ANALYSIS OF CLINICAL FEATURES, ULTRASOUND ABDOMEN AND UPPER GI ENDOSCOPY OF PATIENT PRESENTING WITH DYSPEPSIA

DISSERTATION SUBMITTED IN FULFILLMENT OF THE REGULATIONS FOR THE AWARD OF M.D. GENERAL MEDICINE.

DEPARTMENT OF GENERAL MEDICINE.
PSG INSTITUTE OF MEDICAL SCIENCES & RESEARCH
THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI, TAMILNADU

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GUIDE

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CERTIFICATE

This is to certify that the thesis entitled Analysis of clinical features, ultrasound abdomen and upper GI scopy of patients presented with features of dyspepsia is a bonafide work of Dr. G. JAGADEESWARAN, done under my direct guidance and supervision in the department of General medicine, PSG Institute of Medical Sciences & Research, Coimbatore in fulfillment of the regulations of Tamilnadu Dr. MGR Medical University for the award of MD degree in General Medicine.

GUIDE & HOD **PRINCIPAL**

DECLARATION

I hereby declare that this dissertation entitled was prepared by me under the direct guidance and supervision of Professor Dr. K. JAYACHANDRAN MD, Dr.L.VENKATAKRISHNAN MD, DM., PSG Institute of Medical Sciences & Research, Coimbatore.

The dissertation is submitted to the Tamilnadu Dr. MGR Medical University in fulfillment of the University regulations for the award of MD degree in General Medicine. This dissertation has not been submitted for the award of any other Degree or Diploma.

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INTRODUCTION

Dyspepsia is a very common human experience, for which there are numerous causes. As the alimentary tract can only indicate disquiet in a limited number of ways, symptoms can only be a guide as to the underlying problem.

Dyspepsia may be defined as a group of symptoms which alert doctors to disease of the upper gastrointestinal (GI) tract. This definition embraces structural, or organic, causes (such as peptic ulcer, cancer) and functional, where no identifiable lesion can be found.

Dyspepsia means 'bad digestion'. Numerous definition of dyspepsia have been attempted. Most authors consider that the symptoms should derive from the upper GI tract, but there is a wide range of conditions which can produce symptoms that are labeled dyspeptic. These range from serious conditions such as ulcer and cancer through to the common situation where about 50% of patients presenting with dyspepsia have no apparent structural abnormality (Mansi et al. 1993).

The functional dyspeptic group may be broken down into four groups; those whose symptoms suggest an ulcer but non present (ulcer like); those who appear to have gastro-oesophageal reflux but no oesophagitis (reflux-like) ; those with symptoms suggesting delay in

gastric emptying (dysmtoility - like) ; and a substantial group of those with non-specific features (non-specific or unspecified).

BACKGROUND

However, the prevalence of dyspepsia in the general population is much greater, with around 2% of patients experiencing dyspeptic symptoms, and may self-medicating. Patients and indeed doctors believe they known mean by dyspepsia or indigestion, even if the reality is different, which makes it important to define the condition as closely as possible.

Symptoms are unlikely to be the only means of defining dyspepsia, as the body has a limited number of ways of communicating bodily disquiet to the cerebral cortex. This is particularly true of the alimentary tract.

There are a large number of afferent fibres conveying information to the brain, which then processes the information and only a small proportion of it reaches the conscious level. That information which has already been processed by the autonomic system then has to communicated verbally against a background of different cultures and languages, and the considerable constraints which there impose. It is, perhaps, unsurprising that we are having difficulty in definition bearing

in mind that dyspepsia is not one condition, but has numerous causes- structural being the easiest to understand and study, whilst disturbances in function are so much more difficult to elucidate.

STRUCTURAL (ORGANIC) DYSPEPSIA

Symptoms alone do not allow a clear separation of organic causes for dyspeptic symptoms form non-ulcer dyspepsia (or functional dyspepsia). Some symptoms such as dysphagia have a high probability of underlying pathology requiring investigation and treatment. The dyspeptic symptoms that these patients experience are often longstanding and non-specific.

FUNCTIONAL DYSPEPSIA (NON-ULCER DYSPEPSIA)

It is clear that even functional dyspepsia is not one single condition but that there are several facets to it. A working group (Colin-Jones et al. 1988) therefore it is separated into those who sounded as though there was gastro-oesophageal reflux (reflux-like), those in whom there seemed to be delay in gastric emptying (dysmotility - like), those in whom the symptoms suggested an ulcer but none had been found (ulcer-like), and a final category of non-specific, where a substantial number of patients could not easily be put into one of the categories.

REFLUX LIKE:

Heartburn, some relief from antacids, Regurgitation, Retrosternal discomfort; on stooping, after large meals on lying flat

ULCER-LIKE:

Three or more of the following are necessary, but upper abdominal pain must be a predominant complaint

1. **Pain that is well localized in the epigastrium (i.e. can be localized to a single small area by pointing with one or two fingers).**
2. **Pain relieved by food, often (more than 25% of the time).**
3. **Pain relieved by antacids and/ or H_2-blockers, often**
4. **Pain occurring before meals or when hungry, often**
5. **Pain at times wakens the patient from sleep**
6. **Periodic pain with remissions and relapses (periods of at least 2 weeks with no pain, inter-spersed with periods of weeks to months when there is pain).**

DYSMOTILITY – LIKE:

Pain is not dominant symptom, upper abdominal discomfort should be present in all cases. This discomfort should be chronic and characterized by three or more of the following:

1. **Early satiety**
2. **Postprandial fullness**
3. **Nausea**

4. **Retching and / or vomiting that is recurrent**
5. **Bloating in the upper abdomen not accompanied by visible distention.**
6. **upper abdominal discomfort often aggravated by food**

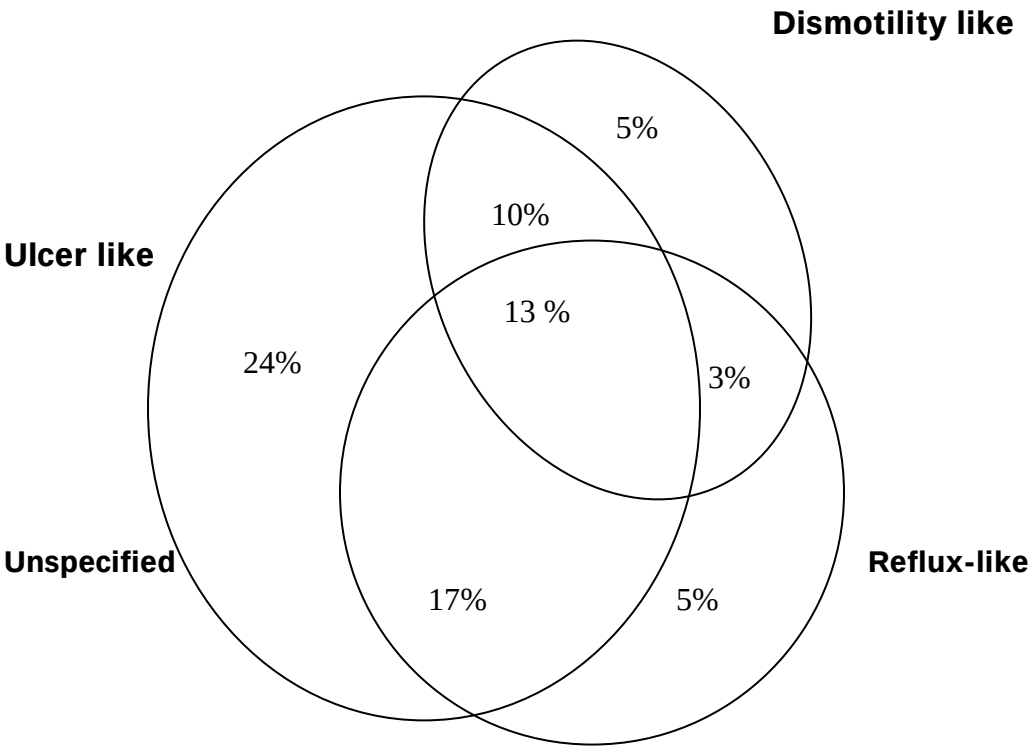
Add, If vomitis, 'cannot face food' IBS symptoms in past

IDIOPATHIC, ESSENTIAL OR NON-SPECIFIC DYSPEPSIA

The fourth category is a substantial group of probably about 25-30% of patients who do not easily fall into one of the above categories (Colin- Jones et al.1988). This is usually because if a mixture of symptoms. They should be treated as functional dyspepsia but as further information becomes available from more research, so hopefully this group can be broken down more specifically.

