

**STUDY ON PREVALENCE OF GASTROESOPHAGEAL REFLUX
DISEASE IN REGURGITANT INFANT AND CHILDREN AND
EVALUATION OF IGERQ SCORE**

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CERTIFICATE

This is to certify that the dissertation titled “**STUDY ON PREVALENCE OF GASTROESOPHAGEAL REFLUX DISEASE IN REGURGITANT INFANT AND CHILDREN AND EVALUATION OF IGERQ SCORE**” submitted by **Dr.K.ELAYARAJA** to the Faculty of pediatrics, The Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the requirement for the award of M.D. Degree(Pediatrics) is a bonafide research work carried out by him under our direct supervision and guidance.

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This is submitted to **The Tamilnadu Dr. M.G.R. Medical University**, Chennai in partial fulfillment of the rules and regulations for the M.D. Degree Examination in Pediatrics.

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INTRODUCTION

GERD is the most common esophageal disorder in children of all ages. Gastroesophageal reflux (GER) is the involuntary passage of gastric content across the lower esophageal sphincter (LES) into the esophagus. It is a physiological event occurring in every individual, particularly after meals. Physiological GER is GER associated with absence of symptoms, accompanied occasionally by vomiting. It is a normal esophageal function that serves a protective role during meals or in the postprandial period. The regurgitated or vomited material can be considered as the tip of an iceberg. Physiological GER becomes pathological when reflux increases in frequency and intensity, and is associated with esophageal and respiratory symptoms. GERD is reflux associated with mucosal injury or symptoms severe enough to impair quality of life¹.

EPIDEMIOLOGY

Gastro esophageal reflux becomes evident in the 1st few months of life, peaks at 4 mo, and resolves in most by 12 months and nearly all by 24 months. Most infants have minor degree of reflux which may cause concern to the parents, but does not require extensive investigations and medications². Daily regurgitation is present in 50% of infants younger than 3 months, >66% at 4 months, but only 5% at 1 year³. Frequent regurgitation defined as regurgitation that occurs more than three times a day, complete resolution of regurgitation is frequent and expected by 10 months in 55%, by 18 months in 60-80% and by 24 months in 98%⁴

The incidence⁵ of significant reflux and associated complications in infants is about 1: 300 to 1:1000 ,the incidence decreases as age advances, so that infants with significant symptoms improves at 8 – 10 months when it starts to sit upright and transition to solid diet. Symptoms in older children tend to be chronic, with waxing and waning course. Treem et al⁶ studying the evolution of GER in older children found 50% to have spontaneous resolution or marked improvement.

GERD likely has genetic predispositions; family clustering of GERD symptoms, endoscopic esophagitis, hiatus hernia, Barrett's esophagus, and adenocarcinoma has been identified.

PATHOPHYSIOLOGY

Majority of newborn have an incompetent lower esophageal sphincter and it is known that with growth and development, the LES become competent and GER no longer be documented. The reasons for maturation of LES are not clear: but lengthening of the intra abdominal esophagus and upright position adds the benefit of gravity. The competence of the gastro-esophageal junction is maintained by anatomical and physiological factors.

ANATOMICAL FACTORS⁸

1. Diaphragm pinch mechanism at hiatus formed by right crus pulls the esophagus to right and downwards to narrow the lumen
2. Gastro esophageal angle of His, formed between lower end of esophagus and gastric fundus (flip valve mechanism)
3. Rosette like configuration of gastric mucosa (GUBAROFF valve)
4. Phrenico esophageal ligament
5. Gastro esophageal junction consists of inner circular and outer longitudinal fiber .the inner fiber, collar of HELVETIUS, corresponds to high pressure zone, gives strength to LES.

PHYSIOLOGY FACTORS^{8,9}

1. **HIGH PRESSURE ZONE:** The most important in preventing the reflux, is the pressure generated by LES, which is low at birth, increases as age advances. Normal tonic pressure is 20mmHg, < 10 mmHg is considered abnormal. But 5-7 mmHg is enough to prevent the significant reflux.
2. Intra abdominal segment of esophagus plays a key role in preventing the reflux by
 - A. increased intra abdominal pressure equally distributed to esophagus and stomach, hence collapses the intra abdomen segment
 - B. negative intra thoracic pressure of esophagus causes suction effect and closes intra abdomen esophagus
- 3) Mucosal folds of esophagus acts like a choke, causes reduction in flow on contraction
- 4)

THREE MAJOR TIERS OF DEFENSE SERVE TO LIMIT THE DEGREE OF GER¹⁰

1. First line of defense is anti reflux barrier consisting of the LES, diaphragmatic pinchcock, and angle of HIS
2. Second line of defense is esophageal clearance which assumes greater importance in limiting the duration of contact between the luminal contents and epithelium. Gravity and esophageal peristalsis serve to remove the volume from the esophageal lumen, where salivary and esophageal secretions to neutralize acid
3. Third defense is mucosal resistance, comes to play when esophageal clearance is defective¹⁰. Mucosal defense is divided into
 - Pre- epithelial (protective factors in swallowed saliva, Esophageal secretions containing bicarbonates, mucin, prostaglandinE2, Epidermal growth factor)
 - Epithelial (tight junction, intercellular glycoprotein)
 - Post epithelial factors.

Transient LES relaxation (TLESR)¹¹ is the primary mechanism allowing reflux to occur. It is defined as spontaneous, abrupt, prolonged, complete relaxation of LES with inhibition of crural diaphragm, occur independent of swallowing, reducing LES pressure to 0–2 mm Hg (above gastric), and last >10 sec.

TLESRs, regulated by a vagovagal reflex, composed of afferent mechanoreceptors in the proximal stomach, brainstem generator, and efferent via vagus activating inhibitor neuron releasing nitric acid to relax the LES¹². Gastric distention (postprandially, or due to abnormal gastric emptying or air swallowing) is the main stimulus for TLESRs³⁰

GER occurs due to

1. Normal intra abdominal pressure but abnormal LES TONE and increased frequency of TLESR
2. Increased intra abdominal pressure by straining/ respiratory efforts
3. Delayed gastric emptying

Factors determining the manifestation of reflux:

- Acidity of refluxate
- Volume of the reflux
- Duration of contact between the acid and esophagus
- Duration of esophagus exposure to acid

- Types of reflux (bile reflux)
- The defense mechanisms

CLINICAL MANIFESTATION OF GERD

GERD is a spectrum of disease that can best be defined as manifestation of esophageal or adjacent organ injury, secondary to the reflux of gastric contents into esophagus or into the oral cavity or airways. Regurgitation is the most common presentation of infantile GER, with occasional projectile vomiting^{3,10}.

In infancy and young children, verbal expression of symptoms is often vague, persistent crying, irritability, arching back, feeding and sleeping difficulties are possible equivalents of adult heartburn. Infants with GERD learn to associate eating with discomfort and thus develops aversion behavior³¹, affects parent –child interaction. Regurgitation produces caloric insufficiency and malnutrition in minority of infants. Failure to thrive secondary to GERD is a classic manifestation¹⁴.

GERD in older children is more adult -like ,heartburn has become the predominant GER symptom .Atypical symptoms , such as epigastric pain, flatulence, hiccups, chronic cough, asthma , chest pain ,hoarseness, earache accounts for 30-60% of GERD. Alarming symptom in infants¹⁵ are failure to thrive, irritability, feeding /sleeping difficulties, apnea. Alarming in children are weight loss, dysphagia, bleeding, anemia and chest pain¹⁶. Peptic stricture represent an undesirable point of reflux, manifested by dysphagia.

Occasional children present with neck contortions (arching, turning of head) designated as Sandifer syndrome¹⁷.

