

**PREVALENCE OF CHRONIC RHINOSINUSITIS IN CHILDREN WITH
DYSPEPSIA AT KENYATTA NATIONAL HOSPITAL AND GERTRUDE'S
CHILDREN'S HOSPITAL**

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This is a copy of my dissertation as partial fulfillment of the requirements by the University of Nairobi for the award of the degree of Masters in Medicine in ENT/ Head and Neck surgery.

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DECLARATION

I declare that this thesis is my original work and has not been presented for a degree award at any other university.

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CERTIFICATE OF APPROVAL

This research study was submitted to the Kenyatta National Hospital and University of Nairobi research and ethics committee for approval through the permission of the following supervisors;-

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LIST OF ABBREVIATIONS

CRS.....Chronic Rhinosinusitis

ENT-HN.....Ear, Nose, Throat, Head and Neck

KNH.....Kenyatta National Hospital

GCH.....Gertrude’s Children’s Hospital

GERD.....Gastroesophageal Reflux Disease

SPSSStatistical Package for the Social Sciences

H. Pylori.....Helicobacter Pylori

RSTF.....Rhinosinusitis Task Force

PIPrincipal Investigator

DEFINITION OF TERMS

Dyspepsia is defined as painful, difficult, or disturbed digestion, which may be accompanied by symptoms such as nausea and vomiting, heartburn, bloating, and stomach discomfort.

Chronic rhinosinusitis is inflammation of mucosa of paranasal sinuses and nasal cavity lasting more than 12weeks.

Helicobacter pylorus is a Gram-negative, microaerophilic bacterium that causes chronic inflammation of the gastrointestinal tract mucosa.

Gastroesophageal reflux disease is a chronic symptom of mucosal damage caused by gastric juice that refluxes into the esophagus exceeding the normal limit.

ABSTRACT

Background: Chronic rhinosinusitis (CRS) is a complex condition with multitude of risk factors and has been found to affect both adult and paediatric population. Dyspepsia (GERD and H.Pylori) maybe one of the factors associated with chronic rhinosinusitis.

Objectives: Study determined the prevalence of CRS in children diagnosed with dyspepsia. This was achieved by establishing the prevalence of H.pylori and GERD among children diagnosed with dyspepsia and the coexistence of CRS with dyspepsia.

Study methodology: This is hospital based cross sectional descriptive study that was carried out at Kenyatta National Hospital and Gertrude's Children's Hospital. 96 children diagnosed with dyspepsia were proportionately selected from the two hospitals using simple random sampling. Informed consent was obtained from the parent/guardian. CRS and dyspepsia were clinically diagnosed and Rome III criteria used for dyspepsia. H.pylori Antigen Stool Test done and findings recorded in a questionnaire. Data in filled questionnaires was entered, and analyzed using SPSS vs. 20. Baseline characteristics were compared Spearman Rho' Chi Square test was used to test associations. Logistic regression was used to analyze statistically significant data.

Results: Ninety six dyspeptic paediatric patients were analyzed for H. pylori Antigen stool test, Reflux symptom index and CRS. CRS prevalence was 41.7%, H. pylori prevalence was 60.4% and GERD was 78.5%. CRS with GERD had p value = 0.00001 (OR-38.07), CRS with H. Pylori had p value = 0.0252(OR-2.95) and CRS with GERD and H. Pylori had p value = 0.001(OR-20.05).

Conclusion: GERD is one of the CRS causative risk factor with a significant correlation and clinicians should have high index of suspicion.

CHAPTER ONE: INTRODUCTION

1.1 Background

Dyspepsia is a medical condition characterized by chronic or recurrent upper abdominal pain, bloating, belching, nausea, heartburn, vomiting, retrosternal pain, and/ or loss of appetite. [1, 3] It is a common problem, frequently caused by gastroesophageal reflux and helicobacter pylori affecting patients' quality of life. [2] The prevalence of dyspepsia is estimated at 10% to 45% in the general population with an annual incidence rate of 1% to 6%. [4, 5, 6] It is common in the paediatric population affecting 10% to 15% of children between the ages of 4 and 14 years. [7]

Chronic rhinosinusitis can be caused by GERD or Helicobacter Pylori infection manifesting within the sinonasal region among other causes. H.pylori is observed in 30% to 100% of children with endoscopic nodular gastritis. [9] Globally, Gastroesophageal reflux disease (GERD) impacts on health, impairing the quality of life in a substantial proportion of worldwide population. It is common in first year of life to teenage, suggesting that the disease process can begin at any stage. [11]

1.1.1 Chronic Rhinosinusitis

Chronic rhinosinusitis occurs frequently, with a significant impact on quality of life. The economic impact is negative due to its rising treatment costs. An estimation of 6 billion dollars is spent in the United States annually on rhinosinusitis treatment. [13] CRS affects 5 to 15% worldwide populations. [14] M. Desrosiers did a study in Canada that described the similarity of CRS impact on quality of life with other health conditions (like arthritis, cancer, asthma etc). [15]

1.1.2 Anatomy

Paranasal sinuses are air filled sacs found in the skull bone that surround the nasal cavity. They are divided in four pairs: Maxillary, Frontal, Ethmoidal, and Sphenoidal sinuses. They develop from ridges and furrows in the lateral nasal wall as early as 8th week of intrauterine life till early adulthood. [17]

The mucosa is attached directly to the bone and lined with ciliated, pseudostratified columnar epithelium, with goblet cells interspersed among the columnar cells in the nasal and paranasal regions. The paranasal sinuses are partially enclosed cavities opening to the lateral wall of the nasal cavity through meatus. Osteomeatal complex lies in the middle meatus, lateral to the middle turbinate, where the maxillary, frontal, and anterior ethmoid sinuses drain through their ostia. The posterior ethmoid opens into the superior meatus, while the sphenoid sinuses drain into the sphenoethmoid recess. Secretions are drained out via their natural ostia, through mucociliary clearance mechanism.

1.1.3 Pathophysiology of CRS

CRS is a risk factors associated with CRS are, allergy, asthma, immunodeficiency, GERD/H.Pylori, anatomic obstruction, genetics, congenital and environmental factors (irritants-cigarette smoke, microbial), medication inducing rhinitis medicamentosa, aspirin intolerance and diminishment of the ciliary function. [18]

CRS is characterized by osteal occlusion, with stagnation of secretions due to impaired mucociliary clearance. This can be triggered by mechanical obstruction at osteameatal complex and mucosal oedema. Inflammatory mediators like cytokines act as attractants for immune cells are released by local cells while histamine and other cytokines trigger dilation of local venules and capillaries, thus leukocytes enter tissue with ease. [13] This leads to oedema caused by the process of signaling, blocks osteal drainage, airflow becomes

inadequate, increasing intrasinus pressure thus blood flow to the tissues is significantly reduced. [19] The maxillary ostia must be open at least 5 mm, to maintain the balance of gas exchange. Pain is caused by more vascular constriction, increasing sinus pressure which stimulates C-fibre sensory receptors.

The impaired epithelial function is noted by significant reduction of ATP content and glucose concentration. The parasympathetic system, causes hypersecretion by goblet cells which increases Na/KATPase. This increases secretion viscosity due to changes in mucin composition which causes stagnation of fluid in extracellular space. [20, 21, 22] In the affected sinus, there is bacterial growth which leads to tissue inflammation thus development of infection. Mucosa then thickens leading to further blockage and the ostium is obstructed.

1.1.4 CRS relationship with GERD/H.Pylori

The symptoms of dyspepsia (GERD and H.pylori) have been associated with CRS. GERD is related to conditions like chronic coughing, dysphonia, dysphagia, globus sensation, laryngospasm, subglottic stenosis, benign and malignant lesions of the vocal cords. [23] There are three mechanisms associated with reflux in the sinonasal regions.

