in both ulcer models. This study shows a reduction in the gastric lesion area and promotes significant regeneration of the gastric mucosa. Thus, the Sangu Parpam sample confirms its anti-ulcer activity inboth the Pylorus ligation method and the Ethanol/HCL induced ulcer method. This research work justifies and confirms the traditional claim that Sangu parpam is one of the important medications for peptic ulcer disease.

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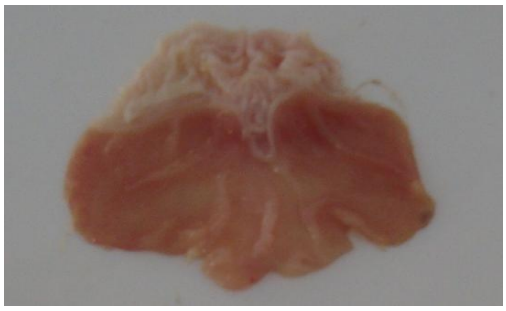
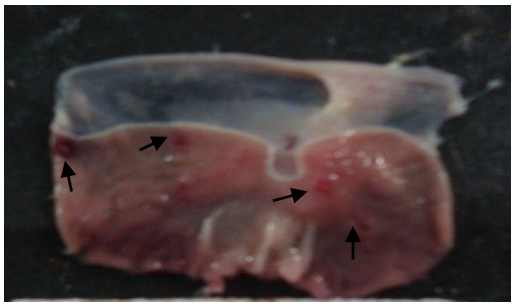
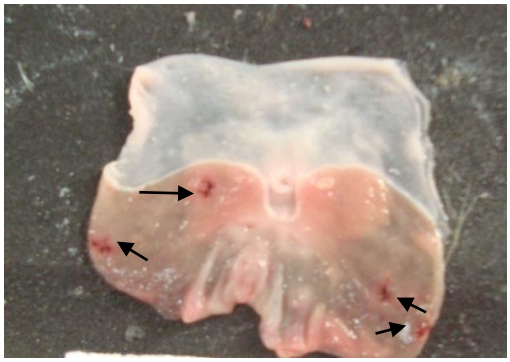
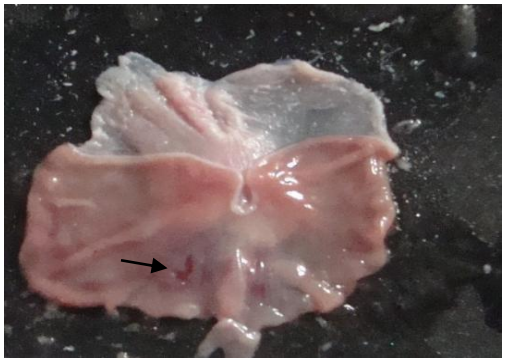
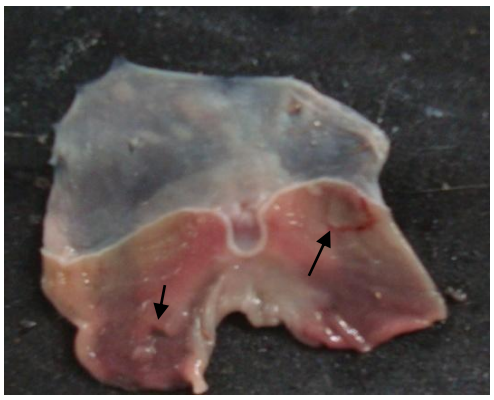
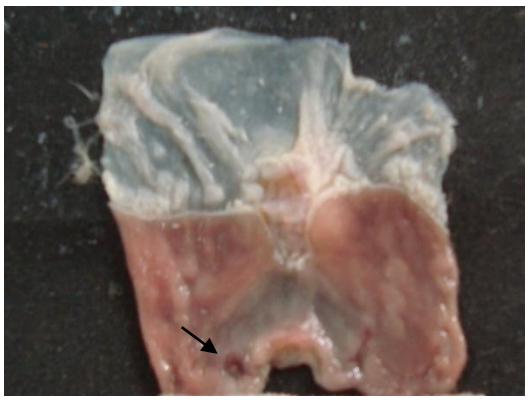
Control 	**Only PL shows severe damage of**  **mucosal layer**
 **PL+ Ranitidine 30 mg/kg**	 **PL+ Sangu parpam -9.36mg/200g Shows Moderately damage of mucosal layer**
 **PL+ Sangu Parpam 46.8mg/200g Shows Moderately damage of mucosal layer**	**PL + Sangu parpam -**  **93.6/200gm Shows Protected mucosal layer**