The respiratory presentations are also age dependent¹⁷: GERD in infants may manifest as obstructive apnea or as stridor or lower airway disease in which reflux complicates primary airway disease such as laryngomalacia or bronchopulmonary dysplasia. Otitis media, sinusitis, lymphoid hyperplasia, hoarseness, vocal cord nodules, and laryngeal edema have all been associated with GERD.

In contrast, airway manifestations in older children are more frequently related to asthma or to otolaryngologic disease such as laryngitis or sinusitis.

COMPLICATIONS OF GERD

1. Esophagitis can manifest as irritability, arching, and feeding aversion in infants; chest or epigastric pain in older children; and, rarely, as hematemesis, anemia, or Sandifer syndrome at any age¹⁷.

The severity of the complications is not invariably related to duration or severity of symptoms. Reflux esophagitis is reported 2-5% in general population¹⁸ and histologically occurs in 61-83% with significant reflux.

2. Prolonged and severe esophagitis leads to formation of strictures, generally located in the distal esophagus, producing dysphagia, and requiring repeated esophageal dilations and often fundoplication.

3. Long-standing esophagitis predisposes to metaplastic transformation of the normal esophageal squamous epithelium into intestinal columnar epithelium, termed **Barrett's esophagus**, a precursor of esophageal adenocarcinoma, common in whites. The main determining factor in the development of Barrett's is severity of reflux. Children with neurological impairment, chronic lung disease, esophageal atresia and chemotherapy have the most severe reflux and are at greatest risk of Barrett's esophagus¹⁹

NUTRITIONAL

1. Esophagitis and regurgitation may be severe enough to induce failure to thrive because of caloric deficits.
2. Anemia due to occult blood loss

EXTRAESOPHAGEAL: RESPIRATORY ("ATYPICAL" COMPLICATIONS)

1. Recurrent cough / recurrent pneumonia
2. Recurrent wheeze

GERD may produce respiratory symptoms by

1. Direct contact of the refluxed gastric contents with the respiratory tract (aspiration, laryngeal penetration, or microaspiration) or
2. By reflexive interactions between the esophagus and respiratory tract (inducing laryngeal closure or bronchospasm).

Differential diagnoses to consider in the evaluation of an infant or a child with chronic vomiting are milk and other food allergies, pyloric stenosis, intestinal obstruction (especially malrotation with intermittent volvulus), nonesophageal inflammatory diseases infections, inborn errors of metabolism, hydronephrosis, increased intracranial pressure, rumination, and bulimia¹⁷

DIAGNOSIS

For most of the typical GERD presentations, a thorough history and physical examination suffice to reach the diagnosis initially. This initial evaluation aims to identify the pertinent positives in support of GERD and its complications and the negatives that make other diagnoses unlikely.

Since reflux is common in infants, and there is no 'gold standard' investigation, interest has been focused on development of an infant GER questionnaire, (the Infant Gastro esophageal Reflux Questionnaire, the I-GERQ, and its derivative, the I-GERQ-R)²⁰, which also permit quantitative scores to be evaluated for their diagnostic discrimination .Most of the esophageal tests are of some use in particular patients suspected of GERD²¹

Radiological studies such as radiological contrast studies, scintigraphy and ultrasonography are techniques that evaluate postprandial reflux and barium study should not be the first line of investigation. Barium meal used to study the esophagus and upper gastrointestinal tract in children with vomiting and dysphagia to rule out

achalasia, esophageal strictures and stenosis, hiatal hernia, and gastric outlet or intestinal obstruction.

Extended **esophageal pH monitoring**²⁹ of the distal esophagus, no longer considered the sine qua non of a GERD diagnosis, provides a quantitative and sensitive documentation of acidic reflux episodes, but not all reflux are acidic. The distal esophageal pH probe is placed at a level corresponding to 87% of the nares-LES distance, based on regression equations. Normal values of distal esophageal acid exposure (pH <4) are generally established as <5–8% of the total monitored time, but these quantitative normals are insufficient to establish or disprove a diagnosis of pathologic GERD. The most important indications for esophageal pH monitoring are for assessing efficacy of acid suppression during treatment, evaluating apneic episodes in conjunction with impedance -metry and evaluating atypical GERD presentations. Dual pH probes, adding a proximal esophageal probe to the standard distal one, are used in the diagnosis of extra esophageal GERD, identifying upper esophageal acid exposure times of $\approx 1\%$ of the total time as threshold values for abnormality.

Endoscopy allows diagnosis of erosive esophagitis and complications such as strictures or Barrett's esophagus. There is poor correlation between endoscopic appearance and histopathology; hence esophageal biopsies are mandatory to exclude eosinophilic infiltrations and other causes like allergies.

Radionuclide scintigraphy²² Involves the ingestion of technetium labeled food or formula followed by scanning to detect the distribution of the isotope in the stomach, esophagus, and lungs. Potential advantages are ability to demonstrate non

acidic reflux, lesser radiation, assessment of the rate of gastric emptying and aspiration into the lung.

Impedance –metry, is a technique that measures electrical potential difference and not pH depend, distinguish acid and non –acid reflux, important in negative or normal endoscopy or pH metric findings.

Laryngotracheobronchoscopy evaluates for visible airway signs that are associated with extra esophageal GERD, such as posterior laryngeal inflammation and vocal cord nodules; it may permit diagnosis of silent aspiration (during swallowing or during reflux) by bronchoalveolar lavage with subsequent quantification of lipid-laden macrophages in airway secretions.

Esophageal manometry does not demonstrate the reflux but shows the pathophysiology of reflux by measuring the frequency and duration of TLESR, used along with pH metry, particularly for dysmotility disorder.

Modified BERNSTEIN ACID perfusion test: used when standard evaluations for GER have been negative but a high index of suspicion is present. Acidification of the distal esophagus reproduces the airway symptoms in this patient.

ESOPHAGEAL BIOPSY: Helps to determine the presence and severity of esophagitis and complications like peptic stricture, Barrett’s esophagus and exclude other causes.

HISTOLOGICAL CLASSIFICATION OF REFLUX ESOPHAGITIS ²³

O	Normal
1a	Basal zone hyperplasia
1b	Elongated stromal papillae
1c	Vascular ingrowth
2	Polymorpho nuclear cells in epithelium ,lamina propia
3	Polymorphs with epithelial defect
4	Ulceration
5	Abnormal columnar epithelium

ACCURACY OF DIAGNOSTIC MODALITIES²⁴

Barium swallow	:	50%
Manometry	:	41%
24 hours pH metry	:	95%
Esophagoscopy	:	50%
Esophageal biopsy	:	96%

MANAGEMENT

Phase

TREATMENT

1. Parental reassurance. observation. lifestyle changes exclude overfeeding.
2. Dietary treatment (decrease regurgitation, no decrease in GER) Thickened formula, thickening agents, hydrolysates in cow's milk allergy.
3. Alginates (some efficacy in moderate GERD, relative safe) Antacids only in older children
4. Prokinetics treats the pathophysiologic mechanism of GERD
5. Proton pump inhibitors (drug of choice in severe GERD) H2 receptor antagonists less effective than PPIs
6. Laparoscopic surgery (endoscopic procedures under evaluation)

Non-pharmacological therapy

Conservative therapy and lifestyle modification form the foundation of GERD therapy. Dietary measures for infants include normalization of feeding techniques, volumes, and frequency if abnormal.