1. The refluxate flows into the sinonasal mucosa causing direct effect, which results in an inflammatory response. The mucosa develops edema impairing mucociliary clearance. This causes obstruction of the paranasal sinus ostia thus growth of microorganisms which leads to infection. Using 24-hour pH probe studies, Contencin et al demonstrated reflux to the nasopharynx. They placed pH probe in oesophagus and hypopharynx and the refluxate frequently extended superiorly above the cricopharyngeus level.[24] Phipps et al. showed a high prevalence of GERD in a normal healthy population where 63% of patients had CRS and 32% of these patients had nasopharyngeal reflux.[25]

2. The other theory involves autonomic nervous system. It is termed as vasomotor rhinosinusitis where there is imbalance of autonomic nervous system to the sinonasal mucosa. This revolves around a vagally mediated neurogenic mechanism, which results in overactive parasympathetic stimulation causing vasodilatation. This results to sinonasal edema, hypersecretion and secondary ostial obstruction. Patients with asthma and GERD were found to have exaggerated vagal responsiveness compared to normal patients. This study was demonstrated in the lower airway by Lodi et al. [26] and Harding et al. [27]. It was later demonstrated by Loehrl et al [28] in patients with chronic upper airway inflammation.

3. The last mechanism involves the role of H. pylori in CRS, which also plays a role in gastric ulcers and gastritis. [29, 30] It has also been demonstrated in oral cavity, oral lesions, and saliva. [31, 32, 33] Several articles have reported colonization by H.pylori in the sinonasal mucosa, inducing hypoxia and acidic environment in sinonasal region causing infiltration of inflammatory cells and release of chemical and inflammatory mediators. [18, 34, 35]

There are three hypotheses for presence of H. pylori in sinonasal mucosa. These are sinonasal mucosa is the primary source for H.pylori, Oro-nasal reflux transits H. pylori to the nose and middle ear and Gastro-esophageal acidic reflux transport H.pylori to sinonasal area. [35]

1.1.5 Diagnosis

Dyspepsia is a common symptom of gastrointestinal disease globally distributed. The Rome III committee for functional gastrointestinal disorders, defined dyspepsia as “it is a symptom considered to originate from the gastroduodenal area”. [36]

The Rome III committee proposed two diagnostic categories of dyspepsia. Symptoms were distinguished between meal-induced dyspepsia and meal-unrelated dyspepsia, forming its basis

as: postprandial distress syndrome (PDS) and epigastric pain syndrome (EPS). Below is the diagnostic criteria.

ROME III, DIAGNOSTIC CRITERIA OF DYSPEPSIA

Diagnostic criteria must include:

1. One or more of the following:
 - a. Bothersome postprandial fullness
 - b. Early satiation
 - c. Epigastric pain
 - d. Epigastric burning AND
2. No evidence of structural disease that is likely to explain the symptoms

(Criteria fulfilled for the last 3 months with symptom onset at least 6 months prior to diagnosis)

1.1.6 The Reflux Symptom Index

GERD can be diagnosed using various methods: clinical features, pH monitoring and endoscopically with imaging. Clinically, it can be diagnosed using Reflux symptom index where within the Past one Month, the condition is scored and rated. If score is more than 10, then the patient has GERD.

Ordinal Scale: 0- No problem 5- Severe problem Score: >10- patient has GERD

Symptoms	Score					
	0	1	2	3	4	5
Hoarseness or other voice problems	0	1	2	3	4	5
Clearing throat	0	1	2	3	4	5
Excess throat mucus or postnasal drip	0	1	2	3	4	5
Difficulty swallowing food, liquid, or pills	0	1	2	3	4	5
Coughing after eating or after lying down	0	1	2	3	4	5
Breathing difficulties or choking episodes	0	1	2	3	4	5
Troublesome or annoying cough	0	1	2	3	4	5
Sensations of something sticking in throat or lump in throat	0	1	2	3	4	5
Heartburn, chest pain, indigestion, or stomach acid coming up	0	1	2	3	4	5

1.1.7 Chronic rhinosinusitis

In 1996, the American Academy of Otolaryngology-Head & Neck Surgery multidisciplinary Rhinosinusitis Task Force (RTF) defined a diagnostic criteria for rhinosinusitis including Major and Minor symptoms. [3] In 2003, the RTF's definition was amended to require confirmatory radiographic or nasal endoscopic or physical examination findings in addition to suggestive history. [4, 5] There should be > 2 major symptoms (inclusive of nasal obstruction) and any minor symptoms.

Diagnosis of rhinosinusitis (RSTF- Benninger et al 2003): [37]

Major factors

Facial pain/pressure

Minor factors

Headache

Nasal obstruction/blockage	Fever (all nonacute rhinosinusitis)
Nasal discharge/purulence/discolored postnasal drainage	Halitosis
	Fatigue
Hyposmia/anosmia	Dental pain
Purulence in nasal cavity on examination	Cough
	Ear pain/pressure/fullness

Anterior Rhinoscopy - Discolored nasal drainage, polyposis or polypoid swelling or

Endoscopy: - Edema or erythema of the middle meatus or ethmoid bulla or

- Generalized erythema or edema (requires imaging).

European Position Paper on Rhinosinusitis and Nasal Polyps 2012 describes CRS as sinonasal inflammation characterized by two or more symptoms, one of which should be either nasal blockage/ obstruction/congestion or nasal discharge (anterior/posterior nasal drip): \pm facial pain/pressure, \pm cough; and either endoscopic signs of disease and/or relevant changes on the CT scan of the sinus [48].

1.2 Literature Review

Currently in Kenya, children have been diagnosed with dyspepsia. H.pylori in children was found to be at 73.3% in a study done by Kimang'a in 2010 [44]. This was not associated with CRS, so there is no data showing the prevalence of CRS in children with dyspepsia. In both KHN and GCH, the association of both CRS and dyspepsia is noted but there is no data to collate.

Phillips C.D. et al, 1998 conducted a descriptive study on the role of gastroesophageal reflux in children with chronic sinusitis and found out that 63% had esophageal reflux of which 32% had nasopharyngeal reflux. The patients with GERD were put on treatment and 79% improved. The conclusion was that GERD treatment actually managed sinus disease [25].

Ali Ozdek et al, 2003 concluded that H pylori is a risk factor of CRS and it can be detected in the sinonasal mucosa. He collected mucosal tissue samples from 25 patients. The case group comprised of 12 patients who had CRS while control group had 13 patients with concha bullosa. The samples were collected from ethmoid cells. H.pylori DNA was positive in 4 of 12 patients with CRS, 3 of these had GERD. Negative results were found in the control group. [38]

A prospective clinical trial study was done by Cvorovic L. et al in 2008, who confirmed that H.pylori was a risk factor for CRS [39]. In the study, H.pylori colonization in the nasal polyp specimens of patients with CRS was investigated. The cases included 23 adult patients with sinonasal polyposis and 15 controls with concha bullosa. The results indicated H.pylori was positive in 6 patients who had nasal polyposis while it was negative in the controls.

F.Khajen et al, did a prospective study in 2011, in Southern Iran. The results were positive in 57% of the cases and 10% of the control. The title was on prevalence of H.pylori in patients with nasal polyposis. It included 26 cases with nasal polyposis and 20 controls were scheduled for nasal septoplasty. [40]

A retrospective study on chronic rhinosinusitis and nasal polyposis was done in January 2007 to November 2008 by Marina Serrato et al. In this study, 30 patients were evaluated and scheduled for functional endoscopic sinus surgery. A questionnaire was used to evaluate both CRS and GERD symptoms. In 40% of the patients, GERD was confirmed to be associated with CRS. The patients were put on GERD treatment and 33% of these patients improved. [41]

In 2003, Morinaka S. et al did a study on 11 patients who had CRS and had undergone sinus surgery under local anaesthesia. H.pylori was only detected in 2 patients. [42]

CHAPTER TWO: RATIONALE AND OBJECTIVES

2.1 Justification

CRS is a common condition in the ENT department with multitude of risk factors. Dyspeptic symptoms are very common in general population, with prevalence estimates ranging between 10% and 45%. Kimanga et al, 2010, found out that H.pylori in dyspeptic patients was at 73.3% in children and 54.8% in adults in Kenya. GERD complicates treatment of CRS and the prevalence rates are important in CRS management. Further, study findings will provide baseline information for related studies.