OVERLAP OF SUBGROUPS

Although some patients fall nicely into one of the specified categories above, may patients have a considerable overlap. A number of studies have demonstrated symptoms overlap, with many patients having symptoms that fit with two or even three of the categories (Talley et al.1922) (Fig.1.1).



This makes classification for the purposes of research studies or indeed treatment very difficult and is real weakness in the subgroup system. It has therefore been suggested that, rather than looking at specific set of symptoms, the dominant symptoms should be sought, such as whether pain is prevalent and where pain is not prevalent (this latter group particularly including those with nausea and bloating rather than actual pain). This grouping is simple , but again incompleted in separating out the different factors that may be contributing to functional dyspepsia (Perri et al.1998).

OVERLAP WITH OTHER CONDITIONS

Aerophagia

Air- swallowing leads to belching, which many patient consider to be a feature of dyspepsia. This is often justified, because a substantial number of patients with significant GORD will tend to swallow air as a response to their discomfort, a symptom which usually disappears when adequate control of the reflux takes place. It is helpful therefore to consider excessive swallowing of air sometimes being secondary to another condition excessive the upper GI tract, especially GORD. It can, however, sometimes be a primary problem, i.e. habitual gulping of air which is usually accompanied by a forward movement of the head as the swallowing and subsequent belching takes place. This latter category has nothing to do with dyspepsia. It is habitual abnormality of swallowing technique, often stress-related, but excess air in the stomach can cause discomfort.

IRRITABLE BOWEL SYNDROME

There is substantial overlap with irritable bowel syndrome. Approximately one-third of patients with functional dyspepsia will at some time either have had or will develop lower intestinal symptoms

RUMINATION:

Rumination is an uncommon problem in which food that has just been swallowed is regurgitated back into the mouth, is often chewed again and then reswallowed. It occurs more commonly in young children and those who are mentally disabled. Regurgitation occurs from the movement of the diaphragm altering pressure just at the moment that the bolus reaches the cardia. This again is a habitual phenomenon and is unrelated to functional dyspepsia.

FOOD ALLERGY

The public are convinced that stress and food intolerance are the keys to dyspeptic symptoms. The evidence that food allergy is responsible is unconvincing except in those in whom specific foods cause reproducible symptoms such as, for example, urticaria in response to shellfish. These latter patients usually know very specifically that they have a problem with a particular food because of these reproducible symptoms. Richer, fatty foods do variably cause symptoms in patients with functional dyspepsia.

ROLE OF HELICOBACTER PYLORI

Helicobacter pylori is associated with an increased risk of peptic ulceration and active chronic gastritis. It had been hoped that a specific subgroup of functional dyspepsia would be found to be do to H.Pylori infection.

AIM OF THE STUDY

Analysis of clinical features, ultrasound abdomen and upper GI scopy of patients presenting with of dyspepsia. To find out the most common cause.

REVIEW OF LITERATURE

PREVALENCE:

The study of Doll et al. (1951) reported 5-year prevalence, Jones and Lydeard (1989) 6 months, and Thompson and Heaton's figures (1982) were 1-year prevalence. Both the studies of Weit and Backett (1968) and Tibblin (1985) were concerned only with dyspepsia among men. Moreover, Tibblin's study was restricted to 50-year-old men. The population study of 40-years olds by Hollnagel et al. (1982) revealed a lifetime prevalence not very different from the reported 1-year period prevalence. In this study, women reported epigastric pain significantly more often than men. Another study from Scandinavia (Johsen et al. 1988) reported a lifetime prevalence of 22%. Here men reported heartburn and acid regurgitation more often women, and women reported abdominal pain as frequently as men did.

Some studies report a higher prevalence of dyspepsia among women (Tally et al. 1992b; Holtman et al.1994), but the difference in prevalence between sexes is marginal and diverges somewhat between the studies. The sex difference reported from Olmsted County, Minnesota (Tally et al.1994) is probably caused by the inclusion of nausea in the term of dyspepsia, and women report nausea

more frequently than women, while more women than men reported abdominal pain.

Most studies confim a decreasing prevalence of reported dyspepsia with increasing age.

Dyspeptic symptoms could be characterized by their severity, frequency and duration.

THE NATURAL HISTORY OF DYSPEPSIA

Inherent in the definition of dyspepsia is that complaints are persistent or recurrent (Heading 1991; Talley et al.1991; Thompson 1995). Recently several studies have addressed symptom turnover and repeated the findings of Weir and Backett (1968) the dyspeptic symptoms are persistent and recurrent. Weir found that 65% of those identified as dyspeptics had the same symptoms 3 years later.

Some studies report a higher prevalence of dyspepsia among women (Talley et a.1992b; Holtman et al. 1994), but the difference in prevalence between sexes is marginal and diverges somewhat between the studies. The sex differences reported from Olsted Country, Minnesota (Talley et al. 1994), is probably caused by the inclusion of nausea in the term of dyspepsia, and women report nausea more frequently than men. Overall, men seem to report heartburn

more frequently than women, while more women than men report abdominal pain.

Most studies confirm a decreasing prevalence of reported dyspepsia with increasing age. This trend is pronounced among men (Jones et al. 1990). While approximately 35 % of people aged 20-29 experienced dyspepsia the previous year in Olmsted, only 15.5 % reported such complaints among people age 65 and older (Talley et al. 1992 b, 1995a)

The broad definition of dyspepsia developed in the 1970s-'any symptom referable to the upper gastrointestinal tract' – has given way to more sophisticated systems of classification

Some of the implications of testing for H.Pylori in general practice are summarized by Agreus and Talley (1997) who propose that if the waiting time for endoscopy is excessively long it may be best to offer a test and treat options for H.Pylori among those patients aged less than 45 years who do not report alaram symptoms and who are not on non-steroidal anti-inflammatory drugs (NSAIDs).

Where the waiting time for endoscopy is short, testing for H.pylori and referring positive cases for endoscopy may be more desirable. It may be that, as further epidemiological work is completed, particularly with regard to the role of H.pylori in functional dyspepsia and gastric

cancer, attitudes towards endoscopy and empirical testing and treating for H.Pylori will change again.

CAUSES OF DYSPEPSIA

Luminal GI Tract

Food intolerance

Peptic ulcer disease

Gastroesophageal reflux

Gastric or esophageal neoplasms

Gastroparesis (diabetes, postvagotomy, scleroderma, chronic intestinal pseudo-obstruction, postviral, idiopathic)

Infiltrative gastric disorders (Menetrier's disease, Crohn's disease, eosinophilic gastroenteritis, sarcoidosis, amyloidosis)

Gastric infections (Cytomegalovirus, fungus, tuberculosis, syphilis)

Parasites (Giardia lambia, Strongyloides stercoralis)

Chronic gastric volvulus

Chronic gastric or intestinal ischemia

Irritable bowel syndrome

Functional dyspepsia

Medications

Ethanol

Aspirin, NSAIDs (including COX-2 selective agents)

Theophylline

Digitalis preparations

Glucocorticoids

Iron, potassium chloride

Niacin, gemfibrozil

Narcotics

Colchicine

Quinidine

Estrogens

Levodopa

Nitrates

Sildenafil

Orlistat

Acarbose

Pancreaticobillary Disorders

Chronic pancreatitis

Thyroid disease, hyperparathyroidism

Adrenal insufficiency

Renal insufficiency

Myocardial ischemia, congestive heart failure

Intra-abdominal malignancy

Pregnancy

FOOD INTOLERANCE:

Food intolerance may result from a number of mechanisms: mucosal "irritation" (from a noxious stimulus) or irritation of preexistent ulcer, stimulation of mucosal visceral afferent receptors, gastric overdistension, alterations in gastric emptying or intestinal motility, increased gas production, malabsorption, or, in rare instances, true food allergies

Meal- associated symptoms and a host of physical, psychological, and emotional factors related to meals may lead to altered eating habits. Anecdotal reports suggest that patients with chronic dyspepsia eat smaller, more frequent meals, possibly because of altered gastric accommodation or emptying or because of increased visceral sensitivity. High fat meals slow gastric emptying, p;romote gas retention in the small intestine, and may thereby exacerbate dyspeptic symptoms. These effects are attenuated experimentally by intravenous administration of deloxiglumide, a cholecystokinin-A antagonist or orlistate, a lipase inhibitor.