Thickening of formula with a tbsp of rice cereal per oz of formula results in fewer regurgitation episodes, greater caloric density (30 kcal/oz), and reduced crying time, although it may not modify the number of non regurgitant reflux episodes. A short trial

of a hypoallergenic diet can be used to exclude milk or soy protein allergy before pharmacotherapy.²¹

Older children and adults should be counseled to avoid acidic or reflux-inducing foods (tomatoes, chocolate, and mint) and beverages (juices, carbonated and caffeinated drinks, alcohol).

Weight reduction for obese patients and elimination of smoke exposure are other crucial measures at all ages.²¹

Positioning measures are particularly important for infants, who cannot control their positions independently.

Seated position worsens infant reflux, due to increased intragastric pressure and should be avoided in infants with GERD. The supine and right lateral positions are associated with high incidence of GERD, but evidence that the supine position reduces the risk of sudden infant death syndrome, during sleep supine positioning is preferred²⁵.

When the infant is awake and observed, prone position and upright carried position can be used to minimize reflux. But some evidence suggests a benefit to left side position and head elevation during sleep.

Head elevation should utilize elevation of the head of the bed, rather than excess pillows, to avoid abdominal flexion and compression that might worsen reflux.

Pharmacotherapy is directed at ameliorating the acidity of the gastric contents or at promoting their aboral movement.

Antacids are the most commonly used antireflux therapy. Provide rapid but transient relief of symptoms by acid neutralization. Their efficacy is strongly influenced by time of administration, achieved after meals. The long-term regular use of antacids cannot be recommended because of side effects of diarrhea (magnesium) and constipation (aluminum) and rare reports of more serious side effects (osteopenia, neurotoxicity) on chronic use.

Histamine-2 receptor antagonists (H2RAs; cimetidine, famotidine, nizatidine, and ranitidine) are widely used antisecretory agents that act by selective inhibition of histamine receptors on gastric parietal cells. There is a definite benefit of H2RAs in treatment of mild-to-moderate reflux esophagitis.

H2RAs have been recommended as first-line therapy because of their excellent overall safety profile. Cimetidine appears to have severe endocrine and neurological side effects, hence not advised in children.. Commonly used is ranitidine with dose of 10mg /kg in 2 divided dose. The common problem with H2RA is rapid onset of tolerance (tachyphylaxis)²⁶.

Proton pump inhibitors (PPIs; omeprazole, lansoprazole, pantoprazole, rabeprazole, and esomeprazole) provide the most potent antireflux effect by blocking the hydrogen-potassium ATPase channels of the final common pathway in gastric acid secretion.

PPIs are superior to H2RAs in the treatment of severe and erosive esophagitis. Prodrug is converted to active form in acidic medium of stomach, hence preferred 15 minutes before meals, and tablets should not be crushed or chewed. The usual recommended omeprazole starting dose is 1mg/kg once daily and patient with extra esophageal manifestation needs higher dose (twice daily). PPIs promote healing of reflux oesphagitis and effective in maintenance therapy. Prolong use may lead to gastric bacterial flora overgrowth and hypergastrinemia causing enterochromaffin like cell hyperplasia.

Prokinetic agents include metoclopramide (dopamine-2 and 5HT-3 antagonist), bethanechol (cholinergic agonist), and erythromycin (motilin receptor agonist). Most of these increase LES pressure; improve gastric emptying or esophageal clearance. None affects the frequency of TLESRs.

Metoclopramide in infants is limited because of severe extra pyramidal effect and neuroendocrine problems. Domperidone, dopamine agonist is effective, better tolerated than metoclopramide. The recommended dose is 1mg/kg in 4 divided doses.

Cisapride, a 5HT₄ antagonist, increases LES tone, stimulates gastric emptying, via indirect release of acetylcholine from the myenteric plexus. It is only prokinetic with some evidence of efficacy. In general well tolerated, with most common adverse events of diarrhea and colic. Cisapride possesses antiarrhythmic properties, prolongs QT duration on co administration with macrolides. Ketoconazole, terfenadine²⁷. Dose is 0.6-0.8 mg/kg /daily in 3 divided doses given before meals.

Surgery

Surgery is reserved for infants and children with the most severe form of GERD who have failed maximal medical therapy²⁸. In most patients, indication for fundoplication is life threatening conditions like apnea or aspirations, recurrent pneumonia, severe esophagitis, large hiatus hernia. Fundoplication is done to strengthen the antireflux mechanism by wrapping the gastric fundus around the esophagus. Two types of surgery are available Nissen fundoplication (complete wrapping) and Thal fundoplication (partial wrapping) .The major complications of fundoplication are breakdown of wrap, intrathoracic herniation of wrap, dysphagia (due to excessive tightness of placcation),gas bloat syndrome. In many children (especially with neurological impairment) fundoplication is performed with gastrostomy to prevent gas bloat syndrome and initiate supplement feeding²¹. Strictures are best treated with balloon dilation. If the strictures permit passage of an endoscope, biopsies under the stenosis should be taken to exclude the Barrett's esophagus.

STUDY JUSTIFICATION

GERD is a highly prevalent gastro intestinal disorder and one of the common gastro intestinal diseases in clinical practice. GER is both a physiological event and a manifestation of disease and there is some controversy regarding the problem magnitude. So the prevalence of GERD in regurgitant children was taken for study .More over very little data is available in our population regarding the prevalence of GERD.

GERD is a spectrum of manifestations, includes esophageal and respiratory symptoms such as regurgitation, poor weight, refusal of feeds, irritability, recurrent pneumonia, recurrent cough, stridor, asthma. In most instance GERD can diagnosed clinically and investigations were required only in special situation like atypical presentation and unresponsive to conventional therapy. To avoid unnecessary investigations, a clinical evaluation standardized by a questionnaire was taken and its role in diagnosing the disease was evaluated.

REVIEW OF LITERATURE

S k Mittal et al, at, Maulana azad, New India, studied the prevalence of GERD by symptom profile in 602 babies of age 1-24 months. The primary care taker was interviewed with Orenstein designed questionnaire and a IGERD score was obtained.UGI endoscopy was carried out and biopsy was taken in cases of score >5.they found that 10% of subjects had score of >5 Suggestive of GERD and 25 (22%) of 112 regurgitant infants of 1-6 month, 46% of 30 regugitant babies of 6-12 month, 85% in 12-24 months of 20 babies with score >5 .scopy was done in 31 babies >5,showed endoscopic esophagitis in 16% and histological esophagitis in 92%.they concluded that Prevalence of symptoms suggestive of GERD are 13% in 1-6 month age,8.4% and 8.7% in 6-12 month and 12-24 month of age. Thus the prevalence declines as age advances.

Aggarwal et al, studied the reproducibility and validity IGER questionnaire in developing country in 602 infants of age 1-24 months. Mothers were interviewed with the preformed questionnaire and a GERD score were arrived.95 infants were subjected to 24 hours PH study with reflux index of > 10% as pathological in infants. Before the PH study, each was again interviewed by independent observer. Upper GI scopy with biopsy was performed in 35 cases. A good correlation was seen between the observers with a Pearson coefficient of 0.906.the mean GERD score was 4.6+/-3.99 compared to 3.54+/-3.96in normal .and sensitivity and specificity for the score were 43% and 79% compared to 86% and 85% in Orenstein study .they that the questionnaire was easily reproducible and adaptable but appears to be less valid than previous study.