2.2 Research questions:

What is the prevalence of chronic rhinosinusitis in children with dyspepsia at Kenyatta National Hospital and Gertrude's Children's Hospital?

2.3.1 Broad Objective

To determine the prevalence of chronic rhinosinusitis in children with dyspepsia at Kenyatta National Hospital and Gertrude's Children's Hospital.

2.3.2 Specific Objectives

1. To determine the prevalence of H.pylori among children diagnosed with dyspepsia at KNH and GCH.
2. To determine the prevalence of GERD in children with dyspepsia at KNH and GCH.
3. To determine the relationship between CRS, H.pylori and GERD in children who have dyspepsia at KNH and GCH.

CHAPTER THREE: METHODOLOGY

3.1 Study Design

The research design was hospital based descriptive cross-sectional study.

3.2 Study Area

This study was carried out at Kenyatta National Hospital (KNH) and Gertrude's Children's Hospital (GCH).

3.3 Study Population

Study targeted children between the ages of 5 years to 17 years with dyspeptic symptoms visiting gastroenterology clinic at the Pediatric Department, Kenyatta National Hospital and Gertrude's Children's Hospital.

3.4 Inclusion and Exclusion Criteria

This study only included paediatric population of children aged between 5yrs to 17yrs diagnosed with dyspepsia using Rome III criteria and whose Parents/Guardians consented to participate.

3.5 Limitations

The pH monitoring method for diagnosing GERD is not possible due to equipments unavailability in Kenya. Rigid nasal endoscopy is an invasive procedure and expensive to the patient thus only clinical criteria was used to diagnose CRS.

3.6 Study Period:

The study period was 6 months from date of approval.

3.7 Sample Size Determination

$$n = \frac{NZ^2P(1 - P)}{d^2(N - 1) + Z^2P(1 - P)}$$

Where n = Sample size with finite population correction (Fisher's formula). [45]

N = Population size

Clinic attendance: KNH – 8-12 per week; GCH 4-8 per week week

Total 12-20 per week, and an average of 16 per week

Total 16 per week × 8 weeks for data collection = 128

Therefore N = 128

Z = Z statistic for 95% level of confidence = 1.96

P = Expected prevalence of chronic rhinosinusitis = 0.5

d = desired precision = 0.05

$$n = 128 \times 1.96^2 \times 0.5(1-0.5) \div 0.05^2(128-1) + 1.96^2 \times 0.5(1-0.5) = 96.198$$

$$n = 96$$

3.8 Sampling Method

The sampling frame was children who fit the inclusion and exclusion criteria attending Gastroenterology clinic at Paediatric department at Kenyatta National Hospital and Gertrude's Children's Hospital. Sample selection was proportionate by systematic sampling. Subjects were recruited into the study as they came to the clinic until the required number was obtained.

3.9 Data collection tool

Data was collected using a questionnaire, which included bio-data and anterior rhinoscopic examination findings.

3.10 Data collection procedure

Paediatric patients diagnosed with dyspepsia who met the inclusion criteria were recruited into the study, procedure explained and consent sought from parent/ guardian. A study number was assigned to each patient who consented and biodata recorded. A routine H.pylori stool test was done on each patient who gave consent. 1-2 g or 1-2 ml stool specimen was collected in a properly labeled sterile leak-proof container using sterile gloves. The specimen was then transported to the laboratory accompanied by a laboratory request form for H. pylori Stool Antigen (rapid chromatographic immunoassay). The assay is a qualitative, lateral flow immunoassay for H. pylori detection. In this test, the membrane was pre-coated with anti-H.pylori antibodies on the test line region of the test. During testing, the diluted specimen reacted with the particle coated with anti-H. Pylori antibodies. The mixture migrates upward on the membrane by capillary action to react with H.pylori antibodies on the membrane and generate a colored line which indicates a positive result, while its absence indicates a negative result. The control line indicates the volume of specimen and occurrence of membrane wicking. Results are read at 10 minutes after dispensing the specimen. Results are positive when two distinct colored lines appear, negative when one colored line appears in the control line and invalid when control line fails to appear. The researcher provided the stool test kits in the laboratory at each hospital to avoid extra costs to the patient. Anterior rhinoscopic examination was conducted by researcher to reveal the nasal mucosa status, assess presence of inflammation, mucus and any nasal obstruction. All relevant findings were recorded in the questionnaire for data analysis. Children diagnosed with CRS were referred to the ENT specialist clinic.

3.11 Quality control

Only the primary investigator screened the patients and collected the history and clinical examination data to prevent inter personal bias. Each test kit had a batch number and a serial number. Any test kits whose expiry date lapsed was discarded.

All specimens were processed in the same laboratory in each hospital and standard operating procedures for specimen handling, processing and analysis was followed to ensure standardization. Quality control was supervised by a laboratory specialist. An internal procedural control was included in the test. The two laboratories have control standards where positive and negative controls have been tested to confirm the test procedure and verify proper test performance. Every 20th patient was tested again in another laboratory for reproducibility.

Batch number was recorded in the laboratory request form and the proforma to ensure internal validity. The patient proforma was pretested before commencement of data collection and appropriate modifications made. The patient's history and physical examination was conducted by the principal investigator who entered the findings in the questionnaire.

3.12 Data management

At the end of each interview the filled questionnaire was cross checked for completeness and any missing entries corrected. The quantitative data collected was coded, processed and cleaned prevent inconsistencies and outliers. The qualitative data was analyzed through the selection of concepts, categories and themes. It involved reading through the data and developing codes that drew similar connections between categories and themes. Data was analyzed by the use of SPSS (Statistical Package for the Social Sciences) version 20 as per the specific research questions using frequencies and percentages. Relationship between the independent variables and the dependent variable was established using Chi-square test of association and Logistic linear regression.

3.13 Ethical considerations

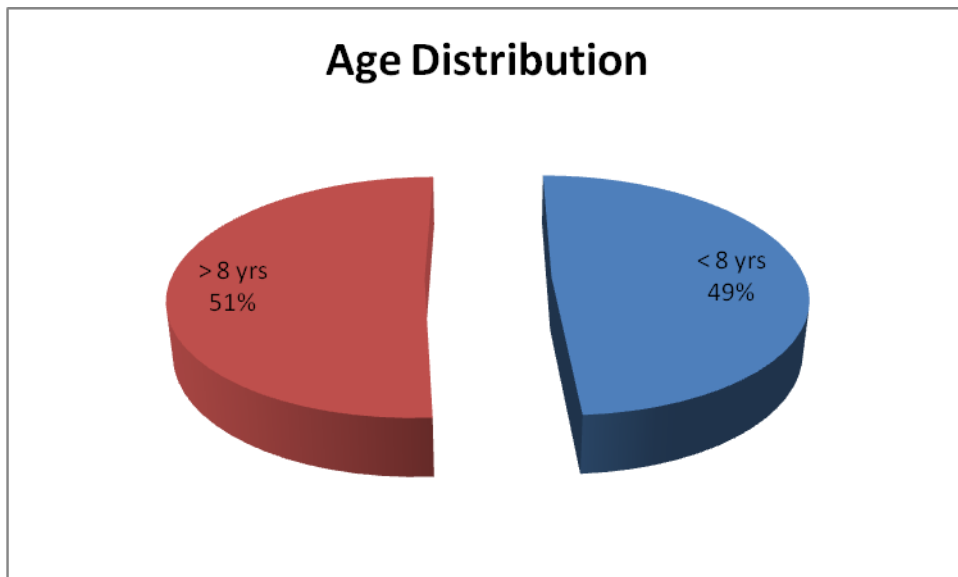
The research was approved by both the KNH/ UON ethics committee and the management of the selected institutions. Permission to conduct research was sought from the relevant authorities at respective hospitals. Respondents gave consent on behalf of their children to participate. They were informed that it was voluntary and that they had the right to decline participation or withdraw at any time (Appendix I. The researcher gave full information about what the research entailed and ensured participants were competent to give consent. Full consent and explanation is in Appendix I. The questionnaires were administered after duly obtaining the consent of the participant's parent/guardian. Participants' privacy was highly maintained by ensuring that they were not exposed to public when filling questionnaires. The researcher ensured the anonymity of respondents by concealing their identity and kept research data confidential for research purposes only. Any concerns arising were noted and resolved immediately.