Fig 1: MACROSCOPICAL VIEW OF PYLORUS LIGATION (PL) INDUCED ULCER

Fig 2: MACROSCOPICAL VIEW OF THE GASTRIC MUCOSA IN HCL/ETHANOL INDUCED ULCER

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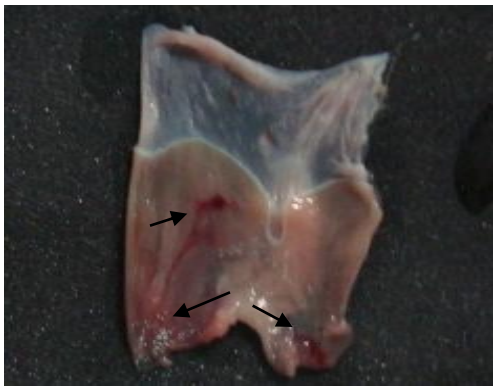
<td> 
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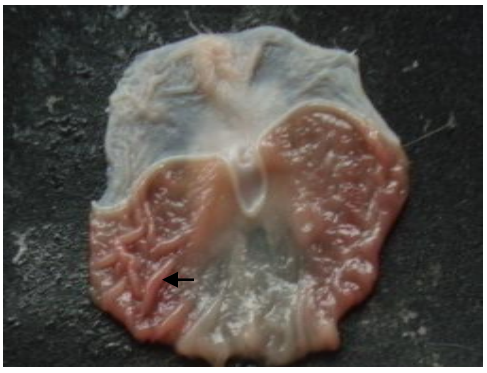
<td> 
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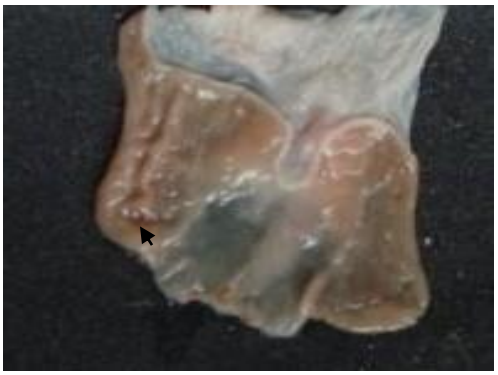
<td> 
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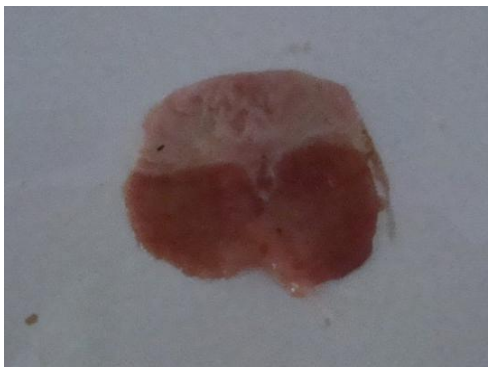
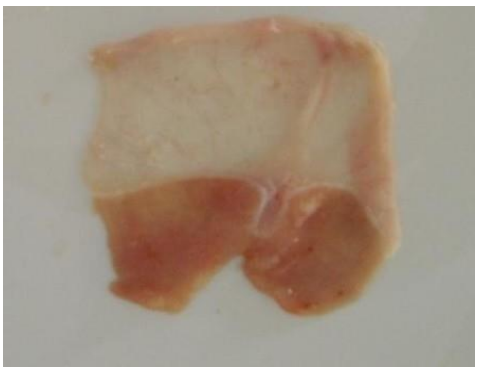
<td> 
HCL/ ETHANOL + SANGU PARPAM
46.8mg/200gm</td>
<td> 
HCL/ETHANOL + SANGU PARPAM
93.6mg/200gm</td>
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