Orenstein et al studied the prevalence of reflux symptoms in 100 normal infants and diagnostic validity of 25 point IGERQ score based on 11 items, provocative care taking practices using PH probe and biopsy they found that the normal infants have high incidence of reflux symptoms with daily regurgitation 40%, crying more than a hour 17%, arching 10%, daily hiccups 36% and many symptoms were significantly prevalent in GERD infants. The positive and negative predictive values were 1.00 and .94-.98, concluded that a simple questionnaire was a valid test with high positive and negative value

Aldo J F costa et al, studied the prevalence of pathological GER in regurgitant infants of less than 1 year based on ROMES criteria; infants with two or more regurgitation for more than 3 weeks, without history suggestive of complications. infants with bronchial asthma, neurological disorder, digestive tract surgery were excluded from the study. Then care taker were subjected to clinical and epidemiological evaluation. The prevalence was 11.5% with 95% CI 9.1 to 13.4% with prevalence 14.6% in 1-3 months, 13.55 in 4-6 months, 6.8% in 7-9 months age. They concluded that prevalence was similar to previous study and useful in identify the cases who do meet the criteria for IR and difficult cases.

Salvatore S et al, at Belgium, studied the predictive value of a questionnaire and the correlation between the pH study, histology, and clinical score. Mother of 100 normal infants and 100 infants suggestive of GERD were asked to fill 35 item based questionnaire. Infants suggestive of GERD were subjected to 24 hours PH study and biopsy taken for 44-100 infants. They concluded that regurgitation was significantly

seen (68% vs 45%, $p < .05$) in infants suggestive of GERD, A pathological pH study was found significant with pneumonia , apnea with fussing. But oesophagitis was present in 39% and 38% of infants with pathological PH study had normal biopsy, 53% of infants with histological esophagitis had normal pH study. The clinical score fails to detect 31% of GERD cases. concluded that there was poor correlation between clinical score, biopsy, and pH study, and questionnaire are poor predictive of severity of GERD.

Vandenplas Y et al, at Belgium, studied on diagnosis and treatment of gastro esophageal reflux disease in infants and children, recommended reassurance and dietary modification in infants with uncomplicated regurgitation. If symptoms persist, prokinetics is recommended before investigations. PH monitoring is recommended in atypical conditions. Cisapride is the drug of choice because of its efficacy and safety. If there is severe esophagi is, acid suppression in combination with prokinetics are recommended. In life threatening condition, and resistant to medical therapy, surgical procedure is advised.

Hamid Reza et al studied the relationship between chronic respiratory symptoms and GERD in fifty-two (4 months-10 years) children who were referred to pediatric surgery ward by 24 hours PH monitoring. Additionally, 10 patients with only one episode of pneumonia were evaluated as the control group. *Results* showed that PH monitoring revealed GER in 42.2% patients as a cause of their chronic respiratory symptoms GER was detected in (45.7%) patients with chronic cough, 40% with recurrent pneumonia , 18% with asthma, *Conclusion* : The possibility of GERD was significantly higher in study group (children with chronic respiratory symptoms) compared to control

group (p-value<0.01).

Mario C .vieira et al studied the validation of endoscopic findings against histological feature of distal esophagus for the diagnosis of reflux esophagitis in infants. They studied retrospectively the records of 167 patients, referred for investigation of reflux esophagitis and found that the endoscopy had a sensitivity of 45%, specificity of 71%, positive predictive and negative predictive value of 89%, and 21% respectively and accuracy of 50% and concluded that there was poor correlation between endoscopic and histological findings.

Walsh JK, et al, studied the temporal relationship of gastro esophageal reflux to apnea in 14 infants with abnormal GER scores and histories of prolonged apnea using 24 hr pH monitor, found that brief obstructive apneic episodes are common during the inset of GER and concluded that GER and apnea are not temporally related and may be two manifestation of a more developmental delay

AIM OF THE STUDY

PRIMARY AIM:

To study the prevalence of Gastroesophageal reflux disease in regurgitant children of age 6 to 24 months in tertiary care hospital.

SECONDARY AIM:

To evaluate the IGERQ score and to study clinical profile of GERD.

METHODOLOGY

Study design : Descriptive study

Place of study : Gastroenterology department, out patient

department, in patient department.

Institute of child Health and

Hospital for Children, Egmore, Chennai

Study period : September 2007 - September 2009

Study population : Regurgitant children of age 6-24 months.

Inclusion criteria : Children of age 6-24 months with

vomiting 2 times /day more than 3 weeks.

Exclusion criteria : Acutely ill child

Children with bronchial asthma,

Children with neurological disorder,

Children with any gastrointestinal surgery,

Child already on treatment for GERD,

Those deferred consent for

endoscopy/biopsy.

MANEUVER

All children between 6 -24 months of age, presented with vomiting for at least 3 weeks were enrolled for the study. Subjects were recruited from gastroenterology department, in patient ward, out patient department. After necessary exclusions, eligible children were considered and detailed history regarding the symptoms suggestive of GERD was elicited.

Clinical examination including basic anthropometry and systemic examination were done. Then mother was interviewed with a preformed validated questionnaire containing 11 items of total score of 25 and assigned a GERD score. A possible clinical diagnosis was made ,if necessary after a trail of empirical anti reflux therapy for 2 weeks. Before the investigations, again the mother was interviewed by another independent observer and assigned a GERD score. Basic investigations like Hb, total count, differential count, motion for occult blood, chest x-ray were done .Barium meal study was done to rule out upper gastro intestinal anatomical abnormality .

Children with symptoms suggestive of GERD were subjected to upper GI endoscopy after informed consent and looked for any macroscopic changes. Biopsy was restricted to children with atypical symptoms, complications, and normal endoscopy .Biopsies were taken from 3 cm above GE junction, preserved in 10% formalin and sent for histopathological examination. Children were observed for 24 hours, none of them had any problem after the procedure.

STATISTICAL ANALYSIS

Data analysis was aided by EPI info programme. Chi square, Odds ratio, P value were calculated for each variables. IGERQ score was evaluated by calculating sensitivity, specificity, positive predictive valve and negative predictive valve. P valve of $< .05$ was considered statistically significant.

OBSERVATIONS

PREVALENCE OF GERD IN REGURGITANT CHILDREN

$$= \frac{\text{Total number of children with GERD}}{\text{Total number of children studied}} \times 100$$

$$= 38/123 \times 100$$

$$= \mathbf{30.80\%}$$

PREVALENCE OF GERD IN REGURGITANT CHILDREN = **30.80%**

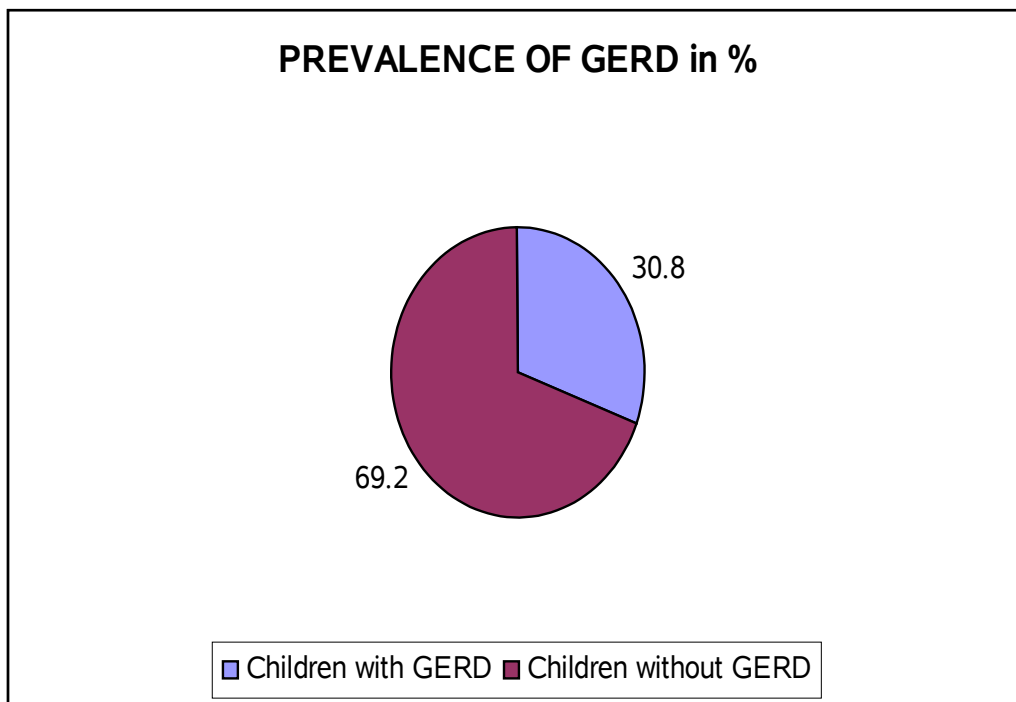


TABLE: 1

AGE WISE DISTRIBUTION OF GERD

Age in months	GERD present		GERD absent		Total	
	n	%	n	%	n	%
6 -11	18	47.4	45	52.9	63	51.2
12-18	9	23.7	24	28.2	33	26.8
19-24	11	28.9	16	18.8	27	22
total	38	100	85	100	123	100

GERD was seen in 18 (47.4%) children of 6-11 month of age, 9(23.7%) children of 12-18 month of age, 11 (28.9%) children of 19-24 month of age, showed the prevalence of GERD in regurgitant children decreases as age advances (P value 0.45).

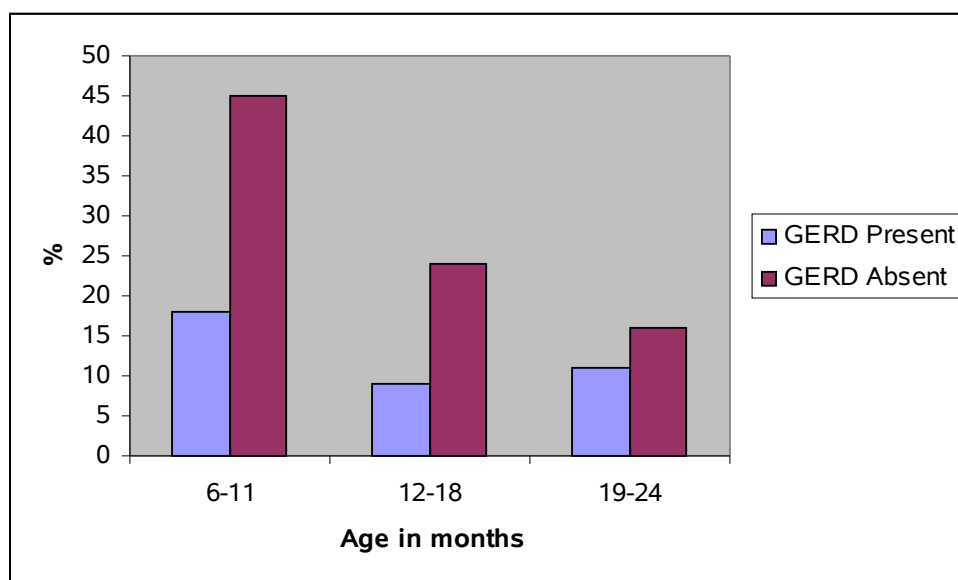


TABLE: 2

SEX DISTRIBUTION OF GERD

sex	GERD present		GERD absent		Total	
	n	%	n	%	n	%
Male	23	60	57	68	80	65.04
Female	15	40	28	32	43	34.96
Total	38	100	85	100	123	100

Out of 38 children with GERD, male constitutes 60% and female constitutes 40% of children with GERD. There was no significant difference in sex wise distribution of GERD in regurgitant children. ($P < 0.48$).

TABLE: 3

EPIDEMIOLOGICAL DISTRIBUTION OF GERD

Area	GERD present		GERD absent		Total	
	n	%	n	%	n	%
Urban	25	65.8	59	69.4	4	3.3
Rural	13	34.2	26	30.6	119	96.7
Total	38	100	85	100	123	100

Out of 38 children with GERD 65% were from urban where the rural population constitutes remaining 35%. There was no significant difference in epidemiological distribution of GERD.

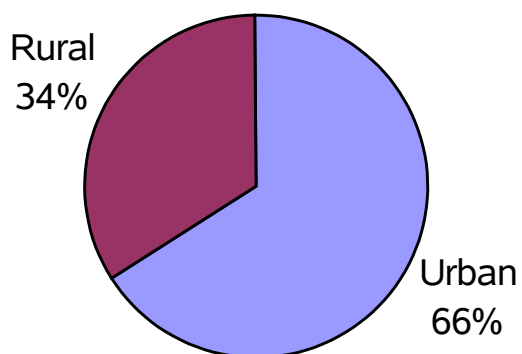


TABLE: 4

CORRELATION OF POOR WEIGHT GAIN TO GERD

Poor weight gain	GERD present		GERD absent		Total	
	N	%	N	%	n	%
present	17	44.7	6	7.1	23	18.7
absent	21	55.3	79	92.9	100	81.3
Total	38	100	85	100	123	100

Chi –square for the trend =24.5, P value < .0001

Poor weight gain was observed in 17 (44.7%) of 38 regurgitant children with GERD and 6 (7%) of 85 regurgitant children without GERD, suggesting poor weight gain was significantly correlated with GERD (P value < .0001)

TABLE:5

CORRELATION OF REFUSAL OF FEEDS TO GERD

Refusal of Feeds	GERD present		GERD absent		Total	
	n	%	n	%	n	%
present	17	44.7	9	10.6	26	21.1
absent	21	55.3	76	89.4	97	78.9
Total	38	100	85	100	123	100

Chi –square for the trend =18.3, P value < .0001

Refusal of feeds was seen in 17 (44.7%) of 38 regurgitant children with GERD and in 9 (10.6%) of 85 regurgitant children without GERD, suggesting Refusal of feeds was significantly correlated with GERD (P value < .0001)

TABLE: 6**CORRELATION OF IRRITABILITY TO GERD**

Irritability	GERD present		GERD absent		Total	
	n	%	n	%	n	%
Present	3	7.9	3	3.5	6	4.9
Absent	35	92.1	82	96.5	117	95.1
Total	38	100	85	100	123	100

Chi –square for the trend =1.07, P value =0.299

Irritability was seen in 3 (7.9%) of 38 regurgitant children with GERD against 3(3.5%) of 85 regurgitant children without GERD, there was no significant difference between irritability and GERD among regurgitant children. (P value 0.229).

TABLE:7**CORRELATION OF STRIDOR TO GERD**

Stridor	GERD present		GERD absent		Total	
	n	%	n	%	n	%
Present	3	7.9	1	1.2	4	3.3
Absent	35	92.1	84	98.8	119	96.7
Total	38	100	85	100	123	100

Chi –square for the trend =3.7, P value =0.05

Stridor was observed in 3(8%) of 38 children with GERD and 1 (1.2%) of 85 children with GERD, there was no significant difference between stridor and GERD among regurgitant children. (P value 0.05).

TABLE:8**CORRELATION OF GI BLEED TO GERD**

GI Bleed	GERD present		GERD absent		Total	
	n	%	n	%	n	%
Present	2	5.3	1	1.2	3	2.4
Absent	36	94.7	84	98.8	120	97.6
Total	38	100	85	100	123	100

Chi –square for the trend =1.8, P value = 0.175

GI bleed was seen in 2 (5.3%) of 38 regurgitant children with GERD against (1.2%) of 85 regurgitant children without GERD. There was no statistical significant difference between GI bleed and GERD among regurgitant children . (P value 0.175).