Specific foods are commonly implicated in dyspepsia. Coffee (Coffeinated or decaffeinated) often causes heart-burn, but its relationship to dyspepsia is unproven. Spicy foods particularly red and black papers, may cause acute gastric mucosal injury and acute epigastric pain. Alcoholic beverages in concentrations greater then

20% (40 proof) also may cause acute gastric mucosal injury, but there is little evidence that moderated doses of alcohol (10 to 20 g/d) causes dyspepsia. Heavy acute ingestion of alcohol and chronic alcohol abuse both may cause dyspepsia, which often is worse in the morning in this setting. Commonly overlooked as a cause of dyspepsia is lactose malabsorption, which may cause bloating, cramps, flatulence and diarrhea.

MEDICATION INTORLERANCE:

Medications may cause symptoms through direct gastric mucosal injury, alterations in gastric motility, provocation of gastroesophageal reflux, or idiosyncratic mechanisms. Chronic use of aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs) provokes dyspepsia in 10% to 25 % of persons who use the agents , but the occurrence of dyspepsia correlates poorly with presence of ulcers. Use of cyclooxygenase – 2 (COX-2) selective inhibitor is associated with the a lower frequency of dyspepsia than in use of nonselective NSAIDs. Other medications that commonly cause dyspepsia include potassium supplements, iron, antibiotics (especially macrolides, sulfonamides and metronidazole) digitalis, glucocorticoids, niacin, gemfibrozil, narcotics, colchicines, quinidiene estrogens and oral contraceptives theophylline, slidenafil, orlistat, acarbase and levodopa.

PEPTIC ULCER DISEASE

Most peptic ulcers are associated with dyspepsia, but most patients with dyspepsia do not have peptic ulcer disease. The frequency of peptic ulcers is increased in patients who ore older that age 40, have Helicobacter Pylori infection, use an NSAID, have dyspepsia at night, experience relief of pain with food or antacids have a history of peptic ulcer disease, are male, or smoke. The prevalence of peptic ulcer disease is declinton and NSAID use in the population.

GASTROESOPHAGEAL REFLUX DISEASE

Estimating the prevalence of GERD in patients presenting with dyspepsia is difficult. More that one third of patients with dyspepsia also have heartburn. Endoscopic evidence of esophagitis is present in 5 % to 15% of patients with dyspepsia (with or without hearburn), but because endoscopic studies fail to detect non erosive gastroesophageal reflux disease, the true prevalence of GERD is higher.

Symptoms of dyspepsia and GERD overlap considerably. Among patients with proven GERD, over one half have dyspepsia in addition to heartburn and up to 20% have dyspepsia alone, without heartburn or regurgitation. When a patient complaints dyspepsia and heartburn, with a heartburn as the dominant symptoms, the term "reflux – like dyspepsia" has been used. The Rome II committee excluded reflux-

like dyspepsia from the dyspepsia classification system and recommended that such patients be considered to have probable GERD.

GASTRIC OR ESOPHAGEAL MALIGNANCY:

Gastric or esophaeal malignancy is present in fewer than 1 % to 3% of patients with dyspepsia is referred for endoscopy. The majority of cancers are advanced (stage III or higher) at the time of presentation and fewer than 5 % arise in patients younger than 45 years of age.

PANCREATIC AND BILIARY TRACT DISORDERS

Biliary pain is characterized by discrete episodes of acute steady, upper abdominal pain or pressure that increase over several minutes and persists for up to several hours. The acute, relatively dramatic presentation of biliary pain should be distinguishable form dyspepsia in most patients.

Gallstones do not cause dyspepsia. Despite the high prevalence of both dyspepsia and gallstones in adults, epidemiologic studies have confirmed that cholelithiasis is not associated with dyspepsia. Therefore, patients with dyspepsia should not be investigated routinely for cholelithiasis, and cholecystectomy for cholelithiasis is not indicated for dyspepsia alone.

Symptoms of pancreatic disorders may be mistaken for dyspepsia. The pain of acute pancreatitis usually is severe, deep seated, often dramatic in its manifestation, and accompanied by nausea and vomiting. Chronic pancreatitis is characterized by bouts of dull, steady, upper abdominal pain that may radiate to the back; is aggravated by meals; and is easily confused with other causes of dyspepsia. Discomfort associated with pancreatic or ampullary cancer may be mistaken for dyspepsia but often is accompanied by weight loss, anorexia and jaundice.

SYSTEMIC DISORDERS:

Coronary ischemia may present with epigastric discomfort rather that chest pain. Pregnancy, acute or chronic renal failure, hyper- and hypothyroidism, adrenal insufficiency and hyperparathyroidism all may be accompanied by dyspepsia, nausea, or vomiting.

GASTROINTESTINAL DISORDERS THAT UNCOMMONLY MANIFEST AS DYSPEPSIA

The parasites Giadia lambia and Strongloides stercoralis, which reside in the upper intestinal tract, may cause dyspepsia. Gastroparesis is manifested by nausea, early satiety, postprandial epigastric pain, and vomiting. Many cases are caused by diabetes mellitus, scleroderma, vagotomy, chronic intestinal pseudo-obstruction, neurogenic disorders, or gastric resection, or follow a viral illness; rare cases are

idiopathic. Recurrent gastric volvulus may manifest with intermittent bouts of upper abdominal pain, bloating, bleching, retching or vomiting. Small intestinal malabsorptive disorders such as celiac sprue may manifest with dyspepsia and flatulence. Gastric or small intestinal involvement with Crohn's disease may cause upper abdominal symptoms as may infiltrative (lymphoma, amyloid, Mebetirer's disease), infectious (tuberculosis, syphilis, fungal) and inflammatory (sarciudisus, lymphocytic, gastritis, eosinophilic gastroenteritis) disorders of the stomach, which are diagnosed on upper endoscopy with biopsy. Chronic mesenteric or gastric ischemia may manifest with postprandial dyspepsia rather than the classic constellation of periumbilical abdominal pain, sitophobia (fear of eating), and weight loss.

APPROACH TO UNINVESTIGATED DYSPEPSIA

In evaluating patients with dyspepsia who have not previously undergone diagnostic investigation ("uninvestigated dyspepsia"), the physician must decide whether diagnostic studies, especially upper endoscopy, or a course of empirical treatment should be first step. The goal is to distinguish patients who have a higher likelihood of having a serious organic disorder (warranting early diagnostic evaluation and a definitive diagnosis) from the remainder of patients who may be

treated initially with empirical antisecretory therapy or H.pylori eradication therapy.

HISTORY OF PHYSICAL EXAMINATION

A complete clinical history should be obtained and a physical examination performed in all patients with dyspepsia. It is customary to ask patients about the nature, location, frequency, and chronicity or symptoms as well as the relationship of symptoms to meals or specific dietary factors. Careful inquiry to the patient's social or family history may uncover stresses that are contributing to acute symptomatic worsening or current concerns about chronic symptoms. Symptoms and signs of systemic disorders that may cause dyspepsia, such as cardiac disease, diabetes, and thyroid disese, should be considered. Signs such as abdominal organomegaly or a mass, ascites, or positive fecal occult blood test result necessitate further evaluation. In addition, the "laying on of hands" may be therapeutic for functional patients and provide reassurance that the symptoms are being taken seriously.