CHAPTER FOUR: FINDINGS

4.1: BIO-DATA

In this study, a sample of 64 children from KNH and 32 children from GCH was used and it comprised of 61(63.6) males and 35(36.5%) females. The mean age of children from KNH was 9.0(\pm 2.9) years with a range of 5 to 17years and the mean age at GCH was also 9.0(\pm 3.2) years with a similar range of 5 to 17 years. The mode was 7years while median was 9years. The children below the age of 8 years (5-8 yrs) were 49% and the children above 8 years (9-17yrs) were 49%.

Figure 1: Pie Chart Illustrating Age Distribution



The mean weight of children from both hospitals was within normal range. The distributions of gender, age and weight were the same across KNH and GCH (Mann-Whitney U test p-values>0.05).

Figure 2: Graph Illustrating Gender in KNH and GCH

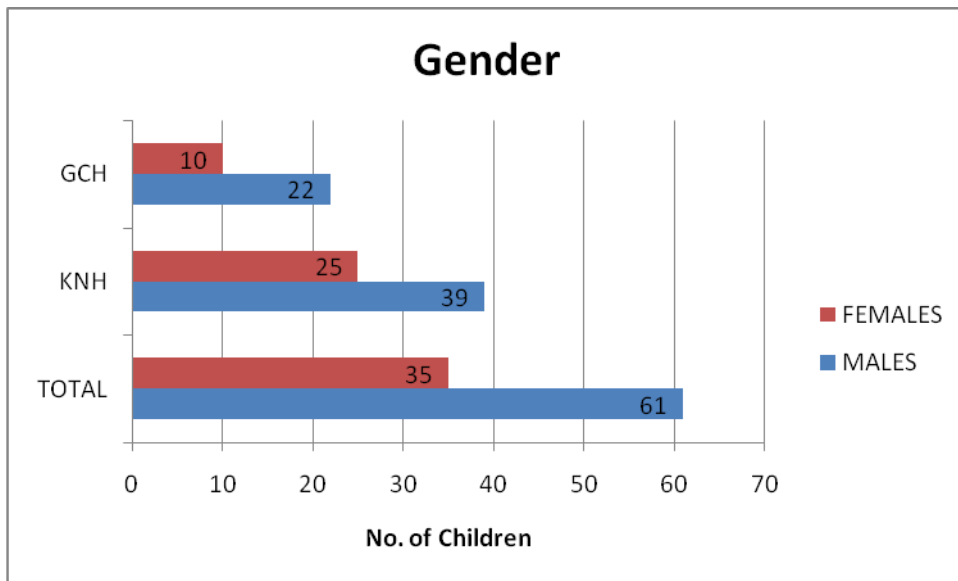


Table 1: Prevalence of CRS, H. PYLORI and GERD

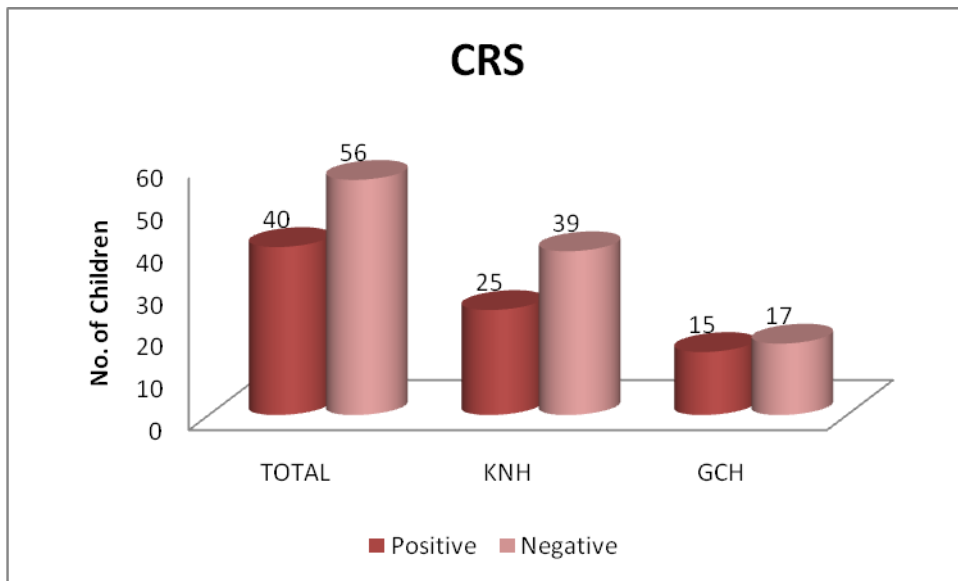
	GERD		H. Pylori		CRS	
	Positive	Negative	Positive	Negative	Positive	Negative
Total	75(78.1%)	21	58(60.4%)	38	40(41.7%)	56
KNH	53(82.8%)	11	38(59.4%)	26	25(39.1%)	39
GCH	22(68.8%)	10	20(62.5%)	12	15(46.9%)	17

The prevalence of the three groups above had different figures as demonstrated in the in the table.

4.2: CRS

The overall prevalence of CRS in children with clinically proven dyspepsia was 41.7%. The prevalence at KNH was 39.1% and at GCH was 46.9%. The distributions of CRS prevalence was the same across KNH and GCH (Mann-Whitney U test) p-values=0.461 which was clinically not significant.

Figure 3: Graph Illustrating Prevalence of CRS in KHN and GCH

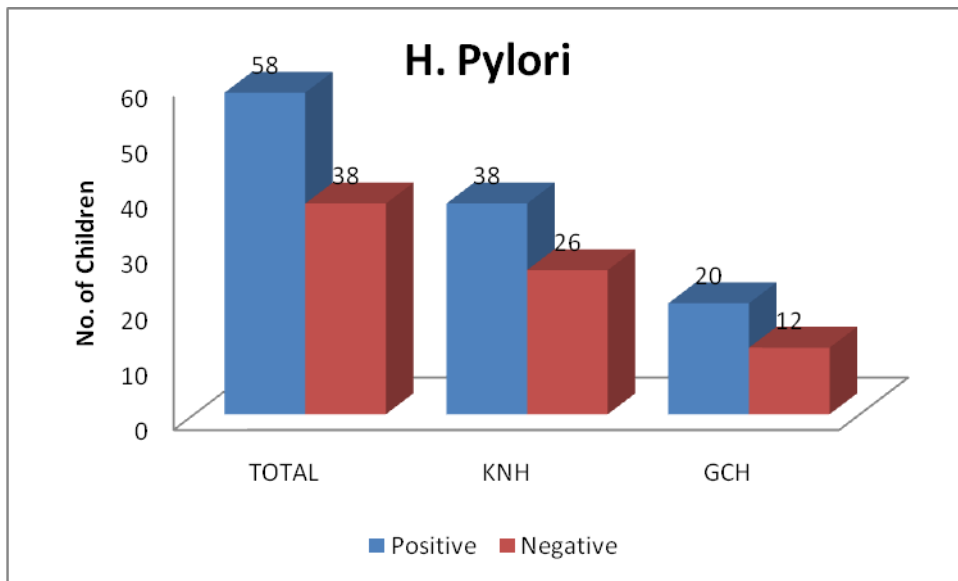


Regression analysis indicated that the age, weight and gender of children did not have any significant (p -values >0.05) effect on the prevalence of CRS.