TABLE:9**CORRELATION OF RECURRENT PNEUMONIA TO GERD**

Recurrent pneumonia	GERD present		GERD absent		Total	
	n	%	n	%	n	%
Present	8	21.1	1	1.2	9	7.3
Absent	30	78.9	84	98.8	114	92.7
Total	38	100	85	100	123	100

Chi –square for the trend =15.2, P value < .0001

Recurrent pneumonia (documented pneumonia > 2 episodes) was present in 8 (21%) of 38 regurgitant children with GERD and in 1(1%) of 85 regurgitant children without GERD, suggesting recurrent pneumonia was significantly correlated with GERD (P value < .0001).

TABLE: 10**CORRELATION OF PALLOR TO GERD**

Pallor	GERD present		GERD absent		Total	
	n	%	n	%	n	%
Present	4	10.5	4	4.7	8	6.5
Absent	34	89.5	81	95.3	115	93.5
Total	38	100	85	100	123	100

Chi-square for the trend =1.4, P value = 0.226

Pallor was seen in 4 (10.5%) of 38 regurgitant children with GERD against 4 (4.7%) of 85 regurgitant children without GERD. There was no significant difference between stridor and GERD among regurgitant children. (P value 0.226).

TABLE: 11
CLINICAL DIAGNOSIS IN GERD

Clinical Diagnosis	GERD present		GERD absent		Total	
	n	%	n	%	n	%
Present	22	57.9	1	1.2	23	18.7
Absent	16	42.1	84	98.8	100	81.3
Total	38	100	85	100	123	100

Out of 38 regurgitant children with GERD, 58 % of cases were able to diagnosed. Clinically and failed to detect GERD in 42% of regurgitant children.

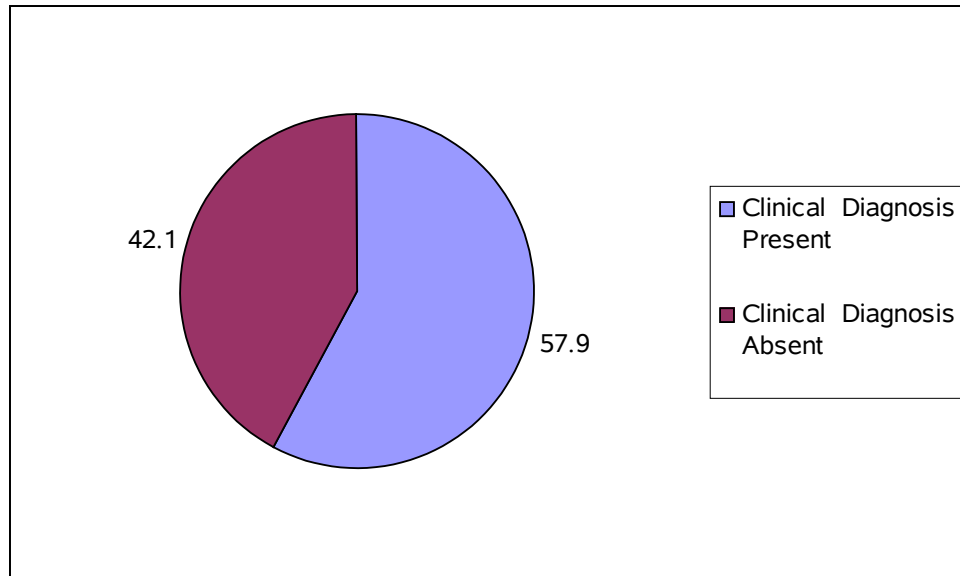


TABLE: 12

BARIUM STUDY IN GERD

Barium study	GERD present		GERD absent		Total	
	n	%	n	%	n	%
Present	8	21.1	1	1.2	9	7.3
Absent	30	78.9	84	98.8	114	92.7
Total	38	100	85	100	123	100

Out of 38 regurgitant children with GERD, BARIUM study showed the features of Gastro esophageal reflux in 8 (22 %) of children with GERD and abnormal in 1 in Regurgitant children without GERD, but not suggestive of GOR.

TABLE: 13

CORRELATION OF UPPER GI ENDOSCOPY TO GERD

Upper GI endoscopy	GERD present		GERD absent		Total	
	n	%	n	%	n	%
abnormal	18	47.3	6	7.1	24	19.5
normal	20	52.7	79	92.1	99	80.5
Total	38	100	85	100	123	100

Out of 38 regurgitant children with GERD, endoscopy found features suggestive of GERD in 18 cases (47%) and in 6 (7%) out of 85 regurgitant children with out GERD.

18 Cases shown the features suggestive of GERD as follows;

Confluent erosive patches (esophagitis) 27.7%

Gross GOR 16.6%

Gastric mucosal prolapse 16.6%

Lax lower esophageal sphinter 22.2%

Hiatus hernia 16.6%

6 abnormal findings –non specific mucosal changes

HISTOLOGICAL FEATURES OF ESOPHAGEAL BIOPSY:

Esophageal biopsies were taken in 16 regurgitant children who presented with atypical presentation and endoscopy negative children .out of 16, features suggestive of GERD was in seen in 14 regugitant children (88.6%) and in 2 children showed non specific changes.

1. Intra epithelial eosinophils	82%
2. Intra epithelial neutrophils	48%
3. Basal zone hyperplasia	34%
4. papillary lengthening	28%
5. Epithelial defect	14%
6. Dilated capillaries	8%

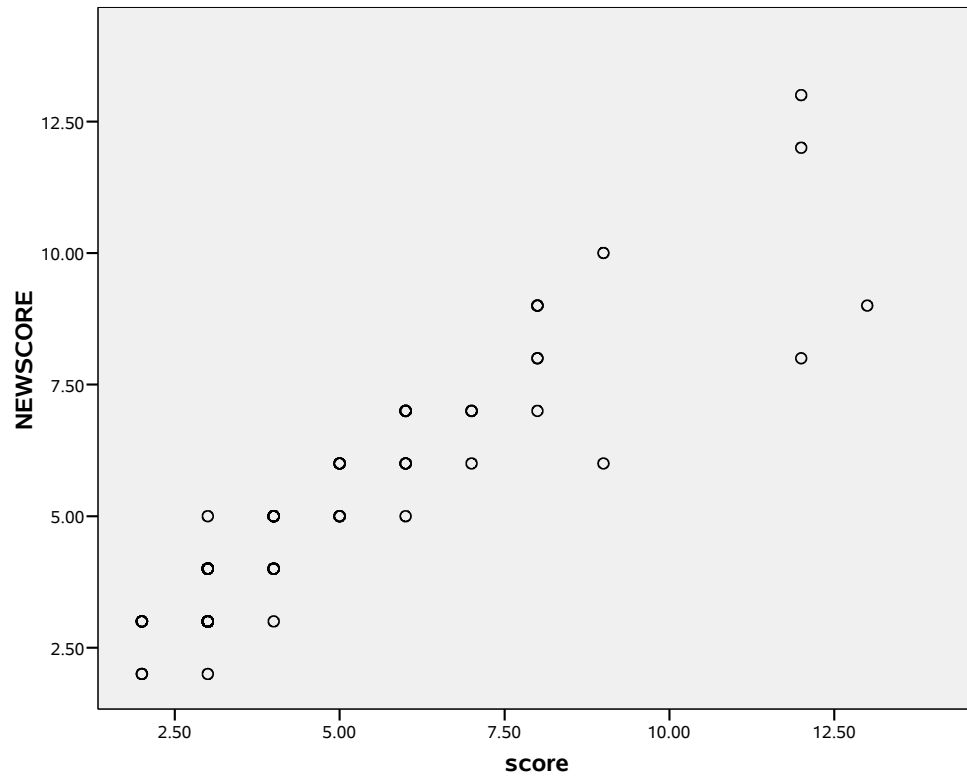
TABLE: 14

CORRELATION BETWEEN SCORE 1 AND SCORE 2

	N	Mean	S D	correlation
Score 1	123	4.24	2.11	0.92
Score 2	123	4.56	2	1

Each subjects were interviewed by two independent observer separately and score 1 and 2 were obtained. The mean score for both was approximately 4 and there was good correlation seen between the two score taken by two independent observer.