Patients should be asked about lower gastrointestinal and extraintestinal symptoms. Dyspepsia is common in patients with irritable bowel syndrome and other functional gastrointestinal disorders. Patients with chronic, uncomplicated dyspepsia who also

have lower abdominal pain or discomfort and altered bowel habits should be treated for a presumptive diagnosis of irritable bowel syndrome. The presence of multiple extraintestinal complaints such as fatigue, headaches, myalgias, and urinary urgency also is suggestive of a functional disorder.

EXCULDE OFFENDING MEDICATIONS:

The use of prescriptions and nonprescription medications should be reviewed, and medications commonly associated with dyspepsia-especially aspirin, NSAIDs or COX-2 inhibitor-should be discontinued when possible.

LOOK FOR ALARM FEATURES:

Endoscopy should be performed in all dyspeptic patients with alarm features in order to exclude gastric esopheal malignancy. Alarm features include unintended weight loss, progressive dysphagia, persistent vomiting, overt or occult gastrointestinal bleeding, unexplained anemia, jaundice, lymphadenopathy, and palpable abdominal mass. More that 90% of gastric or esophageal cancers present with at least one alarm feature. Unfortunately, alarm features have a poor positive predictive value for malignancy, because they are present in 10% to 20% of patients with dyspepsia. Most esophageal

and gastric cancers that manifest with dyspepsia, with or without warning symptoms, are advanced and incurable. Therefore although patients with dyspepsia and alarm features should undergo endoscopy, malignancy that is curable is seldom detected in these patients.

INITIAL LABORATORY STUDIES

1. **A complete blood count**
2. **ESR**
3. **Urea**
4. **Creatinine**
5. **Random blood sugar**
6. **Urine routine**

Other studies such as

1. **Stool testing for ova**
2. **Parasites or Giardia antigen**

ROLE OF ENDOSCOPY:

Dyspepsia can be thought of as the end point of a number of discrete pathological condition, and differentiating between them can be a considerable clinical problem. It is agreed that upper gastrointestinal endoscopy is the investigation of choice for diagnosing peptic ulcer disease and carrying out tissue biopsies for gastritis, H.Pylori infection

or upper gastrointerstinal cancer (Tally et al. 1998). Endoscopy has also been shown to have a therapeutic role. The power of negative endoscopy result was initially overlooked, endoscopy services being assessed solely by their yield of positive organic diagnoses.

The development of screening tests for H.Pylori has had major implications for the use of endoscopy. Although endoscopy can be said to have a therapeutic role in addition to its role as an investigative tool, it had been unclear whether H.Pylori screeinig tests would have a similar effect.

MATERIALS AND METHODS:

This study was carried out in 80 patients who presented with clinical features suggestive of dyspepsia, attended in Gastroenterology OP, Medicine OP and admitted in wards between September 07 - August 08 in PSG Hospitals.

All patients were screened according to protocol of a complete medical history, complete blood count, Urea, Creatinine, Blood sugar, Peripheral smear, ECG, Chest X-ray, Ultrasound abdomen and Upper GI scopy.

INCLUSION CRITERIA:

1. Age >20

2. Undiagnosed cases with symptoms suggestive of dyspepsia

EXCLUSION CRITERIA:

1. Known case of decompensated liver disease

2. Pregnant women

3. Known case of ischemic heart disease

4. Known case of malignancy

5. Known case of psychiatric disorder

6. Known case of retroviral infection

7. Known case of CRF

ANALYSIS OF CLINICAL FEATURES, ULTRASOUND ABDOMEN AND UPPER GI ENDOSCOPY OF PATIENT PRESENTING WITH DYSPEPSIA

Name : IP.No:

Age: 20 - 30 ☐ 30-40 ☐

40-50 ☐ 50-60 ☐ >60 ☐

Gender : Male ☐ Female ☐

Occupation:

Symptoms

Dysphagia	Weight loss	odynophagia	
Retrosternal pain	Heart Burn	Nausea	Vomiting
Loss of appetite	Yellowish urine	Belching	Abd.Disocmfort
Epigastric pain	Bloating	indigestion	flatulence
Post prandial fullness	Acid regurgitation	Early satiety	diarhea

Sour taste in throat Air swallowing leads to belching

Chest pain

Pricking

Burning

Past H/o:

Peptic ulcer	Gastrooesophageal reflux	Biliary tract disease
Gall bladder disease	Pancreatitis	Inflammatory bowel disease
Diabetes Mellitus	Stomach infiltrative disease	Abd.Malignancy
Hyperkalemia	Hypercalcemia	Carbohydrate malabsorbtion
Lactose intolerance		

Personal H/o

Smoking	Alcohol	Drug	Recent surgery
Caffine intake	Irregular meals	Eating heavy meals	
Constipation	Irregular bowel habits	Insomia	

Ultrasonogram of abdomen :

Upper GI scopy:

RBS

HB

PCV

TC

DC

MCV

Platelet counts

ESR

Peripheral Smear

S.Bilirubin

SGPT

SGOT

Stool Routine

Stool for occult blood

Urea

Creatinine

ECG

Chest X- ray

RESULTS

S.No	RBS	Hb	PCV	TC	DC	MCV	PLT ($*10^3$)	ESR	S.Bil	SGPT	SGOT	U.®	S. Oc	UR	CR
1.	87	11.8	20.1	5.5	N:51 L-32 M-6 E-9.6 B0.2	61	417	36	0.6	12	21	(-)	(-)	29	0.96
2.	110	19.2	58	6.7	N-58, L-34, M-O.5 E-0.2 B-01	106	149	01	1	22	30	(-)	(-)	34	1.2
3.	95	12. 5	35.7	11.4	N-66, L-24, M-6, E-2	84	305	10	0.9	40	42	(-)	(-)	32	0.9
4.	85	6.5	18.7	3.3	N-88, L-40, M-02 E-0	89	137	95	1	35	40	(-)	(-)	40	1.0
5.	90	12	30	3.5	N-80, L-16, M-4,	85	220	20	1	36	40	(-)	(-)	38	1.02
6.	105	11.4	33.8	9.0	N-53 L-40 M-6 E-1	80	422	77	0.7	36	37	(-)	(-)	17	0.7
7.	80	13	30	4.5	N-70 L-2 0 M-5 E-4	85	220	20	0.6	40	35	(-)	(-)	17	0.8
8.	90	12.4	37.9	51.5	N-90 L-0 M-0 B-0	94	903	66	0.3	46	35	(-)	(-)	25	0.6
9.	75	12.1	36.4	14.6	N-87 L-10 M-02 E-01	89	485	100	0.9	48	107	(-)	(-)	28	1.2
10.	218	8	20	5.0	N-70 L-20 M-7 B-2	70	80	50	1.7	18	25	(-)	(-)	49	0.97

S.No	RBS	Hb	PCV	TC	DC	MCV	PLT ($*10^3$)	ESR	S.Bil	SGPT	SGOT	U.®	S.oc	UR	CR
11	70	13	38	5.0	N-70 L-20 M-8	90	220	20	0.9	32	28	(-)	(-)	29	0.82
12.	78	15	44	9.1	N-67 L-24 M-5 B-.5 E-2.4	88	356	14	0.8	31	26	(-)	(-)	27	0.65
13.	157	7.3	22.7	8.8	N-66 L-27 M-01 B-0 E-06	79	102	14	1.3	45	89	(-)	(-)	63	1.03
14.	70	14.6	43.4	12	N-78 L-16 M-.7 E-.31 B-.2	88	358	10	0.7	29	36	(-)	(-)	41	0.79
15	137	13.7	41.1	6.3	N-80 L-13 M-04 E-03 B-01	93	182	28	2.1	30	34	(-)	(-)	17	0.85
16.	99	13	38.8	6.8	N-54 L-37 M-3 E-3.8 B-0.5	88	230	12	0.8	28	32	(-)	(-)	22	0.80
17.	88	13.2	37.4	7.7	N-67 L-20 M-4. B-0.5 E-6.5	94	291	10	0.6	22	30	(-)	(-)	16	0.4
18.	93	10.3	13.6	17.2	N-96 L-04 M-0 E-0 B-0	58	517	77	0.5	28	34	(+) *1	(+)	22	0.55
19.	84	14.2	41.9	7.6	N-61 L-29 E-08 M-01 B-01	89	345	97	13.2	140	119	(-)	(-)	17	0.92
20.	155	12.4	36.9	16.4	N-87 L-12 M-1 E-0 B-0	102	203	20	3.0	32	79	(-)	(-)	32	1.14