4.3: H.PYLORI

The overall prevalence of H. pylori infection in children clinically diagnosed with dyspepsia was 60.4%. The prevalence in KHN was 59.4% and in GCH it was 62.5%. The distributions of H. pylori prevalence was the same across KHN and GCH (Mann-Whitney U test p -values=0.560). Regression analysis indicated that the age, weight and gender of children did not have any clinical significant (p -values >0.05) effect on the prevalence of H. pylori.

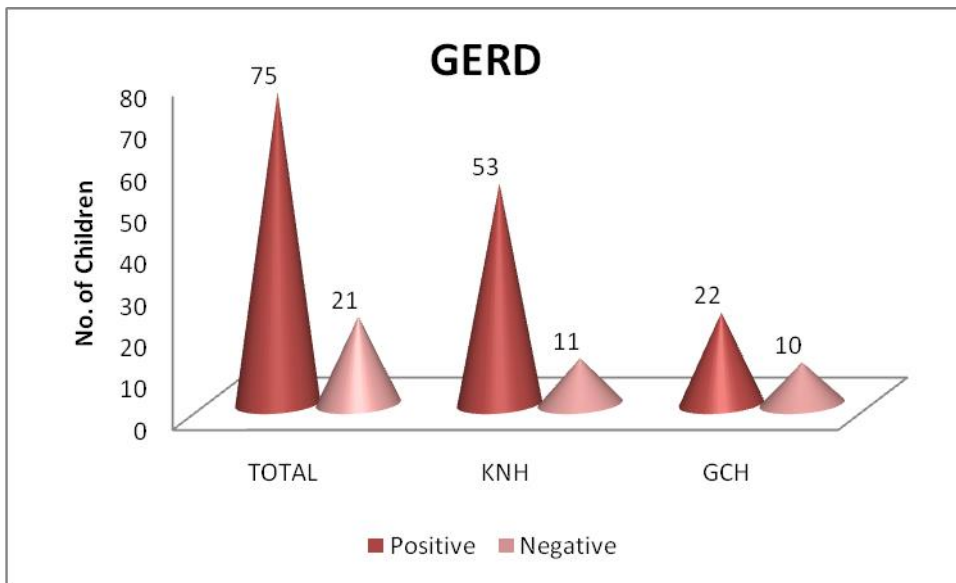
Figure 4: Graph Illustrating Prevalence of H. PYLORI in KNH and GCH



4.4: GERD

The overall prevalence of GERD among children diagnosed with dyspepsia was 78.1%. The prevalence at KNH was 82.8% and GCH was 68.8%. The distributions of GERD prevalence was the same across KNH and GCH (Mann-Whitney U test p-values=0.765). Regression analysis indicated that the weight and gender of children did not have any significant (p-values>0.05) effect on the prevalence of GERD. However, age was clinically significant (Wald Chi-square test p-value=0.023) on the prevalence of GERD which implied that majority of children aged above 10 years tested negative as compared to those aged below 10 years.

Figure 5: Graph Illustrating Prevalence of GERD in KNH and GCH



4.5: Relationship of H.PYLORI and GERD with CRS

TABLE 2: Association of CRS with GERD and H. PYLORI

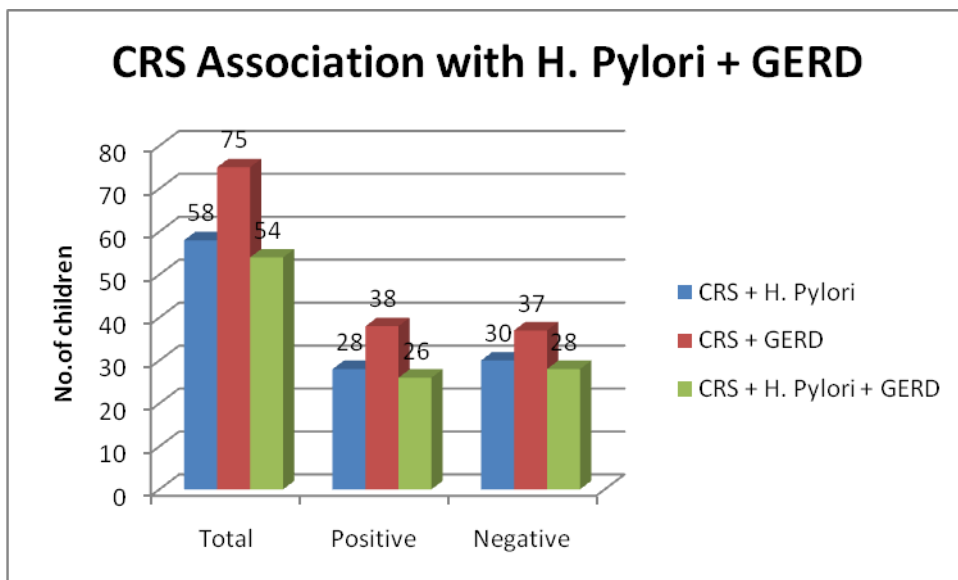
Risk Factors	CRS		TOTAL		p- value	OR
	Positive	Negative	Positive	Negative		
H.Pylori	28(48.3%)	30(51.7%)	58(60.4%)	38(39.6%)	0.0252	2.95
GERD	38(50.7%)	37(49.3%)	75(78.1%)	21(21.9%)	<0.00001	38.07
H. Pylori + GERD	26(48.1%)	28(51.9%)	54(56.3%)	42(43.7%)	<0.001	20.05

The distribution of H. pylori and GERD in association with CRS was different (Friedman’s Analysis of Variance p-value=0.000). Children clinically proven to have dyspepsia with CRS with GERD were 38(50.7%) with p-value = 0.00001. This was very significant and the OR=

38.07 with 95% CI 4.9-7.97. The children with CRS and H.pylori were 28(48.3%) with p-value = 0.0252 which is clinically significant with OR = 2.95. Children who had CRS in association with H. Pylori and GERD were 26 (48.1%) with p value = 0.001, OR = 20.5, 95% CI 2.6- 4.31 and it was clinically significant. One child had CRS with negative H. pylori and GERD (p value = 0.856).

Logistic regression indicated that GERD (p-value = 0.00001) had clinical significant effect on CRS as compared to H. pyroli (0.0252). There was significant correlation (Spearman’s rho p-value=0.00001) between GERD and CRS.

Figure 6: Graph Showing Association between CRS, H. PYLORI and GERD



4.6: Association between AGE and CRS

Table 3: Association between AGE and CRS

Age	Dyspepsia	CRS	p- value	OR
5-8yrs	47	23	0.1587	1.8039
9-17yrs	49	17	0.2142	0.789

CRS was higher in the age group below 8 years at 24% and it was less in the children above 8 years at 17.7%. The p value was 0.1587 with OR = 1.8039 (below 8yrs) and 0.2142 with OR = 0.789 (above 8yrs) and this was not clinically significant. The odds ratio demonstrated that the dyspeptic children below 8yrs were 1.8 times more likely to develop CRS.

CHAPTER FIVE: DISCUSSION, CONCLUSION AND RECOMMENDATION

5.1 Discussion

CRS has been associated with various risk factors and GERD/ H. pylori are one of the causative factors which have been demonstrated. H.pylori has been found to colonize several otolaryngology regions such as the nasal cavity, paranasal sinuses, middle ear, oral cavity, oropharynx, and larynx. GERD is a known risk factor of CRS affecting patients' quality of life [2]. The patients included in this study had clinically proven dyspepsia and a questionnaire was used to diagnose CRS symptoms and the CRS children treated by an otolaryngologist thereafter.