CORRELATION GRAPH BETWEEN SCORE 1 AND SCORE 2



PEARSON CO-EFFICIENT =0.92.

Correlation between two independent observer was good.

EVALUATION OF IGERQ SCORE

Score	GERD present		GERD absent		Total	
	n	%	n	%	n	%
> 5	32	84.2	3	3.5	35	28.4
< 5	6	15.8	82	96.5	88	71.6
Total	38	100	85	100	123	100

From the above data, the IGER Q SCORE was analyzed for score of > 5,

$$\text{Sensitivity} = 32/38 * 100$$

$$= 84\%$$

$$\text{Specificity} = 82/85 * 100$$

$$= 96\%$$

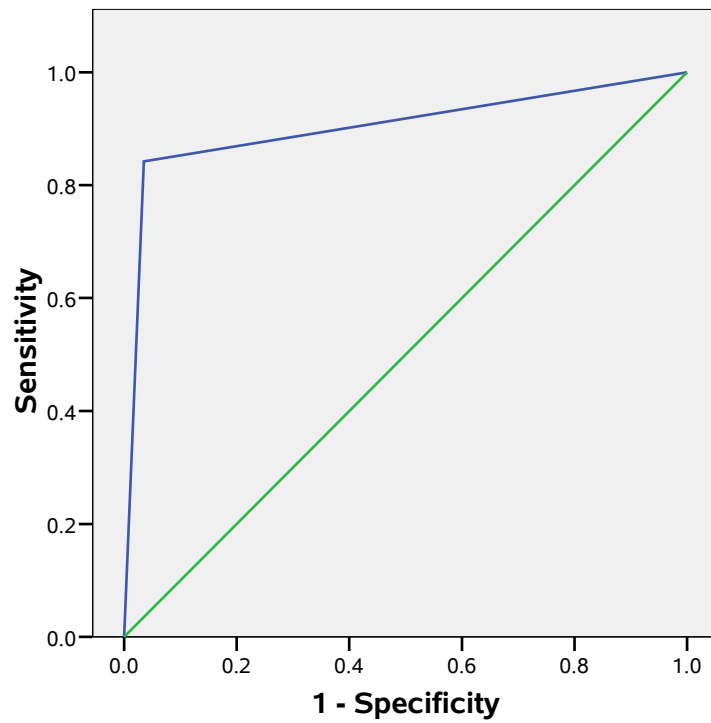
$$\text{Positive predictive value} = 32/35 * 100$$

$$= 91\%$$

$$\text{Negative predictive value} = 82/88 * 100$$

$$= 93\%$$

ROC Curve



Diagonal segments are produced by ties.

IGERQ score has been evaluated for the of score > 5 , and the values were

SENSITIVITY = 84%

SPECIFICITY = 96%

POSITIVE PREDICTIVE VALUE = 91%

NEGATIVE PREDICTIVE VALUE = 93%

DISCUSSION

Total number of regurgitant children of age 6-24 months enrolled in our study was 123. Out of 123, 38 children found to have GERD and the prevalence found to be 30.8% which was higher than previous study. Alto JF costo et al showed the prevalence was 12% but the age group studied in their study was 1-12 months.

In our study the age wise distribution of GERD was found to be 47.4% in age group of 6-11 months, 27.3% in 12-18 months and 28.9% in 19-24 months of age, showed the prevalence of GERD in regurgitant children decreases as age advances. This is similar to Mittal SK et al study.

There was no significant difference in sex wise distribution of GERD in regurgitant children. In our study male constitutes 60% and female constitutes 40% of children with GERD.

Poor weight gain was observed in 44.7% of regurgitant children with GERD and 7% of regurgitant children without GERD, suggesting poor weight gain was significantly correlated with GERD) this is similar to Mittal SK study, where 47% of children with IGERQ score > 5 had inadequate weight gain. Orenstein study showed 26% of children with GERD had inadequate weight gain .

Refusal of feeds was seen in 44.7% of regurgitant children with GERD and in 10.6% of regurgitant children without GERD, suggesting Refusal of feeds was

significantly correlated with GERD. Orenstein data showed 32% of GERD children had refusal of feeds.

Irritability was seen in 8% of regurgitant children with GERD against 3.5% regurgitant children without GERD, there was no significant correlation of irritability with GERD among regurgitant children. This in contrary to Mittal SK study.

Stridor was seen in 8% of children with GERD and 1.25% of children without GERD. There was no significant correlation of stridor with GERD among regurgitant children. Interestingly one case was associated with hypoplasia of right lung.

Recurrent pneumonia (documented pneumonia > 2 episodes) was present in 21% of regurgitant children with GERD and in 1% of regurgitant children without GERD, suggesting recurrent pneumonia was significantly correlated with GERD. Martin et al study suggested an association of GERD with recurrent pneumonia and reactive airway disease.

In our study 58 % of GERD were able to diagnosed clinically and failed to detect GERD in 42% of regurgitant children. Vandenplas suggested that clinical diagnosis of GERD was sufficient in most instances and investigations are required in typical cases or those not responding to conventional therapy.

In this study endoscopy features suggestive of GERD found in 47% of children with GERD and in 7% of regurgitant children with out GERD. Confluent erosive patches suggesting oesophagitis(27.7%), Gross GOR (16.6%), Gastric mucosal prolapse (16.6%), Lax lower esophageal sphincter (22.2%), Hiatus hernia (16.6%). 6 abnormal findings –nonspecific mucosal changes. Mittal SK study showed 51.6% Of children with score of > 5 had endoscopic esophagitis.

In this study, each subject was interviewed by two independent observer and separate score was obtained for each subjects. The mean score was found to be approximately 4 and there was good correlation seen by Pearson correlation of 0.94. This is similar to S K Mittal study where the correlation between two independent observers was 0.96.

In our study on evaluating the IGERQ score, Sensitivity was 84%, Specificity 96%, Positive predictive value 91%, Negative predictive value 93% for score >5. Orenstein study found to have sensitivity Of 86%, specificity of 85%, positive predictive value of 30% and negative predictive value 99%. Mittal S K study showed sensitivity of 43%. The evaluation of IGERQ scoring system was similar to Orenstein study except positive predictive value.

SUMMARY AND CONCLUSION

- The prevalence of GERD in regurgitant infant and children is 30.8%.
- The regurgitation is a sensitive marker of GERD and its significance increase as the age advances. Hence vomiting in older children strongly associate with occurrence of GERD.
- Refusal of feeds and poor weight in regurgitant children significantly correlated with GERD.
- Recurrent pneumonia in regurgitant children is strongly associated with GERD and hence GERD may be the cause for recurrent pneumonia in older children with vomiting.
- Clinical diagnosis of GERD is sufficient in most instances and a questionnaire may aid in diagnosis the disease.
- Investigations are required only in atypical and doubtful situations.
- Endoscopy with biopsy is useful in detecting most of the reflux esophagitis.
- IGERQ (infant gastro esophageal reflux questionnaire) score is easily adaptable.
- IGERQ score >5 has high specificity ,positive and negative predictive value but reproducibility in our population needs further evaluation.