S.No	RBS	Hb	PCV	TC	DC	MCV	PLT ($*10^3$)	ESR	S.Bil	SGPT	SGOT	U.®	S.oc	UR	CR
21	76	9.9	27.8	9.9	N-76, L-20 M-03 E-01 B-0	100	61	14	12.9	49	130	(-)	(-)	17	0.93
22.	139	10.5	27.7	4.1	N-88 L-12 M-0 E-0 B-0	85	48	10	2.4	28	32	(-)	(-)	31	0.99
23.	70	13.6	39.5	20.2	N-45 M-01 L-50 E-04 B-0	89	237	65	2.9	32	28	(-)	(-)	19	0.8
24.	99	9.3	20.4	8.7	N-61, L-35 M-0 E-04 B-0	90	123	27	1.2	33	59	(-)	(-)	35	1.21
25	116	15.1	40.5	20.8	N-95. M-01 L-04 E-0 B-0	87	252	11	0.9	23	22	(-)	(-)	28	0.65
26.	277	11.5	32.2	14.2	N-72 L-20 M-4 E-2.7 B-07	79	269	40	0.8	20	21	(-)	(-)	31	0.84
27.	120	5.9	18.5	5.8	N-70 L-25 M-04 E-01 B-0	65	75	17	0.9	13	25	(-)	(-)	51	0.8
28.	97	8.1	25.1	11.3	N-54 L-29 M-01 E-06 B-0	56	603	12	32.3	117	190	(-)	(-)	12	0.9
29	98	10.1	30.8	6.6	N-71 L-15 M-8 E-3.7 B-0.1	98	551	63	0.5	14	16	(-)	(-)	21	1.04
30.	99	12.3	37	11	N-68 L-26 M-01 E-05 B-0	86	476	68	0.8	21	32	(-)	(-)	13	0.76

S.No	RBS	Hb	PCV	TC	DC	MCV	PLT ($*10^3$)	ESR	S.Bil	SGPT	SGOT	U.®	S.oc	UR	CR
31.	108	8.3	9.1	10.4	N-54 L-30 E-14 M-01 B-01	54	445	17	0.5	16	26	(-)	(-)	19	0.72
32.	84	12.9	39	6.4	N-64 L-2 M-03 E-05 B-01	94	166	38	1.7	27	44	(-)	(-)	13	0.8
33.	136	12.8	38	13	N-69 L-25 M-01 E-05 B-0	85	398	95	0.7	16	18	(-)	(-)	2.5	1.81
34.	90	14.4	42.5	7	N-52 L-37 M-6 E-2.8 B-0.9	86	276	11	0.3	28	21	(-)	(-)	25	1.0
35.	78	14.9	43.8	10.2	N-62, L-26, M-06 E-06 B-0	85	361	12	0.6	24	28	(-)	(-)	15	1.03
36.	70	11.5	33.3	9.2	N-94 L-0.7 M-01 E-01 B-0	103	206	82	0..8	21	23	(-)	(-)	32	0.83
37.	101	14	42.4	7.5	N-74 L-20 M-0 E-06 B-0	90	169	45	1.0	19	21	(-)	(-)	10	0.81
38.	86	14.1	43.2	8.0	N-58 L-30 M-7.1 E3.5 B-06	79	256	20	0.3	30	22	(-)	(-)	20	0.68
39.	77	13.3	38.5	7.8	N-56 L-33 M-5 E-4.8 B-0.4	87	187	13	0.8	31	20	(-)	(-)	16	1.76
40	93	15.9	47	7.9	N-74 L-22 M-03 E-01	93	339	08	0.6	26	20	(-)	(-)	20	1.03

S.No	RBS	Hb	PCV	TC	DC	MCV	PLT ($*10^3$)	ESR	S.Bil	SGPT	SGOT	U.®	S.oc	UR	CR
41	145	10.4	30.5	7.6	N-71 L-26 M-01 E-02	83	164	94	0.8	71	119	(-)	(-)	29	1.14
42	89	13.7	42.4	8.5	N-71 L-40 M-04 E-6	92	277	12	0.9	32	30	(-)	(-)	21	1.04
43	83	13.9	40	10.4	N-51 L-39 M-7. E-1.2 B-0.3	89	275	14	0.4	23	28	(-)	(-)	16	1.0
44	78	12.9	38	97	N-58 L-32 E-1.2 M-7 B-.03	82	301	33	0.6	17	21	(-)	(-)	15	1.0
45.	115	14.9	43	6.3	N-53 L-29 E-7.4 M-6 B-0.3	101	234	10	0.7	16	28	(-)	(-)	22	0.78
46.	79	12.4	36.5	8.3	N-60 L-29 M-5 E-3.4 B-0.6	84.5	238	32	08	17	24	(-)	(-)	32	1.46
47.	93	12.4	38.1	6.7	N-63. L-28 E-1.2 M- 6.6 B-01	80	256	13	0.8	18	28	(-)	(-)	15	0.72
48	90	8.3	20	7.3O	N-44 L-33 E-1 B-2	75	229	42	1	15	13	(-)	(-)	35	1.3
49.	120	9.4	28.3	13.2	N-81 L-10 E-6 M-1	88	214	13	0.9	35	40	(-)	(-)	40	0.8
50.	90	10.5	31.5	6.9	N-78 L-18 M-3	92	118	71	2.5	30	52	(-)	(-)	15	0.9

S.No	RBS	Hb	PCV	TC	DC	MCV	PLT ($*10^3$)	ESR	S.Bil	SGPT	SGOT	U.®	S.oc	UR	CR
51	90	2.4	9.1	5.1	N-48 L-49 M-1 E-1	49	330	126	1	9	12	(-)	(-)	15	1
52	124	16.4	48.3	11.7	N-87 L-7.4 M-4 E-1	105	318	19	1.7	142	79	(-)	(-)	24	0.9
53	100	16.2	47	15.5	N-83 L-10 M-4 E-1	88	280	20	0.5	42	22	RBC-8	(-)	40	1
54	240	8.3	24.6	11.9	N-70 L-25 M-4 E-1	85	389	78	0.7	11	14	(-)	(-)	17	1
55	160	12	35	7	N-70 L-20 M-5 E-5	90	220	63	1	155	40	(-)	(-)	14	1
56	127	7.2	21	8.6	N-83 L-15 M-1 E-1	83	275	53	3.4	172	670	(-)	(-)	50	1.37
57	80	12	35	6	N-70 L-25 M-4 E-1	85	324	20	1	35	34	(-)	(-)	30	1
58	119	10.6	30	3	N-70 L-28 M-1 E-1	95	223	38	0.5	10	19	(-)	(-)	15	0.8
59	90	11	35	4	N-70 L-28 M-1 E-1	80	220	25	0.9	35	40	(-)	(-)	28	1.13
60	85	9.7	28	8.9	N-88 L-4 M-7 E-1	93	287	25	3.4	29	43	(-)	(-)	15	0.84
61	115	8.4	25	10	N-85 L-10 M-3 E-1	70	334	74	0.3	12	15	(-)	(-)	40	1.45
62	83	15.2	44	7.8	N-66 L-38	89	278	14	1.7	37	49	(-)	(-)	17	0.82

S.No	RBS	Hb	PCV	TC	DC	MCV	PLT (*103)	ESR	S.Bil	SGPT	SGOT	U.®	S.oc	UR	CR
63	103	14.5	43	16	N-74 L-14 M-7 E-3	92	371	75	1.5	52	68	RBC-40 WBC-20	(-)	24	0.7
64	100	13.3	39	11	N-86 L-7 M-5 E-1	90	243	6	0.8	10	21	(-)	(-)	17	0.5
65	232	13	38	17	N-84 L-8 M-5 E-1	89	218	63	1	21	30	(-)	(-)	18	0.8
66	110	12	37	5	N-42 L-46 M-8 E-1	80	176	6	0.4	13	14	(-)	(-)	30	1.1
67	110	10	30	11.5	N-74 L-20 M-4 E-1	84	326	11	0.3	19	13	(-)	(-)	40	1.01
68	91	4.7	16	6.9	N-64 L-25 M-6 E-5	53	436	66	0.7	41	31	(-)	(-)	28	0.86
69	102	12	38	11.9	N-56 L-31 M-9 B-1	81	379	45	0.7	21	31	WBC-30	(-)	30	1.0
70	90	12	38	7.8	N-75 L-18 M-5 E-1	82	378	9	1	31	41	(-)	(-)	25	1
71	118	8.9	29	8.3	N-73 L-22 M-3 E-2	66	338	17	.6	12	20	(-)	(-)	30	0.98
72	90	12	35	6	N-70 L-20 M-3 E-2	83	220	20	0.7	30	23	(-)	(-)	25	0.9
73	110	11.4	34.6	20.3	N-80 L-15 M-3 E-1	89	374	55	1.4	172	381	(-)	(-)	30	1
74	80	12.3	35	7.0	N-56 L-31 M-5 E-1	85	220	20	.9	30	20	(-)	(-)	20	0.9

S.No	RBS	Hb	PCV	TC	DC	MCV	PLT (*103)	ESR	S.Bil	SGPT	SGOT	U.®	S.oc	UR	CR
75	120	11	34	10	N-58 L-28 M-8 E-1	81	357	28	0.4	9	9	(-)	(-)	24	0.75
76	90	10.3	30	3	N-69 L-29 M-2 E-1	80	496	30	1	20	20	(-)	(-)	10	.6
77	132	16.6	48	6.6	N-62 L-31 M-3 E-1	100	214	3	0.7	42	31	(-)	(-)	25	0.9
78	120	11.3	33	4.8	N-42 L-50 M-5 E-1	113	265	27	1	20	25	(-)	(-)	20	1
79	110	10	30	11.5	N-74 L-20 M-4 E-1	84	326	11	0.3	19	13	(-)	(-)	40	1.01
80	124	16.4	48.3	11.7	N-87 L-7.4 M-4 E-1	105	318	19	1.7	142	79	(-)	(-)	24	0.9

PERIPHERAL SMEAR:

1. Microcytic hypochromic anemia
2. Polycythemia
3. Normal study
4. Normocytic normochromic aneamia
5. Normal study
6. Normal study
7. Normal study
8. Neutrophic, Leukocytosis and shift to left and marked thromocytosis
9. Normal study
10. Microcytic hypochromic anemia with thrombocytopenia
11. Normal study
12. Normal study
13. Microcytic hypochromic anemia with mild thrombocytopenia
14. Normal study
15. Normal study
16. Normal study
17. Normal study
18. Microcytic hypochromic anemia with neutrophilic leukocytosis and thrombocytosis
19. Normal study
20. Macrocytic anemia

21. Normocytic normochromic anemia with thrombocytopenia with mild leukocytosis
22. Normochronic normocytic anemia
23. Normal study
24. Normochronic normocytic anemia
25. Normal study
26. Microcytic hypochromic anemia
27. Microcytic hypochromic anemia with thrombocytopenia
28. Microcytic hypochromic anemia with thrombocytosis
29. Normrocytic hypochromic anemia with thrombocytosis
30. Normal study
31. Microcytic hypochromic anemia with relative thrombocytosis and eosinophilia
32. Normal study
33. Normal study
34. Normal study
35. Normal study
36. Normal study
37. Normal study
38. Normal study
39. Normal study
40. Normal study

41. Microcytic hypochromic anemia
42. Normal study
43. Normal study
44. RBC's are normocytic and normochromic with mild esinopilia
45. Normal study
46. Normal study
47. Normal study
48. Normocytic normochromic to microcytic hypochromic anemiawith eosinphilia
49. Normal study
50. Normocytic normochromic anemia with neutrophilia and thrombocytopenia
51. Dimorphic anemia (predominently microcytic hypochromic)
52. Megaloblastic anemia
53. Normal study
54. Normocytic normochromic anemia
55. Normal study
56. Dimorphic anemia with neutrophilia
57. Normal study
58. Normocytic normochromic anemia with leucopenia
59. Normal study
60. Normochromic normocytic anemia

61. Microcytic hypochromic anemaia with relative neutrophilia.
62. Normal study.
63. Normal study
64. Normal study
65. Normal study
66. Normal study
67. Normochromic normocytic anemia
68. Microcytic hypochromic anemia with thrombocytosis
69. Normal study
70. Normal study
71. Microcytic hypochromic anemia
72. Normal study
73. Mild Leucoytosis
74. Normal study
75. Normal study
76. Normocytic hypochromic to microcytic hypochromic anemia with leucopenia and thrombocytosis
77. Normal study
78. Megablastic anemia
79. Normocytic normochromic anemia
80. Macrocytic hypochromic anemia

USG ABDOMEN :

1. Fatty liver
2. Mild increase in renal echoes – for renal parameter correlation and bilateral renal cortical cyst and left renal calculus and bilateral minimal pleural effusion.
3. Normal study
4. Mild splenomegaly
5. Normal study
6. Mild splenomegaly. Right renal cortical cyst
7. Normal study
8. Normal
9. Right lobe hepatic lesions, portal ve3in thrombosis, minimal right subscapular collection, minimal right paracolic gutter collection, grade 2 prostatomegaly
10. Cirrhosis of liver with portal HT
11. Normal study
12. Right iliac fossa probe tenderness suggested CT if clinically indicated
13. Normal study
14. Acute pancreatitis with peripancratic fluid focal hypoerechoic area in region of collection

15. Hepatomegaly – suggestive of chronic splenomegaly with ascites, gall bladder shidge, liver disease left renal cortical cyst thrombosis in portal vein.
16. Normal study
17. Internal echoes in urinary bladder to rule out urinary tract infection
18. Normal study
19. Multiple peripancreatic superior mesenteric, paraortic and right iliac lymphadenopathy and dilated HBSR, hepatic dult and CBD and gall bladder shidge- suggested further evaluation
20. Hepatomegaly with chronic liver disease
21. Chronic liver disease, moderate ascites, Edematous gall bladder wall
22. Fatty liver
23. Normal study
24. Fatty liver
25. Para Aortic lymphadenopathy
26. Normal study
27. Cirrhosis of liver, Moderate splenomegaly, gall stones, early features of portal HT, mild prostatomegaly.
28. Gall bladder calculus with minimal wall thickening, mild splenomegaly
29. Severe right pleural fluid.
30. Para – aortic lymphadenopathy,
31. Normal study

32. Mild splenomegaly with ascites
33. Normal study.
34. Normal study
35. Fatty liver with mild hepatomegaly
36. Mild hepatomegaly with altered ehoes bilateral minimal pleural effusion.
37. Right renal calculi, mild prostatomegaly tickened gall bladder wall
38. uncomplicated right overian cyst
39. Normal study
40. Mild fatty infection of liver, bilateral renal calculus
41. Cirrhosis of liver. Moderate ascites and minimal bilateral pleural effusion, gall bladder calculi with sludge
42. Normal study
43. Fatty liver
44. Normal study
45. Normal study
46. Mild hepatomegaly with Fatty liver
47. Fatty liver gall stones
48. Liver-subtle coarse echo pattern
49. Normal study
50. Cirrhosis of liver with portal hypertention with reversal of flow,moderate ascitis.
51. Normal study

52. Mild hepatomegaly with fatty infiltration thin ascitis+
53. Mild hepatomegaly
54. Chronic pancreatitis, dilated main pancreatitic duct with multiple intraductal calculi
55. Normal study
56. Cirrhosis of liver with portal hypertension
57. Normal study
58. Chronic parenchymal liver disease, portal hypertension ,moderate splenomegaly
59. Normal study
60. Normal study
61. Mild spleenomegaly
62. Fatty liver
63. Mild hepatomegaly with fatty liver.
64. Normal study
65. Gall bladder stone
66. Normal study
67. Normal study
68. Grade 1 prostatomegaly
69. Normal study.
70. Normal study
71. Normal study

72. Normal study
73. Cholilithiasis
74. Normal study
75. Normal study
76. Normal study
77. Normal study
78. Normal study
79. Mild splenomegaly
80. Normal study

UPPER GI SCOPY:

1. Normal study
2. Erythematous Gastritis
3. Lax- lower esophageal sphincter Grade I Reflux esophagitis
4. Lax- lower esophageal sphincter solitary nodule at 3 cm mild gastritis
5. Grade A Reflux esophagitis
6. Normal mucosal study
7. Erosive Gastritis
8. Hiatus Hernia
9. Normal mucosal study
10. Esophageal varices, grade 2, mild portal gastropathy.
11. Mild antral gastritis
12. Normal mucosal study
13. Normal mucosal study
14. Normal study
15. Mild portal gastropathy. Grade I esophageal varices
16. Normal mucosal study
17. Normal study
18. Normal mucosal study
19. Periampullary growth
20. Esophageal varies- grade I-II, mild portal gastropathy

21. Esophageal varies - grade I- II, endoscopic variceal ligation done. Mild portal gastropathy.
22. Esophageal varies - grade I, Mallory weiss Tear, Mild portal gastropathy.
23. Normal study
24. Esophageal varies - grade I, II, Mild portal gastropathy.
25. To rule out limits plastica, to rule out infiltrating ca, stomach
26. Normal study
27. Scleroced varices, solieified fundal varices mild portal gastropathy.
28. Normal study
29. Atrophic gastritis
30. Growth esophagus, malignant stricture
31. Normal study
32. Normal study
33. Antral gastritis
34. Normal study
35. Normal study
36. Normal study
37. Normal study
38. Normal study
39. Normal study
40. Grade I reflux esophagitis

41. Esophageal varices Grade II – III, mild portal gastropathy, small fundal varices, OGV type II
42. Grade II reflux esophagitis, Erosive gastroduodenitis, healing duodenal ulcer, anterior wall
43. Antral gastritis
44. Normal study
45. Ca esophagus: 23-27 malignant stricuture endoscopic dilatation done
46. Grade I reflux esophagitis
47. Erosive duodenitis
48. Antral gastritis
49. Antral gastritis,Active duodenal ulcer
50. Esophageal varices :grade 1 with mild portal gastropathy
51. hiatus hernia prolapse gastropathy
52. Normal study
53. Grade 1 reflux esophagitis erosive antral gastritis
54. Lax lower esophagial sphincter,grade 1 esophagial varices, erosive antral gastrits
55. Lax lower esophageal sphincter with grade 1 reflux esophagitis with esophageal

    Candidiasis
56. Grade II-III Esophagial varices with mild portal gastropathy with small fundal varices with multiple gastric ulcer

57. Hiatus hernia with lax lower esophageal sphincter, grade 1 reflux esophagitis.
58. Esophageal varices-grade 1,portal gastropathy
59. Lax lower esophagial sphincter,grade II reflux esophagitis
60. Severe esophagial candidiasis
61. Normal study
62. Lax lower esophageal spinchter-grade 1 reflux esophagitis,
63. Normal study
64. Normal study
65. Esophageal candidiasis
66. Normal study
67. Small hiatus hernia ,grade 1 reflux esophagitis, prolapse gastropathy, Carcinoma stomach-mid body
68. Grade1 reflux esophagitis
69. Normal study
70. Erosive gastroduodenitis
71. Mild antral gastritis
72. Normal study
73. Normal study
74. Mild erythematous gastritis
75. Erosive duodenitis
76. Normal study

77. Hiatus hernia, grade 1 reflux esophagitis
78. Lax lower esophageal sphincter,Grade 1 reflux esophagitis, Antral gastritis
79. Antral gastritis
80. Normal study

DISCUSSION:

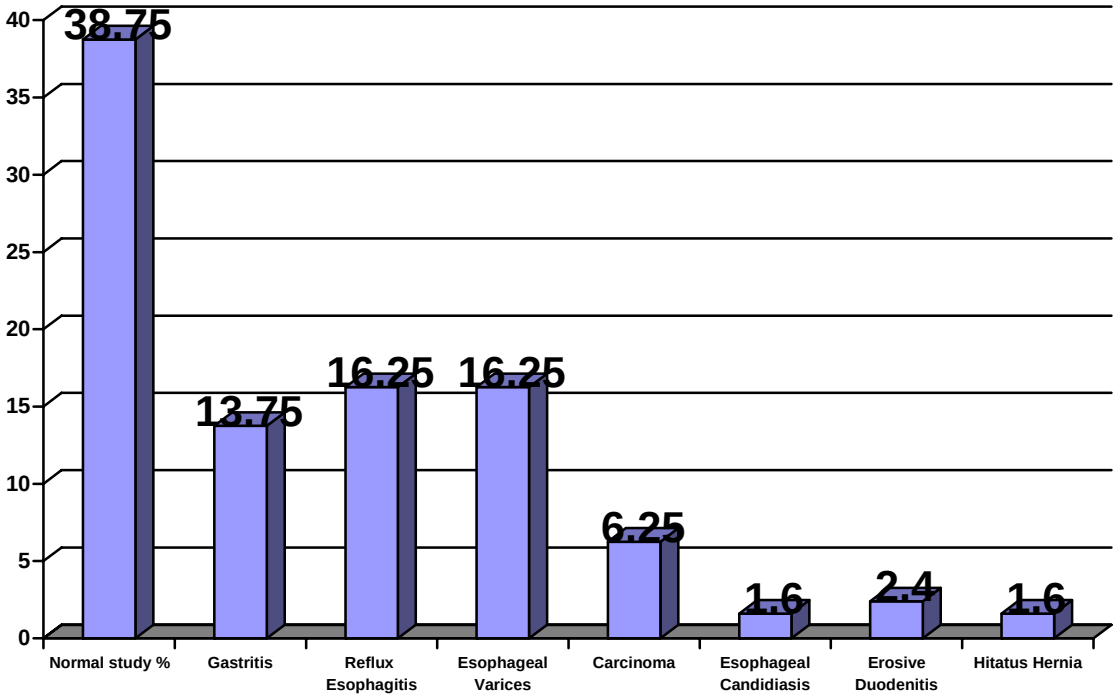
Patient with symptoms of dyspepsia, (both inpatient and out patients) were included in this retrospective study conducted between September 07- August 08. The patient's records included upper GI scopy and ultra sound abdomen and other basic investigations like complete blood count, Urea, Creatinine, Random Blood sugar, Urine Routine, Stool for occult blood, Peripheral smear.

From these investigations, the results were studied and analysis was made.

Data analysis

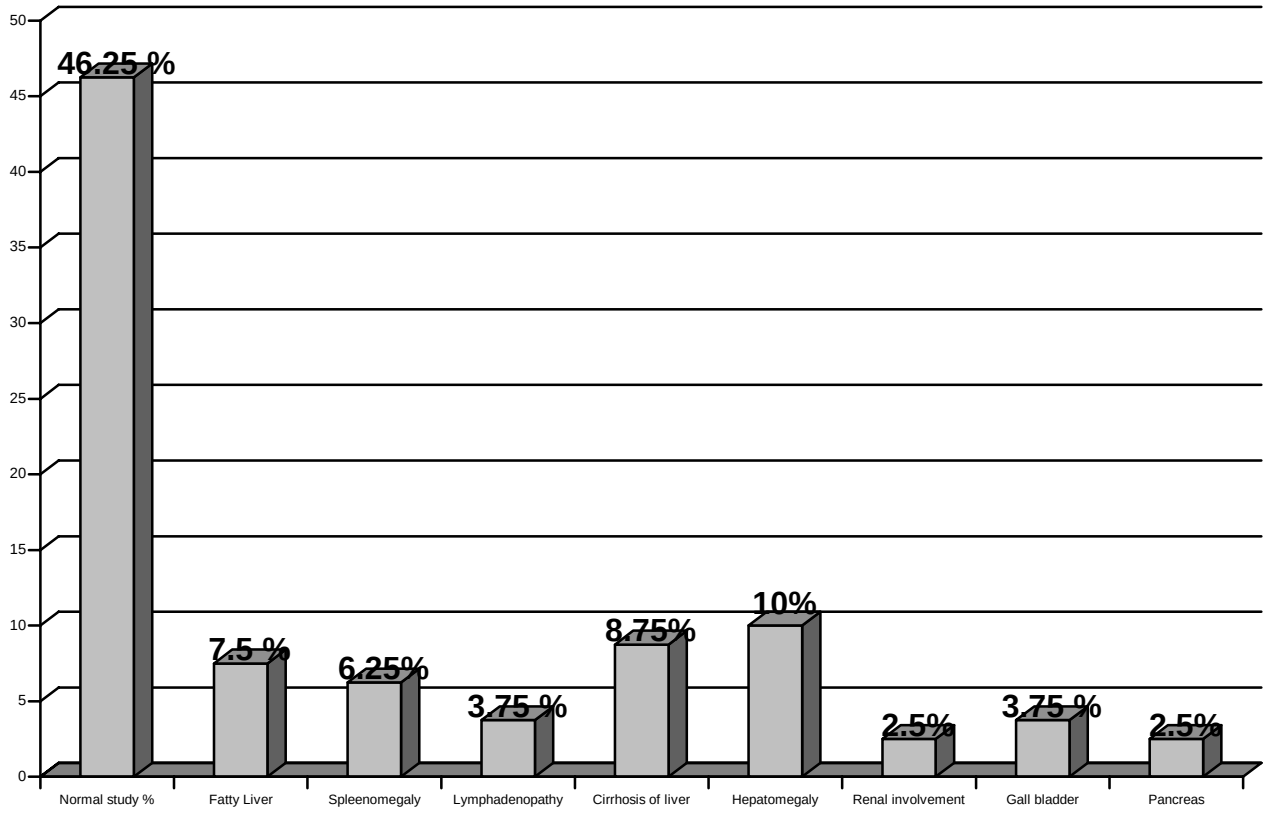
Upper GI scopy:

Normal study	H.Pylori Gastritis	Reflux Esophagitis	Esophageal Varices	Gastroesophageal/ Gastric Carcinoma	Esophageal Candidiasis	Erosive Duodenitis	Hitatus Hernia
38.75%	13.75%	16.25%	16.25%	6.25%	1.6%	2.4%	1.6%



ULTRA SOUND ABDOMEN:

Normal study	46.25%
Fatty Liver	7.5 %
Spleenomegaly	6.25 %
Lymphadenopathy	3.75 %
Cirrhosis of Liver	8.75 %
Hepatomegaly	10%
Renal involvement	2.5 %
Gall bladder	3.75 %
Pancreas	2.5 %



From the above UGI scopy analysis it is evident that,

Nearly 40 % of the patients were Normal.

H.Pylori gastritis in 13.75 % of the patients, H.Pylori tests was done (Rapid Urease study and also by antral biopsy).

Reflux oesophagitis in 16.2 % of the patients and gastroesophageal / gastric carcinoma seen in 6.25%.

The esophageal varices was recorded in 16.2 % these patient's ultrasound abdomen shows evidence of cirrhosis of liver and others like esophageal candidiasis, erosive duodenitis, Hiatus hernia shows 1.6 %, 2.4 %, 1.6% respsectively.

Also Ultra sound abdomen findings shows normal study in 46.25% of patient.

Fatty liver in 7.5 %, Gall stone in 3.75 % and 16.2 % of patients shows evidence of cirrhosis of liver, 2.5 % of patient with renal involvement presented initially with features of dyspepsia.

In Many studies like, Talley et al 1998 upto 60% of patients presenting with dyspepsia are diagnosed as having functional dyspepsia, defined as at least a 3 month history of dyspepsia in which there is no identifiable structural or biochemical etiology

Bernersen et al 1990; Johnson et al : 1991 went on to carry out diagnosting evaluation on community based samples and shows that up to 80% of individuals with dyspepsia had functional dyspepsia

Another studies Svedlund et a in 1985, 87 % of their patients are functional dyspepsia.

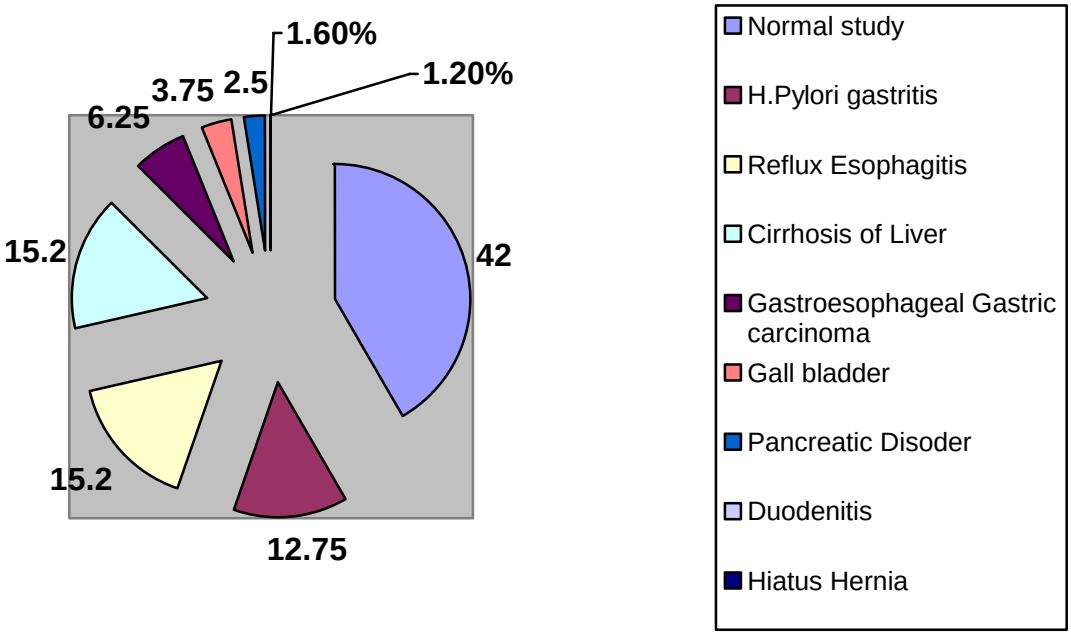
In 1998 Talley et al, employing a population based survery, showed that 29% of individual in the USA fulfilling Rome criteria for Functional Dyspepsia.

CONCLUSION

In our study, after analysing the clinical features, ultrasound abdomen and upper GI scopy the following results were observed,

Out of the 80 subjects,

Normal Study	H.Pylori gastritis	Reflux Esophagitis	Cirrhosis of liver	Gastroesophageal / Gastricarcinoma	Gall Bladder Disorder	Pancreatic Disorder	Duodenitis	Hiatus Hernia
42%	12.75%	15.2%	15.2%	6.25%	3.75%	2.%	1.6%	1.2%



Majority of the patients were found to be normal study. The possibilities for liver, pancreatic, gall bladder disorders should be kept in mind and further evaluation should be done wherever necessary

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