In this study, 96 patients were sampled included 63.6% males and 36.5% were females which was not significant since they were randomly selected. The mean age was 9.0(\pm 2.9) years with the majority of children below 10 years ranging from 5 to 17 years. The mode was 7years and median was 9years. Phillip C.D et al carried out a study involving 30 patients whose ages ranged from 2 years to 18 years GERD [25]. KNH and GCH represent different socio-economical status but the logistic regression was not significant. The statistical value used was p- value = 0.05 which demonstrated the significance of the results in the current study.

The prevalence of CRS was 41.7%%. Age and gender did not have significant effect on the CRS as indicated by regression analysis. CRS prevalence in the paediatric population was found to be 18-45% on radiography [48]. Phillip C.D. et al did a study in 1998 on paediatric patients with CRS and 63% of these children had GERD, while Marina Serrato et al, 2008 did a study involving paediatric and adult age groups which concluded that 40% of the patients had GERD [25, 41].

The prevalence of *H. pylori* in the children included in this study was 60.4%. Regression analysis indicated that the age and gender did not have any significant effect on *H. pylori* (p -values=0.560). The *H. pylori* stool antigen test (rapid chromatographic immunoassay) was utilized done in all the 96 dyspeptic patients. This One Step Antigen *H. pylori* test has been shown to have sensitivity of >94.4% and specificity of >99.9% with relative accuracy of >97.9% and CI of 95% compared with endoscope- based methods [46].

In this study, the GERD prevalence was 78.5%, and age had significant effect on GERD, where majority of children were below 10years and had a Reflux Symptom Index score of over 10 (Appendix III). Marina Serrato et al. (2008) found 40% CRS patients had GERD [41]. Phillip C.D. et al (1998) found that 63% of children with CRS had GERD [25].

The association between *H. pylori* and GERD as risk factors for CRS was investigated. Prevalence of CRS and *H. pylori* was highest among the children at GCH while GERD was highest among children at KNH. Children clinically proven to have dyspepsia with CRS with GERD were 38(50.7%), p -value= 0.00001 with OR=38.07, 95% CI. The children with CRS and *H. pylori* were 28(48.3%) with p - value = 0.0252 with OR = 2.95. The CRS with GERD and CRS with *H. pylori* had p value of < 0.05 and they were clinically significant.

Children who had CRS in association with *H. Pylori* and GERD were 26(48.1%) had p - value = 0.001, OR = 20.5 in the current study. There was only one patient who had CRS without *H. pylori* or GERD and it was clinically not significant p value = 0.856. Age below 8 years in association with CRS was at 24%, p - value = 0.1587, OR = 1.8039 while the children above 8 years was 17.7%, p -value = 0.2142 and it was not statistically significant.

Logistic regression indicated that GERD (p -value=0.00001) had significant effect on CRS as well as *H. pylori* (p value=0.0252). This implied that GERD increased the odds of contracting

CRS by 38.07 times compared to H. pylori with OR = 2.95. Children with dyspepsia below age 8 years had odds ratio of 1.8039.

5.2 Conclusion

In line with this study, CRS has been associated with GERD as well as H. pylori. GERD with CRS had p- value of 0.00001 which is very significant. Children with GERD are 38 times more likely to develop CRS and the children with both GERD and H.Pylori are 20 times more likely to develop CRS.

5.3 Recommendations

Further studies on pH monitoring of the sinonasal, nasopharyngeal and hypopharyngeal regions in CRS patients suspected to have GERD should be carried out. Studies on severity of CRS in association with GERD are recommended to enable the clinician select the best management for these patients. Paeditricians should have a high index of suspicion for CRS in children diagnosed with GERD.

5.4 Limitation

This was a hospital based study and is therefore not representative of the Kenyan population as the patients who participated were attending KNH and GCH.

REFERENCES

1. Talley NJ, Vakil N. Guidelines for the management of dyspepsia. *Am. J. Gastroenterol.* 2005(10): 2324–37.
2. Zajac, P; Holbrook, A; Super, ME; Vogt, M. An overview: Current clinical guidelines for the evaluation, diagnosis, treatment, and management of dyspepsia. *Osteopathic Family Physician* 2013, 5 (2): 79–85.
3. Feinle-Bisset C, Vozzo R, Horowitz M, et al. Diet, food intake, and disturbed physiology in the pathogenesis of symptoms in functional dyspepsia. *Am J Gastroenterol.* 2004; 99:170-181.
4. Piessevaux H, De Winter B, Louis E, et al. Dyspeptic symptoms in the general population: a factor and cluster analysis of symptom groupings. *Neurogastroenterol Motil.* 2009; 21:378-388.
5. El-Serag HB, Talley NJ. Systematic review: the prevalence and clinical course of functional dyspepsia. *Aliment Pharmacol Ther.* 2004;19:643-654
6. Camilleri M, Dubois D, Coulie B, et al. Prevalence and socioeconomic impact of upper gastrointestinal disorders in the United States: results of the US Upper Gastrointestinal Study. *Clin Gastroenterol Hepatol.* 2005; 3:543-552
7. Apley J, Naish N. Recurrent abdominal pain: a field survey of school children. *Arch Dis Child* 1958; 33:169-170.
8. S. Amini-Ranjbar, N. Nakhaee, Diagnostic Utility of Nodular Gastritis in Children with Chronic Abdominal Pain Undergoing Endoscopy. *American Journal of Agricultural & Biological Science.* 2008, 3(2):494-496.
9. Macarthur C, Saunders N, Feldman W. *Helicobacter pylori*, gastroduodenal disease, and recurrent abdominal pain in children. *JAMA.* 1995; 273:729–734.

10. M. Ruge, P. Correa and M. Dixon et al. Gastric mucosal atrophy: interobserver consistency using new criteria for classification and grading. *Aliment Pharmacol Ther.* 2002 Jul; 16(7):1249-59.
11. Vakil N. Disease definition, clinical manifestations, epidemiology and natural history of GERD. *Best Pract Res Clin Gastroenterol.* 2010; 24:759–764.
12. Ryan AM, Duong M, Healy L, et al. Obesity, metabolic syndrome and esophageal adenocarcinoma: epidemiology, etiology and new targets. *Cancer Epidemiol.* 2011; 35:309–319.
13. S. Helms and A. L. Miller. Natural Treatment of Chronic Rhinosinusitis. *Altern Med Rev.* 2006 Sep; 11(3):196-207.
14. Wong IW, Omari TI, Myers JC, et al. Nasopharyngeal pH monitoring in chronic sinusitis patients using a novel four channel probe. *Laryngoscope.* 2004, 114 (9):1582-5.
15. M. Desrosiers, G. A. Evans, P. K. Keith, et al. Canadian clinical practice guidelines for acute and chronic rhinosinusitis. *Allergy Asthma Clin Immunol.* 2011 Feb 7(1):2.
16. M. Rubin, R. Gonzales, M. Sande. Infections of the upper respiratory tract. *Harrison's Principles of Internal Medicine.* 16th Ed. New York, NY, McGraw-Hill, 2005.
17. Bolger WE, Anatomy of the paranasal sinuses. 2001
18. Kim HY, Dhong HJ, Chung SK, Chung KW, Chung YJ, Jang KT. Intranasal *Helicobacter pylori* colonization does not correlate with the severity of chronic rhinosinusitis. *Otolaryngol Head Neck Surg.* 2007, 136(3):390-5.
19. Shin SH and Heo WW. Effects of unilateral naris closure on the nasal and maxillary sinus mucosa in rabbit. *Auris Narus Larynx,* 2005 Jun, 32(2): 139-143.
20. M. Ali, D. Hutton, J. Wilson, J. Pearson. Major secretory mucin expression in chronic sinusitis. *Otolaryngol Head Neck Surg.* 2005 Sep; 133(3):423-8.