BIBLIOGRAPHY

1. De Castecker J. Esophagus; heart burn *BMJ* 2001;323: 736-739.
2. Sutphen JL: Pediatric gastroesophageal reflux disease *Gastroentero. Clinic, North Am.* 19 :617 -629 ,1990.
3. Orenstein et al., 1996. Orenstein SR, Shalaby TM, and Cohn JF: Reflux symptoms in 100 normal infants: Diagnostic validity of the infant gastroesophageal reflux questionnaire. *Clin Pediatr* 1996; 35:607-614.
4. Shepherd R, Wren J, and Evans S. Gastroesophageal reflux in children. clinical profile, course and outcome with active therapy in 126 cases. *clin Pediatrics* 1987; 26:55-60.
5. Behrman E, Kleigman M. Ann M. Arvin. Bronchial asthma-Text book of Pediatrics 15 ed 628-640.
6. Orenstein et al., 2002. Orenstein SR, Shalaby TM, Barmada MM, et al: Genetics of gastroesophageal reflux disease: A review. *J Pediatr Gastroenterol Nutr* 2002; 34:506-510.
7. Ramaenofsky ML. Gastroesophageal reflux: Clinical manifestations and diagnosis .In Ashcraft K (ed) *Pediatric.esophageal surgery* .1986, 151-179
8. Boix Ochoa J: Gastroesophageal reflux: Welch KJ (Ed) *pediatric surgery* 4 Ed 1986:1032-1039.
9. Boix Ochoa J: The physiological approach to the management of gastroesophageal reflux .*J Ped surgery* 1986 (21):1032-1039.
10. Vandenplas Y, Hassall E. mechanisms of Gastroesophageal reflux and

Gastroesophageal reflux disease. *J Pediatrics Gastroenterol Nutr* 2002;35 :119-136.

11. Davidson and Omari, 2001. Davidson G, Omari TI: Path physiological mechanisms of gastroesophageal reflux disease in children. *Curr Gastroenterol Rep* 2001; 3:257-262.
12. Hirsch DP,Hollowy RH,Tytgat GNJ,Boeckxstaens GE.involvement of nitric oxide in human transient lower esophageal sphincter relaxations and esophageal primary peristalsis.*Gastroenterology* 1998; 115:1374-1380.
13. Orenstein et al., 1996. Orenstein SR, Shalaby TM, and Cohn JF: Reflux symptoms in 100 normal infants: Diagnostic validity of the infant gastroesophageal reflux questionnaire. *Clin Pediatr* 1996; 35:607-614.
14. Poets CF. Gastroesophageal reflux: a critical review of its role in preterm infants. *Pediatrics* 2004; 113: e 128 –e132.
15. The jury of the consensus conference. French –Belgian consensus conference on adult gastro-esophageal reflux disease ‘Diagnosis and treatment’ Paris,France21-22 january1999, *Eur J Gastroenterol Hepatol* 2000;12;129-137.
16. De Vault KR, Castello DO. Updated guidelines for the diagnosis and treatment of gastroesophageal reflux disease .*Am J Gastroenterol* 1999.94; 1434-1442.
17. Orenstein, 2000. Orenstein SR: *Pediatric gastroesophageal reflux disease*. In: Orland RC, ed. *Gastroesophageal Reflux Disease*, New York: Marcel Dekker; 2000.
18. Wienbeck M, Barnert J .Epidemiology of reflux disease and reflux esophagitis.*Scand J Gastroenterol* 1989;156;7-13.

19. Hassall E. Co-morbidities in childhood Barrett's esophagus. *J Pediatric Gastroenterol Nutr* 1997;25:255-260
20. Rothman et al., 2003. Rothman M, Orenstein SR, Kleinman L, et al: Development of a revised infant gastroesophageal reflux questionnaire. *Acta Gastroenterol Belgica* 2003;73 (presented at the Second European Pediatric Gastrointestinal Motility meeting, Bruges, Belgium, April 23-26, 2003).
21. Rudolph et al., 2001. Rudolph CD, Mazur LJ, Liptak GS, et al: Pediatric gastroesophageal reflux clinical practice guidelines: Guidelines for evaluation and treatment of gastroesophageal reflux in infants and children. *J Pediatr Gastroenterol Nutr* 2001; 32:S1-S31.
22. Lifschitz CH 'Clinical manifestations, diagnosis and management of gastroesophageal reflux disease in children in [www. Uptodate.com](http://www.Uptodate.com).
23. Leape LL. Esophageal biopsy in the diagnosis of reflux esophagitis. *J.Ped surgery* 1981;379-84.
24. Vivek M Rege . Gastroesophageal reflux. Recent advances in Pediatrics.Suraj Gupte (4) 88-110.
25. Vandeplass Y, Belli D, Benhamou P , et al ., A critical appraisal of current management practices for infant regurgitation-Eur *J Pediatr* 1997; 156;3343 -357
26. Huang JQ. Pharmacological and pharmacodynamic essentials of H₂ antagonists and PPIs for physician. *Best Pract Res Clin Gastroenterol* 2001;15;355-370.
27. Tonini M , De Ponti F ,Di Nucci A ,Crema. Cardiac adverse effects of gastrointestinal prokinetics.*Aliment Pharmacol Ther* 1999;13;1585-1591
28. Fonkalsrud et al., 1998. Fonkalsrud EW, Ashcraft KW, Coran AG, et al: Surgical

treatment of gastroesophageal reflux in children: A combined hospital study of 7,467 patients. *Pediatrics* 1998; 101:419-422.

29. Vandenplas Y, Goyvaerts H, Helven R, Gastroesophageal reflux, as measured by 24-hours pH monitoring, in 509 healthy infants screened for risk of sudden infant death syndrome. *Pediatrics* 1991;88 :834-840.
30. Sifirm D , Tack J ,Lerut T ,Janssens J.Transient lower esophageal sphincter relaxations and esophageal body muscular contractile response in reflux esophagitis. *Dig Dis Sci* 2000; 45; 1293-1300.

ANNEXURE 1

PROFORMA

Name : Age/ sex OP/IP. No.

Address:

History :

Recurrent vomiting

Poor weight gain

Irritability

Refusal of feeds

GI bleed

Apnea/stridor

Recurrent cough

On clinical examination :

Height :

Weight:

CVS :

RS :

P/A :

CNS :

Investigations :

Hb%

TC, DC :

Smear :

Chest x ray :

Barium meal :

Upper GI endoscopy :

Esophageal biopsy :

Diagnosis :

ANNEXURE -2

IGERQ – GERD SCORE: POINTS:

Questions:

How often does the baby usually vomit?

2- 3 times / day 1

3- 4 times / day 2

>5 times / day 3

How much does the baby usually vomit?

A teaspoon to a table spoon 1

A tablespoon to an ounce 2

An ounce or more 3

Does the vomiting seem uncomfortable for the baby? 2

Is the baby refused feedings even when hungry? 1

Does the baby have trouble in gaining weight? 1

Does the baby cry a lot during / after feeding? 3

Does the baby cry / fuss more than normal?

1 - 3 hrs 1

> 3 hrs 2

Do you think the baby hiccups more than most babies? 1

Does the baby spells of arching back? 2

Has the baby ever stopped breathing while awake and
struggling to breathe or turned blue 6

Total score 25

ABBREVIATIONS

- GERD : Gastroesophageal reflux disease
- GER : Gastroesophageal reflux
- IGERQ : Infant Gastroesophageal reflux questionnaier
- LES : Lower esophageal sphincter