21. Kim DH, Chu HS, Lee JY, Hwang SJ, Lee SH, Lee HM. Up-regulation of MUC5AC and MUC5B mucin genes in chronic rhinosinusitis. *Arch Otolaryngol Head Neck Surg.* 2004 Jun; 130(6):747-52.
22. R. Pawankar. Mast cells in allergic airway disease and chronic rhinosinusitis. *Chem Immunol Allergy.* 2005; 87:111-29.
23. DelGaudio JM. Direct nasopharyngeal reflux of gastric acid is a contributing factor in refractory chronic rhinosinusitis. *Laryngoscope.* 2005, 115(6):946-57.
24. Contencin P, Narcy P: Nasopharyngeal pH monitoring in infants and children with chronic rhinopharyngitis. *Int J Pediatr Otorhinolaryngol* 1991, 22:249–256.
25. Phipps CD, Wood WE, Gibson WS. Gastroesophageal reflux contributing to chronic sinus disease in children. A prospective analysis. *Arch Otolaryngol Head Neck Surg* 1999, 121:255–262.
26. Lodi U, Harding SM, Coghlan HC, Guzzo MR, Walker LH. Autonomic regulation in asthmatics with gastroesophageal reflux. *Chest* 1997, 111:65–70.
27. Harding SM, Guzzo MR, Maples RV, et al. Gastroesophageal reflux induced bronchoconstriction; vagolytic doses of atropine diminish airway responses to esophageal acid infusion. *Am J Respir Crit Care Med* 1995, 151:A589.
28. Loehrl TA, Smith TL, Darling RJ, et al. Autonomic dysfunction, vasomotor rhinitis, and extraesophageal manifestations of gastroesophageal reflux. *Otolaryngol Head Neck Surg* 2002, 126:382–387.
29. Alper J: Ulcers as an infectious disease. *Science* 1993, 260:159–160.
30. Lee A, Fox J, Hazell S. Pathogenicity of *Helicobacter pylori*: a perspective. *Infect Immunol* 1993, 61:1601–1610.
31. Cammarota G, Tursi A, Montalto M, et al. Role of dental plaque in the transmission of *Helicobacter pylori* infection. *J Clin Gastroenterol* 1996, 22:174–177.

32. Mravak-Stipetic M, Gall-Trosel K, Lukac J, et al. Detection of *Helicobacter pylori* in various oral lesions by nested polymerase chain reaction (PCR). *J Oral Pathol Med* 1998, 27:1–3.
33. Li C, Musich PR, Has T, et al. High prevalence of *Helicobacter pylori* in saliva demonstrated by a novel PCR assay. *J Clin Pathol* 1995, 48:662–666.
34. Nurgalieva ZZ, Graham DY, Dahlstrom KR, Wei Q, Sturgis EM. A pilot study of *Helicobacter pylori* infection and risk of laryngopharyngeal cancer. *Head Neck*.2005; 27(1):22–27.
35. Dinis BP, Subtil J. *Helicobacter pylori* and laryngopharyngeal reflux in chronic rhinosinusitis. *Otolaryngol Head Neck Surg*. 2006; 134(1):67–72.
36. D. A. Drossman. The functional gastrointestinal disorders and the Rome III process. *Gastroenterology*, 2006 April, 130(5):1377–1390.
37. Benninger MS, Ferguson BJ, Hadley JA, et al. Adult chronic rhinosinusitis: definitions, diagnosis, epidemiology, and pathophysiology. *Otolaryngol Head Neck Surg*. 2003; 129(3 Suppl):S1-32
38. Ozdek A, Cirak MY, Samim E, et al. A possible role of *Helicobacter pylori* in chronic rhinosinusitis: a preliminary report. *Laryngoscope* 2003, 113(4):679–682.
39. Cvorovic L, Brajovic D, Strbac M, Milutinovic Z, Cvorovic V. Detection of *Helicobacter pylori* in nasal polyps: preliminary report. *J Otolaryngol Head Neck Surg*.; 2008, 37(2):192-5.
40. F.Khajen, Motazedain MH, Safarpour Z, Meshkibaf MH, Miladpoor B. Prevalence of *H.pylori* in patients with nasal polyposis. *Iran Red Crescent Med J*. 2011 Jun 13(6): 436–437.
43. Marina Serrato, Milton Rogério, Evaldo Dacheux, Edgar Sirena, Paulo Romam, Marcela Schmidt. Incidence of Gastroesophageal Reflux symptoms in

- patients with refractory chronic sinusitis upon clinical treatment. *Arch. Otorhinolaryngol.*, São Paulo, 2009,13(3):300-303.
44. S. Morinaka, M. Ichimiya, H. Nakamura, Detection of *Helicobacter pylori* in nasal and maxillary sinus specimens from patients with chronic sinusitis. *Laryngoscope*. 2003, 113(9):1557-63.
45. Shmueli H, Obure S, Passaro DJ, et al. Dyspepsia Symptoms and *Helicobacter pylori* Infection. *Emerg Infect Dis*. 2003 Sept; 9(9): 1103–1107.
46. Kimang'a A.N, Revathi G, Kariuki S, Sayed S, Devani S. *Helicobacter Pylori*: Prevalence and antibiotics susceptibility among Kenyans. *S Afri Med J*, 2010, 100(1), 53-57.
47. Daniel W. *Biostatistics: a foundation for analysis in the health sciences*. 7th Ed. New York, NY: Wiley, 1999; 180(5):268–70.
48. Altman E, Fernandez H. et al Analysis of *Helicobacter pylori* isolates from Chile: Occurrence of selective type I Lewis b antigen expression in lipopolysaccharide. 2008, 57(pt 5): 585
49. Cellini L, Allocati N, Dainelli B. Failure to detect *Helicobacter pylori* in nasal mucus in *H. pylori* positive dyspeptic patients. *J Clin Pathol* 1995; 48:1072–1073.
50. Fokkens WJ, Lund VJ, Mullol J, Bachert C, Alobid I, Baroody F, Cohen N, Cervin A, Douglas R, Gevaert P, et al. European Position Paper on Rhinosinusitis and Nasal Polyps 2012. *Rhinol Suppl*. 2012; (23):3 p preceding table of contents, 1–298.

TIME PLAN

<ul style="list-style-type: none">• PERIOD	<ul style="list-style-type: none">• ACTIVITY
<ul style="list-style-type: none">• July 2013-Dec 2013	<ul style="list-style-type: none">• Proposal writing
<ul style="list-style-type: none">• Jan 2014	<ul style="list-style-type: none">• Proposal Presentation
<ul style="list-style-type: none">• Feb- June 2014	<ul style="list-style-type: none">• Ethical approval
<ul style="list-style-type: none">• June- July 2014	<ul style="list-style-type: none">• Data collection.
<ul style="list-style-type: none">• August 2014	<ul style="list-style-type: none">• Data analysis
<ul style="list-style-type: none">• September 2014	<ul style="list-style-type: none">• Report writing & submission

BUDGET

CONSIDERATION	UNIT	QUANTITY	UNIT COST (Ksh)	TOTAL COST (Ksh)
Biostatician				25,000/=
Stool antigen for H.pylori		96	2500	240,000/=
Printing paper		20	500	10,000/=
Stationery				11,000/=
Contingency(Other)				28,600/=
Total				314,600/=

APPENDIX IA: GENERAL PATIENT INFORMATION AND CONSENT FORM

Title: The Prevalence of Chronic Rhinosinusitis in children with dyspepsia at Kenyatta National Hospital and Gertrude's Children Hospital

Investigator: Dr MWANGI Beatrice, Resident ENT Head and Neck Surgery, University of Nairobi. Contact: 0720615675; email: bmwangi09@gmail.com ; P.O.Box:19749-00202.

The Supervisors: DR.JOHN AYUGI– 0722883041; DR.LAVING AHMED – 0724644122

KNH/ UON-ERC: Prof. CHINDIA Email: uonknh_erc@uonbi.ac.ke

Investigator's statement

My name is DR.BEATRICE MWANGI, ENT, HEAD and NECK Surgery Resident pursuing a Masters Degree in the University of Nairobi. I am carrying out a study to find out the number of children with nasal infection among children complaining of stomach ache. This research is part of the requirements for the award of the mentioned degree. You have been selected as a participant and kindly requested to fill a questionnaire. All information you provide will be for purposes of research only. Participating in this study will not expose you to any physical or psychological harm whatsoever. Throughout this study, your identity will be concealed, and any information you provide will be confidential, and your privacy will be respected. Your participation is purely voluntary and you are free to withdraw at any point without any penalty. There is no compensation involved and the study is free of charge.

Background information

Dyspepsia is a medical condition characterized by chronic or recurrent upper abdominal pain, bloating, belching, nausea, heartburn, vomiting, retrosternal pain, and/ or loss of appetite. Very few studies have been conducted worldwide on this topic; hence the need to know what is happening in our country. Findings will help understand the relationship between stomach ache and nasal infection. This information will be beneficial to the health professions in the two hospitals and general health sector in Kenya as will provide grounds for medical interventions to mitigate risk factors.

What is the relationship between nose and stomach ache?

Your child has a stomach ache because of high acidity in the stomach which is mostly caused by an organism in the stomach called bacteria, (H.pylori). The nose gets affected when the stomach acid flows backwards to the throat involving the nose especially when the patient lies flat.

What is involved in this study?

Once you consent to participate, I will take medical history of the condition (tests done and medicine given), and examine your child by looking into his/her nose with a torch. Stool specimen from your child will be obtained and sent to the hospital laboratory. This information will be filled in a questionnaire similar among all participants.

Will I or my child be penalized for not participating or withdrawal?

No, you or your child will receive the same attention and treatment as those who choose to participate and you are free to withdraw at any point.

Are there any risks involved?

There are no risks involved but you or your child might experience some discomfort when taking stool specimen. However, this will last only a short time.

What benefits will I or my child get if we participate?

Information regarding the nasal problem is essential in selection of the most appropriate treatment early and mitigation of risk factors associated.

What about confidentiality?

All the information we obtain will be kept confidential

How much will it cost me?

No extra cost will be incurred

What are my rights as a participant?

Participation in the study is voluntary. Once inducted in the study, you can choose to discontinue at any time.

What do you do with the information you get?

This information will help the clinicians understand the association of stomach ache and nasal infection better. Like any other scientific information, we will seek to share our findings with other clinicians and the rest of the world.

In case you need any more information kindly enquire at any time.

Please fill in and sign the consent below to let your child participate if aged below 8 years. If your child is between ages 8years and 17years, he or she will sign assent form.

CONSENT FORM

I voluntarily agree to enroll to participate in the research. I confirm that I have understood the nature of the study and that whatever information I give will remain confidential.

Signed..... Relationship..... Date:

ASSENT FORM

I voluntarily agree to participate in the research having had the information read to me and understood the nature of the study and that whatever information I give will remain confidential.

Sign your name here (Minor)..... Date

Principal researcher: Dr. Beatrice Mwangi

Signature Date.....

APPENDIX IB: MAELEZEO YA UTAFITI NA KIBALI

Maelezo kuu:

Mimi ni Daktari ninaye endelea na masomo ya juu kwa utengo wa ENT-HNS; yaani upasuaji wa kitengo cha masikio, mapua, koo, kichwa na shingo katika Chuo kikuu cha Nairobi. Ningependa kuomba idhini yako ya kushiriki katika utafiti wenye unalenga madhara yanayo sababisha maabukizi ya pua kwa sababu ya ugonjwa wa kuumwa na tumbo. Kushiriki kwa utafiti huu ni kwa hiari yako.

Niko hatarini kushiriki?

Hapana humo hatarini.

Je, nita adhibiwa kwa kukosa kushiriki?

Hapana, hautabaguliwa kwa matibabu na yataendelea kama ilivyo pangwa.

Nitaridhishwa aje na utafiti huu?

Utafiti huu utasaidia wauguzi kwa kuwapa mawaita na matibabu ya pua kwa wagonjwa walioadhiriwa ili kupokea matibabu mapema.

Na kuhusu recordi zangu kuwekwa siri?

Recordi zako za ugonjwa hazitatolewa hadharani kwa yeyote.

Itanigharimu pesa ngapi?

Hautahitaji kutumia pesa zozote za ziada.

Na je nikitaka kujiondoa?

Ukitaka kujiondoa waweza kufanya wakati wowote. Tendo hilo halita fanya ubaguliwe kwa aina yeyote.

Umereithika na maelezo umepata?

Kama umerithika na unataka kusiriki katika utafiti huu, utapiga sahihi kwa form iliyo hapo chini.

KIBALI CHA UTAFITI

Kushiriki kwa mtoto wako katika utafiti huu ni kwa hiari yako.

Watoto chini ya miaka 18/wasiojifahamu:

Kibali cha mzazi

Mimi,Bi/Bwana.....mzazi wa.....

Nimekubali kushiriki katika utafiti huu baada ya kuelezwa na daktari Sahihi yangu ni thibitisho ya kwamba nimeelewa umuhimu wa utafiti huu.

Sahihi..... Uhusianotarehe

Kibali cha mtoto (miaka 8-17)

Nimeelezwa kuhusu utafiti huu na daktari na nimeelewa umuhimu wake.

Sahihi yangu ni thibitisho ya kwamba nimekubali kushiriki katika utafiti huu.

Sahihi ya mtafiti Tarehe

Daktari Beatrice Mwangi,

Sahihi Tarehe

APPENDIX II: QUESTIONNAIRE

CODE.....

TITLE: THE PREVALENCE OF CHRONIC RHINOSINUSITIS IN CHILDREN WITH DYSPEPSIA IN KENYATTA NATIONAL HOSPITAL AND GERTRUDES CHILDRENS HOSPITAL.

DATE:

I.BIODATA

1. What your sex: Male..... Female.....
2. Age in years
3. Weight in kilogram.....

II.HISTORY

1. Any nasal blockage? Yes
- No.....

How long??

2. Any facial pain? Yes.....
- No.....

3. Is there nasal discharge? Yes
- No.....

What colour and texture?

4. Is there reduced smell? Yes No.....

5. Is there hotness of body? Yes

No.....

6. Headache? Yes

No.....

7. Is there fatigue? Yes

No.....

8. Any bad smell from the mouth? Yes

No.....

9. Tooth pain? Yes.....

No.....

10. Any episodes of cough? Yes.....

No.....

11. Ear pain? Yes.....

No.....

12. Ear blockage? Yes.....

No.....

III. ENT EXAMINATION

1. Anterior Rhinoscopy:
2. PNS Drip:

IV. LABORATORY RESULTS

1. Laboratory results (routine stool specimen):
2. Reflux symptom index score

APPENDIX III: THE REFLUX SYMPTOM INDEX

Within the Past one Month, How Did the Following Problems Affect?

Ordinal Scale: 0- No problem

5- Severe problem

Symptoms	Score					
	0	1	2	3	4	5
Hoarseness or other voice problems	0	1	2	3	4	5
Clearing throat	0	1	2	3	4	5
Excess throat mucus or postnasal drip	0	1	2	3	4	5
Difficulty swallowing food, liquid, or pills	0	1	2	3	4	5
Coughing after eating or after lying down	0	1	2	3	4	5
Breathing difficulties or choking episodes	0	1	2	3	4	5
Troublesome or annoying cough	0	1	2	3	4	5
Sensations of something sticking in throat or lump in throat	0	1	2	3	4	5
Heartburn, chest pain, indigestion, or stomach acid coming up	0	1	2	3	4	5

SCORE: