"EFFICACY OF NORMAL SALINE NASAL SPRAY ADDED TO STANDARD TREATMENT REGIMEN OF CHRONIC RHINOSINUSITIS: A RANDOMIZED CONTROLLED TRIAL"

By

REGISTRATION NO: BE0120001

Dissertation

Submitted to

KLE Academy of Higher Education and Research,
Belagavi, Karnataka

In partial fulfilment
of the requirements for the degree of

MASTER OF SURGERY

IN

OTORHINOLARYNGOLOGY AND HEAD AND NECK SURGERY

DEPARTMENT OF OTORHINOLARYNGOLOGY AND HEAD AND NECK SURGERY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELAGAVI, KARNATAKA

JUNE/JULY – 2023

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH BELAGAVI, KARNATAKA

ENDORSEMENT BY THE HOD, PRINCIPAL/HEAD OF THE INSTITUTION

This is to certify that the dissertation entitled **"EFFICACY OF NORMAL SALINE NASAL SPRAY ADDED TO STANDARD TREATMENT REGIMEN OF CHRONIC RHINOSINUSITIS: A RANDOMIZED CONTROLLED TRIAL"** is a bonafide and genuine research work carried out by **REGISTRATION NO: BE0120001**

Dr. B.P. BELALDAVAR M.S (ENT), D.L.O, Ph.D,
Professor & Head of Department
Department of Otorhinolaryngology
and Head & Neck Surgery,
J.N.Medical College,
Nehru Nagar, Belagavi -590010

Date: 2/1/23
Place: Belagavi

Dr.(Mrs)N.S.MAHANTASHETTI M.D,(Peads)
Principal
J.N.Medical College,
Nehru Nagar,
Belagavi -590010

PRINCIPAL
J.N. Medical College,
BELAGAVI- 590 010

KAHER BELAGAVI J.N. MEDICAL COLLEGE

Date: 2/1/23
Place: Belagavi

UNDERTAKING

I,**Reg. No. BE0120001,** hereby declare that the information and the data mentioned in my dissertation entitled **"EFFICACY OF NORMAL SALINE NASAL SPRAY ADDED TO STANDARD TREATMENT REGIMEN OF CHRONIC RHINOSINUSITIS: A RANDOMIZED CONTROLLED TRIAL"** belongs to me and is original. I am aware of the definition of plagiarism as detailed below:

- An act or instance of using or closely imitating the language and thoughts of another author without authorization and the representation of that author's work as one's own, as by not crediting the original author.
- A piece of writing or other work reflecting such unauthorized use or imitation.
- The deliberate or reckless representation of another's words, thoughts or ideas as one's own without attribution in connection with submission of academic work, whether graded or otherwise.

I hereby declare that the dissertation prepared by me is original one and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the University.

Date: 27-12-2022
Place: Belagavi

Reg. No: BE0120001

PLAGIARISM CERTIFICATE

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH

JAWAHARLAL NEHRU MEDICAL COLLEGE

(Recognized by Medical Council of India, New Delhi)

Accredited 'A+' Grade by NAAC (3rd Cycle) **Placed in Category 'A' by MHRD (GoI)**

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 - 2471350 0831 - 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 28-11-2022

ACCEPTANCE LETTER

The softcopy of thesis entitled: **"EFFICACY OF NORMAL SALINE NASAL SPRAY ADDED TO STANDARD TREATMENT REGIMEN OF CHRONIC RHINOSINUSITIS: A RANDOMIZED CONTROL TRIAL"** has been submitted for Anti-Plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of **06%** which is within the acceptable limits of **10%** as per the guidelines given by UGC.

Guide.

KAHER BELAGAVI J. N. MEDICAL COLLEGE

Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BE0120001.
Postgraduate Student,
2020-21 Batch,
Department of ENT,
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE LETTER

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 57 **Date: 25/01/2021**

To,

PG student in Otorhinolaryngology and Head & Neck Surgery,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"EFFICACY OF NORMAL SALINE NASAL SPRAY ADDED TO STANDARD TREATMENT REGIMEN OF CHRONIC RHINOSINUSITIS: A RANDOMIZED CONTROL TRIAL",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

ABSTRACT

Title: "Efficacy of normal saline nasal spray added to standard treatment regimen of chronic rhinosinusitis: a randomized controlled trial."

Background and Objective: Chronic Rhinosinusitis is characterized by inflammation of the lining of nose and paranasal sinuses leading to nasal blockage/discharge, facial pressure/pain and loss of smell sensation. It is one of the most common chronic health problems globally. Its impact on health and quality of life of the individual is constantly increasing both in developing and developed countries. However, the easily accessible and affordable intervention in early stages results in considerable improvement of symptoms. The aim of medical treatment is to reduce inflammation, reduce bacterial load and optimize ciliary function by removing mucus. Hence one of the approaches that is followed in conservative management includes a combination therapy with intranasal corticosteroids and empirical antibiotics, which aim at targeting intrinsic mucosal inflammation and reducing microbial load, respectively. Saline irrigation is useful in improvement of mucociliary clearance by the removal of mucus, infected crusts and pro-inflammatory agents. No recent studies have been done that evaluate the role of Normal Saline separately. The aim of this study was to evaluate efficacy of adding normal saline nasal spray to standard treatment regimen of chronic rhinosinusitis.

Materials And Methods: This is a one-year randomized controlled, blinded study conducted between January 2021 to December 2021 in Otorhinolaryngology OPD of Department of Otorhinolaryngology and Head and Neck Surgery, J. N. Medical College, KAHER, Belgaum. Study consisted of 40 chronic rhinosinusitis patients divided into Study and Control group of 20 patients each. The study group was prescribed Normal Saline nasal spray (1 puff in each nostril, thrice daily) in addition

to topical corticosteroids (Mometasone furoate nasal spray, 1 puff in each nostril, twice daily; 1 puff=50µg), and oral antibiotics (Amoxiclav, 30mg/kg, twice daily), while the control group was only prescribed topical corticosteroids and oral antibiotics. Patients were evaluated using Lund–Kennedy Endoscopic Scores (LKES) and SNOT-22 before and after the treatment.

Results: There was a significant improvement in LKES, with pre-treatment and post-treatment scores for control group being 5.35±2.43 vs 3.70±1.95 respectively ($p=0.0116$), whereas for test group, pre-treatment and post-treatment scores were 8.15±2.62 vs 6.05±2.04 respectively ($p=0.0037$). Improvement in SNOT-22 scores were observed as well, with pre-treatment and post-treatment scores for control group being 38.90±12.01 vs 25.70±9.21 respectively ($p=0.0002$), whereas for test group, pre-treatment and post-treatment scores were 49.85±11.38 vs 31.55±9.91 respectively ($p<0.0001$).

Conclusion: At the end of study, it was concluded that addition of normal saline nasal spray to medical management of chronic rhinosinusitis is efficacious which was evident through endoscopic as well as symptomatic improvement in patients. Normal saline is helpful in clearance of mucous, infected crusts and pro-inflammatory agents via mucous thinning, enhanced mucociliary evacuation from sinuses, reduced edema, and decreased antigen load in the nasal and sinus cavities, hence reducing the symptoms of chronic rhinosinusitis.

LIST OF ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
CRS	Chronic Rhinosinusitis
CRSsNP	Chronic Rhinosinusistis without Nasal Polyposis
CRSwNP	Chronic Rhinosinusitis with Nasal Polyposis
Ig	Immunoglobulin
Th	T-helper
SAG	Superantigen
CF	Cystic Fibrosis
SNOT-22	Sino-nasal Outcome Test - 22
LK	Lund Kennedy
LKES	Lund-Kennedy Endoscopic Scoring
DNE	Diagnostic Nasal Endoscopy
NSI	Normal Saline Irrigation
HS	Hypertonic Saline
IS	Isotonic Saline

TABLE OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF PHOTOGRAPHS

INTRODUCTION

Chronic Rhinosinusitis (CRS) is an inflammatory process of the mucosa of nasal cavity and paranasal sinuses. It is one of the most common chronic health problems affecting about 11% of the population globally.[1] Its impact on health and quality of life of the individual is constantly increasing both in developing and developed countries. However, the easily accessible and affordable intervention in early stages results in considerable improvement of symptoms.

The aim of medical treatment is to reduce inflammation, reduce bacterial load and optimize ciliary function by removing mucus.[1] Hence one of the approaches that is followed in conservative management includes a combination therapy with empirical antibiotics and intranasal corticosteroids, that aim at reducing microbial load and targeting mucosal inflammation, respectively. In order to optimize mucociliary clearance, a number of modalities have been tried like saline irrigation and use of mucoactive agents.

Saline irrigation is useful in improvement of mucociliary clearance by the removal of mucus, infected crusts and pro-inflammatory agents. Isotonic saline and hypertonic saline are the most commonly used modalities for nasal irrigation. Although use of hypertonic saline has been shown to have better radiological improvement in comparison to isotonic saline, it is at the cost of higher rates of local adverse effects such as inflammation and rhinorrhea.[1] Normal Saline can be thought of a superior option for the advantage of it having better fluid balance in addition to better nasal patency[2] and tolerability.

In a study by AS DeConde et al, approximately one in three patients who initially elected medical treatment subsequently chose surgical intervention[2][3], which is high, which is followed by a post-operative follow-up of an average of 3 months. Hence, our study aims to know whether addition of intranasal normal saline in medical line of treatment of chronic rhinosinusitis will reduce the need for surgery as well as long term follow-up.

Intranasal Normal Saline is a commonly practiced post-operative therapy in secondary chronic rhinosinusitis. However, its role as a first line treatment modality in primary chronic rhinosinusitis has not been extensively studied. Very few recent studies[4][5][6][7], especially in India[5], have been performed that examine the effect of topical Normal Saline in conservative management of chronic rhinosinusitis. Normal Saline effect in comparison to alternate options of nasal irrigations has been studied. However, no recent studies have been done that evaluate the role of Normal Saline separately.

Our study aims to observe the role of supplementing nasal irrigation in the form of Normal Saline spray along with systemic antibiotics and intranasal corticosteroids in comparison to treatment with only antibiotics and corticosteroids. The results are evaluated both subjectively and endoscopically. The goal was to assess the benefit of adding topical Normal Saline spray in the first line treatment of chronic rhinosinusitis.

OBJECTIVE

The rationale of our study was to evaluate efficacy of adding normal saline nasal spray to standard treatment regimen of chronic rhinosinusitis.

REVIEW OF LITERATURE

Nasal obstruction, congestion, blockage, or discharge—which may be anterior or posterior nasal drip—must be one of two or more symptoms that characterize this inflammation of the nose and paranasal sinuses known as CRS. Other signs may include face pressure or pain, a diminished or absent sense of smell (in adults), or coughing (in children).[8]

There must have been symptoms for at least 12 weeks. Additionally, individuals must exhibit either endoscopic signs of at least one of the following: nasal polyps, mucopurulent discharge primarily from the middle meatus, or oedema/mucosal obstruction primarily in the middle meatus, or mucosal changes within the osteo-meatal complex and/or sinuses as demonstrated by a CT scan.[9]

In India, 1 in 8 people, or 5 to 15% of the urban population, are affected by CRS. There have been reports that sinusitis has a higher prevalence than any other chronic ailment (146/1000 people), and it is supposedly getting more common.[10] The quality of life is greatly reduced by symptoms such nasal blockage, nasal discharge, sleep disruption, anosmia, and facial pain. Exacerbations of respiratory diseases, ineffective symptom control, and acute exacerbations are frequent. Although they are uncommon, complications can arise, such as vision impairment and intracranial infection.

Several theories have been put out in the recent years to explain the clinical CRS spectrum. These hypotheses provide evidence in favour of the dysfunctional interaction of exogenous factors and individual host traits leads to CRS. Overall, this reinforces the notion that CRS is best understood as an improper interaction that takes

place at the sinonasal mucosa, the point of contact between the host and the environment.

Allergens, toxins, and microbiological pathogens are environmental causes of CRS, whereas immune system abnormalities are host causes.. In the paranasal sinuses, nasal allergens have been discovered to cause pathophysiologic alterations that are somewhat analogous in character to the reactions seen in CRS.[11,12] Skin test reactivity and increased IgE are inflammatory profiles associated with atopy, albeit this is not always the case in CRS patients.[13,14] Furthermore, neither clinical nor radiographic criteria used to assess the severity of CRS have been observed to significantly correlate with atopic condition.[15, 16] Overall, most researchers do not think that AR is the main cause of CRS; rather, they think that it is a problem that is superimposed that influences the inflammation found in CRS to a variable but generally minor degree. In CRS, microbes, particularly fungus and bacteria, are thought to be the most important environmental contributors. Among the host factors associated with CRS are deficiencies in the mechanical, innate, and adaptive components of the immune system. The following are some theories on the etiology and pathogenesis of CRS: hypotheses that highlight important environmental elements involved in the disease process (1) the fungal hypothesis, (2) the superantigen hypothesis, (3) the biofilm hypothesis, and (4) the microbiome hypothesis, and hypotheses which describe specific host factors (5) the eicosanoid hypothesis and (6) the immune barrier hypothesis, which describe specific host factors.[17]

The Fungal Hypothesis: Although fungi are no longer considered to be the main causes of CRS, an elevated level of fungal colonization is still considered to be a

significant disease modifier. Intrinsic proteases found in fungi can activate PAR receptors on a variety of cell types to induce cytokines, potentially activating T-helper (Th) 2 responses.[18-22] Fungal extracts can block epithelial JAK-STAT1 signaling, which may suppress Th1 and stimulate Th2 responses.[23] In the case of classic allergic fungal sinusitis, fungi undoubtedly play a significant role.[24]

Last but not least, chitin is a component of fungal cell walls that in numerous animals and human models has been discovered to cause a Th2 response, however its potential significance in CRS is yet unknown.[25–27] Currently, the majority of researchers believe that fungi probably have a significant impact on a subset of CRS patients.

The Bacteria-based Hypothesis: Both healthy individuals and individuals with CRS have bacteria colonizing their sinonasal tracts in addition to fungi., most prominently Staphylococcus aureus.[28, 29] Staphylococcus can live inside the macrophages and epithelial cells of CRS patients in addition to surface colonization.[30–32] The following are the three bacterial-based theories that have been put forth: (a) the superantigen hypothesis, (b) the biofilm hypothesis, and (c) the microbiome hypothesis.

a) The Superantigen Hypothesis: It states that local eosinophilic reactions are amplified by Staphylococcus bacteria's superantigenic (SAGs) exotoxins through a variety of methods, which promotes polyp formation.[33, 34] These toxins act by eliciting a massive and unchecked immune reaction that can activate up to 30% of the T cell population. in affected individuals, in contrast to the 0.001% of T cells that are activated in a typical antigen-specific immunological response.[35] B cells are among the many different cell types that are impacted, and this causes a

localized polyclonal IgE response in nasal polyps.[33, 36, 37-44] Significant portions of controls, CRSsNP patients, and Cystic Fibrosis (CF) patients have been colonized with Staphylococcus, but none of the tissues from these groups demonstrate any SAG effects.[45] Therefore, it is unclear if SAGs are directly responsible for the inflammatory response in the tissues; they may merely serve to amplify it. Therefore, a SAG impact would significantly increase the severity of an already established Th2-skewed response in eosinophilic polyps, resulting in a more clinically severe phenotype. As a result, Staphylococcus superantigens are typically viewed as disease modifiers rather than specific etiologic factors in the development of nasal polyposis.

b) Biofilm Hypothesis: Communities of bacteria are enclosed in sophisticated, highly organized structures called biofilms that are protected by an extracellular matrix. This exterior matrix, which is made up of polysaccharides, nucleic acids, and proteins, gives bacteria a way to lower their metabolic rates in growth-unfavorable conditions, shielding them from both host defences and common antibiotics. S. aureus biofilms are most frequently linked to CRS, the other bacterial species being Pseudomonas aeruginosa, Streptococcus pneumonia, Haemophilus influenza, and Moraxella catarrhalis which are also known to form biofilms. However, there is no concrete proof to support the idea that bacterial biofilms contribute to the initial development of CRS.[46] As a result, from the perspective of CRS pathogenesis, any biofilm hypothesis would default to the superantigen hypothesis.

c) Microbiome Hypothesis: The commensal microorganisms to some extent by concealing antimicrobial proteins and producing lipid by-products that help maintain homeostasis by depressing pathogen growth.[47, 48] This shows that the

resolution of inflammation may be linked to the repair of the microbiome through probiotics or inoculation with a sample of healthy bacteria.[49] Antibiotics or virally induced modifications to the sinonasal microbiome may facilitate the formation of pathogenic organisms that cause CRS., according to a preliminary study in this area.[50] To be confirmed, the CRS microbiome theory needs to be studied more thoroughly.

Host-related Hypotheses of CRS: Defects in this system could hypothetically explain the persistent inflammation that characterizes CRS. The host can be naturally protected from damage brought on by environmental causes through the mucosal immune system. If the foreign pathogenic stimulation is severe enough, an adaptive immune response will be set off with highly specific T and B proliferation. The persistent nature of the inflammatory response in CRS suggests ongoing immune system stimulation. Hence, two broad host-related theories have been suggested: a) eicosanoid theory b) immune barrier theory.

a) Eicosanoid Theory: The metabolism of arachidonic acid results in the production of signaling molecules called eicosanoids that have inflammatory and immunologic properties and are secreted by a wide range of cell types. More recently, it has been suggested that deficiencies in the eicosanoid system, which have long been intimately linked to aspirin sensitivity, may also contribute to CRSwNP in general.[45, 51] Eicosanoids may also modify the effects of Staphylococcal superantigens, according to certain evidence. [52, 53] Leukotriene inhibitors' success in treating nasal polyposis, however, has faded the interest in this route as a key contributor to the onset of CRS.

b) Immune Barrier Theory: According to the immune barrier theory, when exposed to relatively common microbial pathogens, deficiencies in the physical barrier and the innate immune response contribute to the development of CRS.[54, 55] In contrast to most of the earlier work, which focused on downstream cellular infiltration, this attention on events at the mucosal interface is novel. Airway mucus, which traps foreign objects, and intercellular tight junctions between respiratory epithelial cells make up the physical barrier upstream. Together, they provide a semi-permeable barrier that restricts entry across the mucosa. Patients with CF, who have a known aberrant mucociliary flow and a very high prevalence of CRS, both with and without nasal polyps, provided the first evidence for upstream abnormalities causing CRS.[56] This highlights the significance of mucociliary flow to nose and sinus homeostasis and the fact that even carriers of the CF mutation without the clinical disease have a considerably greater prevalence of CRSsNP.[57] Later research revealed that CRS had a more widespread impairment in mucociliary clearance, which prolonged transit times and, thus, exposed patients to foreign material.[58, 59] The mechanical barrier has also been shown to be compromised, as evidenced by decreased tight junctional proteins and higher sensitivity to exogenous protease degradation in later investigations.[60-62] Functional tests showed that the barrier in CRSwNP was permeable, allowing more foreign material to pass through the epithelium.[63, 64] Oncostatin M, a cytokine that can break down tight junctions, has recently been found to be present in higher amounts in afflicted tissues, which may contribute to barrier abnormalities in CRS.[65] Overall, our findings imply that mucociliary failure exists in both types of CRS, but that CRSwNP is more closely associated with a porous barrier. It's still not apparent whether any barrier deficiencies are

brought on by the host's core genetic variation, epigenetic changes brought on by environmental stressors, or a combination of the two. However, it should be noted that epidemiological studies show that CRS patients typically have chronic inflammation in their noses, sinuses, and frequently their lower respiratory tracts.[66, 67] This increases the likelihood that the primary host genes regulating CRS development will control respiratory mucosal immunobiology rather than the systemic immune response.

Depending on whether nasal polyps are present or not, there are two main phenotypes of chronic rhinosinusitis. Most frequently emerging from the osteomeatal complex, nasal polyps are hyperplastic swellings of the nasal mucosa that resemble tumours.[68] When polyps are observed (either directly or endoscopically) in the middle meatus on both sides of the nose, chronic rhinosinusitis with nasal polyps (CRSwNP) is the conclusion. The situation where no polyps are present is referred to as CRSsNP.

Although judgments about the patient's CRS should be based on its cause, in practice therapeutic decisions may be made without knowledge of the patient's polyp status, particularly in primary care.

Steroids, antibiotics, and saline are some of the most frequently utilized either topically (spraying into the nose) or systemically (taken orally) therapies for CRS.

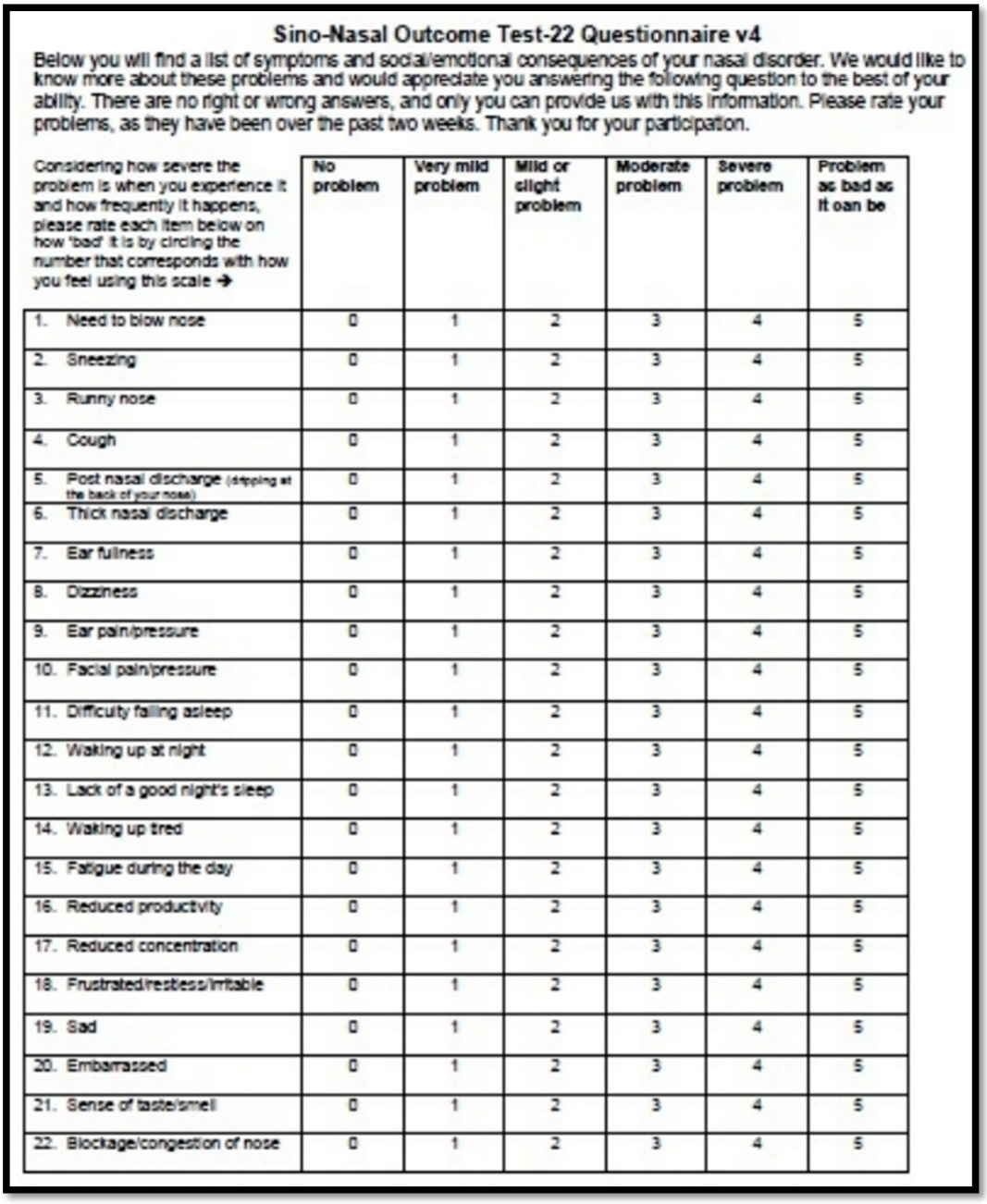
A method called nasal irrigation involves using salt water solutions that are either isotonic or hypertonic to rinse the nasal cavity. Other names for it include nasal douche, wash, and lavage. One nostril receives an injection of saline solution, which then bathes the nasal cavity as it exits the other. One can also use a nebulizer or low positive pressure with a spray, pump, or squirt bottle to perform saline nasal

irrigation. Many individuals with chronic rhinosinusitis utilize this over-the-counter medication as a supplement to their regular medical care.

Saline nasal irrigation's precise method of action is uncertain. Saline nasal irrigation may improve the function of the nasal mucosa through a variety of physiological processes, including direct cleansing of mucus because it can serve as a breeding ground for bacteria; saline thins mucus and aids in its clearance; removal of antigens, biofilm, or inflammatory mediators, which reduces inflammation; as well as improved mucociliary function by increasing ciliary beat frequency.[69] The notion that nasal saline irrigation is safe, affordable, and generally accessible has led to its widespread adoption.

In our study, on individuals with chronic rhinosinusitis, the effects of nasal saline combined with various therapies, such as intranasal corticosteroids, oral antibiotics, are investigated. The results are compared using Sino-nasal Outcome Test (SNOT-22) and Lund-Kennedy Endoscopy Scoring (LKES).

A patient-reported outcome measure called the 22-item Sino-nasal Outcome Test (SNOT-22) was developed to assess how CRS affected health-related quality of life.[70] The information in SNOT-22 covers the effects of CRS on severity of symptoms, productivity, social and emotional impact, and sleep. Items are given a score between 0 (no problem) and 5 (problem as terrible as it can be), which is added up to give a final score between 0 and 110.[71]



Sino-Nasal Outcome Test-22 Questionnaire v4

Below you will find a list of symptoms and social/emotional consequences of your nasal disorder. We would like to know more about these problems and would appreciate you answering the following question to the best of your ability. There are no right or wrong answers, and only you can provide us with this information. Please rate your problems, as they have been over the past two weeks. Thank you for your participation.

Considering how severe the problem is when you experience it and how frequently it happens, please rate each item below on how 'bad' it is by circling the number that corresponds with how you feel using this scale →	No problem	Very mild problem	Mild or slight problem	Moderate problem	Severe problem	Problem as bad as it can be
1. Need to blow nose	0	1	2	3	4	5
2. Sneezing	0	1	2	3	4	5
3. Runny nose	0	1	2	3	4	5
4. Cough	0	1	2	3	4	5
5. Post nasal discharge (dripping at the back of your nose)	0	1	2	3	4	5
6. Thick nasal discharge	0	1	2	3	4	5
7. Ear fullness	0	1	2	3	4	5
8. Dizziness	0	1	2	3	4	5
9. Ear pain/pressure	0	1	2	3	4	5
10. Facial pain/pressure	0	1	2	3	4	5
11. Difficulty falling asleep	0	1	2	3	4	5
12. Waking up at night	0	1	2	3	4	5
13. Lack of a good night's sleep	0	1	2	3	4	5
14. Waking up tired	0	1	2	3	4	5
15. Fatigue during the day	0	1	2	3	4	5
16. Reduced productivity	0	1	2	3	4	5
17. Reduced concentration	0	1	2	3	4	5
18. Frustrated/restless/irritable	0	1	2	3	4	5
19. Sad	0	1	2	3	4	5
20. Embarrassed	0	1	2	3	4	5
21. Sense of taste/smell	0	1	2	3	4	5
22. Blockage/congestion of nose	0	1	2	3	4	5

Fig. 1: ***Sin-Nasal Outcome Test-22 Questionnaire***

In 1995, Lund and Kennedy, heading the Staging and Therapy Group for Chronic Rhinosinusitis, proposed the Lund-Kennedy (LK) endoscopic scoring.[72] To date, it remains the most frequently utilized and referenced endoscopic scoring system in rhinology outcomes research. The five words that make up the LK Endoscopy

Score are polyposis, discharge, edema, scarring, and crusting. Each term is evaluated on an ordinal scale from 0 to 2 for each side. Higher scores indicate worse observed disease

LUND-KENNEDY ENDOSCOPIC SCORING [72]

Criteria of Assessment	Scores		
	0	1	2
Polyps in middle meatus	Absent	Restricted to middle meatus	Beyond middle meatus
Discharge in middle meatus	Absent	Thin and clear discharge	Thick and purulent discharge
Edema of the middle meatus	Absent	Mild-moderate	Moderate-severe
Scarring in middle meatus	Absent	Mild-moderate	Moderate-severe
Crusting in middle meatus	Absent	Mild-moderate	Moderate-severe

Fig. 2: ***Lund-Kennedy Endoscopic Scoring***

In our study, the SNOT-22 scoring and LKES are compared before and after treatment and efficacy of normal saline as a treatment option for chronic rhinosinusitis is evaluated.

MATERIALS AND METHODS

STUDY SETTING- Hospital Based study

STUDY DESIGN- Randomized Control Study

METHOD OF RANDOMIZATION- computer generated randomization

STUDY PERIOD- 1 year

STUDY POPULATION- All cases of chronic rhinosinusitis attending Otolaryngology OPD in KLES Dr. Prabhakar Kore Hospital, Belgaum from January 2021 - December 2021

SAMPLE SIZE (n)- 40

The minimum sample size formula based on mean and standard deviation is

$$n = \frac{(z_\alpha + z_\beta)^2(s_1^2 + s_2^2)}{(\bar{X}_1 - \bar{X}_2)^2}$$

where z_α is linked with the level of significance and z_β is linked with the power of the test. For 5% level of the significance z_α =1.96 and z_β =0.84 for 80% power of the test.

Ref:

$\bar{x}_1$ is the mean of first study group (7.8) whereas $\bar{x}_2$ is the mean of second study group (5.9).

s_1 is the standard deviation of the first study group (2.2) whereas s_2 is the standard deviation of the second study group (1.6).

With the values hence obtained, the sample size calculated is 16.

To make our study more confirmative, the sample size will be raised to 20.

There will two groups each with size 20.

STATISTICAL ANALYSIS- Our study is focused on comparison of two groups. The mean and the standard deviation for the continuous quantitative variables were calculated. The Unpaired Student's t test and other appropriate statistical methods were used to compare the continuous variables between groups. Using the Student's Paired t test, two quantitative variables within a group were compared.

Rates, ratios, and percentages were used to express the categorical data. Using the Chi-square test or Fisher's exact test, the relationship between the result, clinical, and demographic factors was evaluated.

Discrete variables were represented by median.

Nonparametric tests were used for comparing discrete variables.

Graphs that adequately reflected the comparison were utilized.

For all tests, a value of p which was less than 5% (0.05) was considered significant.

SPSS software was used for statistical analysis.

INCLUSION CRITERIA-

- 18 to 65 years of age
- Confirmed case of chronic rhinosinusitis
- Patients ready to participate in the trial

EXCLUSION CRITERIA-

- Less than 18 years of age
- More than 65 years of age
- Patients with immunocompromised status
- Past history of nasal surgery
- A known case of head and neck malignancy

- Previous radiotherapy to head and neck
- Mucociliary clearance disorders

METHODOLOGY-

- Patients' details and history were obtained.
- Clinical examination was performed.
- Any other symptoms apart from nose complaints were noted.
- A thorough clinical examination was done for all patients with anterior and posterior rhinoscopy and Diagnostic Nasal Endoscopy (DNE).
- Pre-treatment Lund-Kennedy Endoscopic Score (LKES) as well as 22-item Sinonasal Outcome Test (SNOT- 22) were assessed and noted in the same proforma.
- After clinical diagnosis, using computer generated randomization, the subject was allocated to either the Study group or the Control group.
- The patients in study group were prescribed Normal Saline nasal spray (1 puff in each nostril, three times a day) in addition to the standard treatment regimen of CRS comprising of topical corticosteroids (Mometasone furoate nasal spray, 1 puff in each nostril, two times a day; 1 puff=50µg), and oral antibiotics (Amoxiclav, 30mg/kg, two times a day), while the control group was only prescribed topical corticosteroids and oral antibiotics.
- Patients were re-assessed after 3 weeks of receiving treatment using post-treatment SNOT-22 score and DNE.
- Lund-Kennedy endoscopy scoring system was used to compare pre-treatment and post-treatment endoscopy outcomes.

Diagnostic Nasal Endoscopy (DNE)

Patients' each of the nasal cavities were packed using 4% lignocaine and xylometazoline 15 minutes before the procedure. Supine position with 30° head-end elevation was given and DNE was performed using 0° 4mm adult endoscope for all patients.

The three passes performed were:

- 1st Pass

Between the inferior turbinate and septum, the endoscope was carefully passed into the nasal cavity. For spurs or deviations, the septum was examined. Examination of the inferior turbinate for hypertrophy was carried out.. The posterior choana was examined for any obstructing pathology. The scope was advanced and nasopharynx inspected. On withdrawing, the scope was rolled into the inferior meatus, The opening of nasolacrimal duct was examined for on the roof of the inferior meatus.

- 2nd Pass

The posterior choana was reached after passing the scope along the floor. The middle turbinate was reached by advancing the scope medially upward along the anterior surface of the sphenoid and over the roof of the posterior choana. The superior turbinate and meatus were noticed visualized. One could see the spheno-ethmoidal recess was noted too.

- 3rd Pass

Middle meatus was entered by rolling endoscope under the inferior border of middle turbinate. The middle meatus's contents were are observable on withdrawing the scope from posterior to anterior direction.

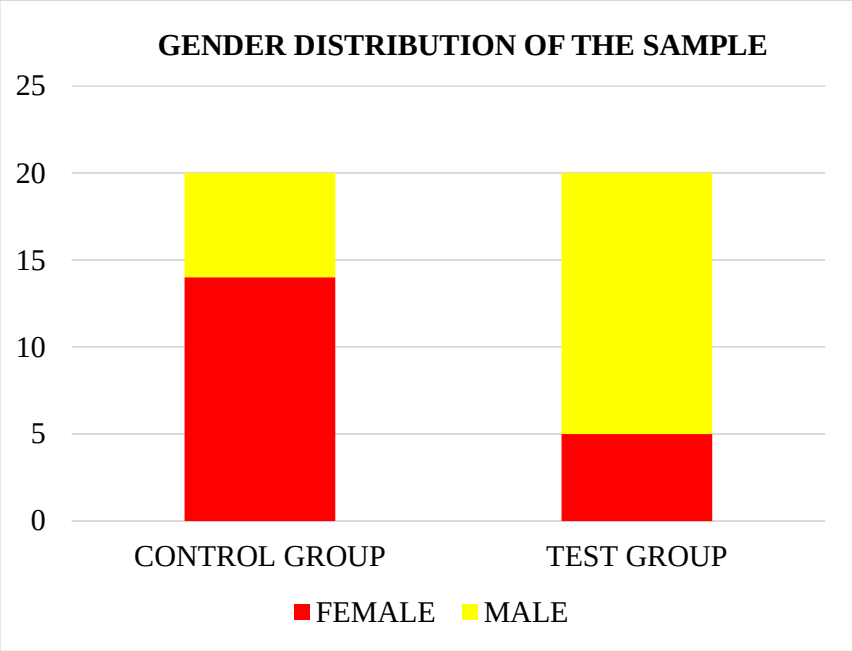
ETHICAL CONSIDERATIONS-

Ethical clearance for the study was obtained from the JNMC Institutional Ethics Committee on Human Subjects Research and the reference number was MDC/DOME/57.

RESULTS AND ANALYSIS

- **GENDER DISTRIBUTION**

In our study 40 patients were included, 19 females (47.5%) & 21 males (52.5%).

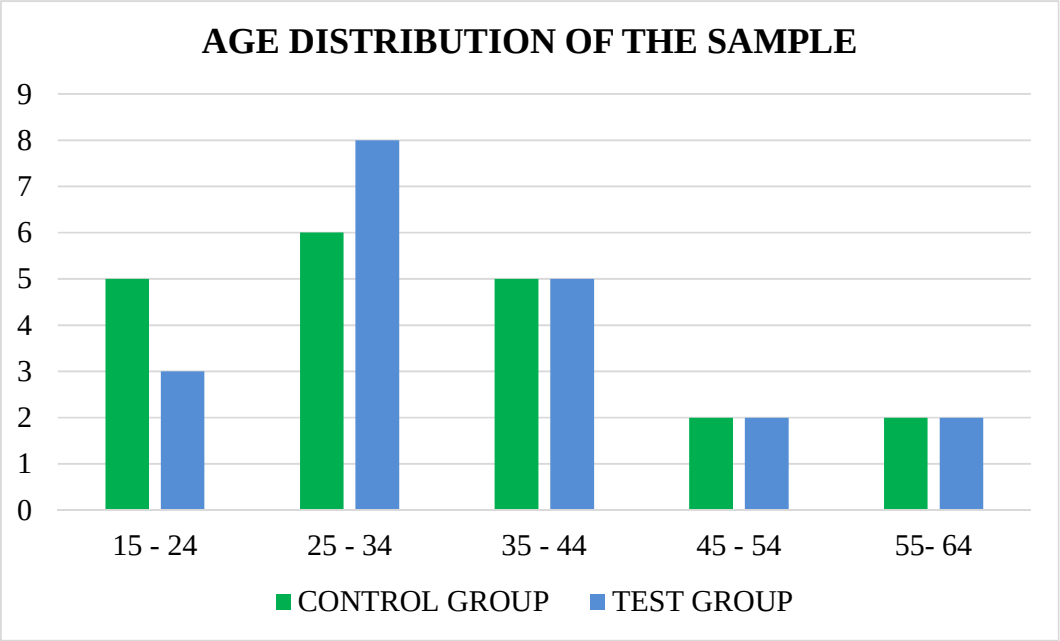


Graph 1: ***Gender distribution***

	CONTROL GROUP		TEST GROUP	
GENDER	**NUMBER**	**%**	**NUMBER**	**%**
FEMALE	14	70	5	25
MALE	6	30	15	75
TOTAL	20	100	20	100

Table 1: ***Gender distribution***

- **AGE DISTRIBUTION**



Graph 2: ***Age Distribution***

Our study includes 5 age groups:

18-24 years – 20%

25-34 years – 35%

35-44 years – 25%

45-54 years – 10%

55-65 years – 10%

	CONTROL GROUP		TEST GROUP	
AGE	**NUMBER**	**%**	**NUMBER**	**%**
15 - 24	5	25	3	15
25 - 34	6	30	8	40
35 - 44	5	25	5	25
45 - 54	2	10	2	10
55- 64	2	10	2	10
TOTAL	20	100	20	100

Table 2a: ***Age Distribution- Grouping***

In the control group, mean age was 34.4±11.73 years with minimum age being 22 years, maximum age being 61 years.

In the test group, mean age was 35.95±12.66 years with minimum age being 19 years, maximum age being 63 years.

	MEAN	S.D.	MIN	MAX
CONTROL GROUP	34.40	11.73	22	61
TEST GROUP	35.95	12.66	19	63

Table 2b: ***Age Distribution- Statistical analysis***

- **DURATION OF SYMPTOMS**

In the control group, mean duration of symptoms was 3.37±4.83 years with minimum being 3 months, maximum being 20 years.

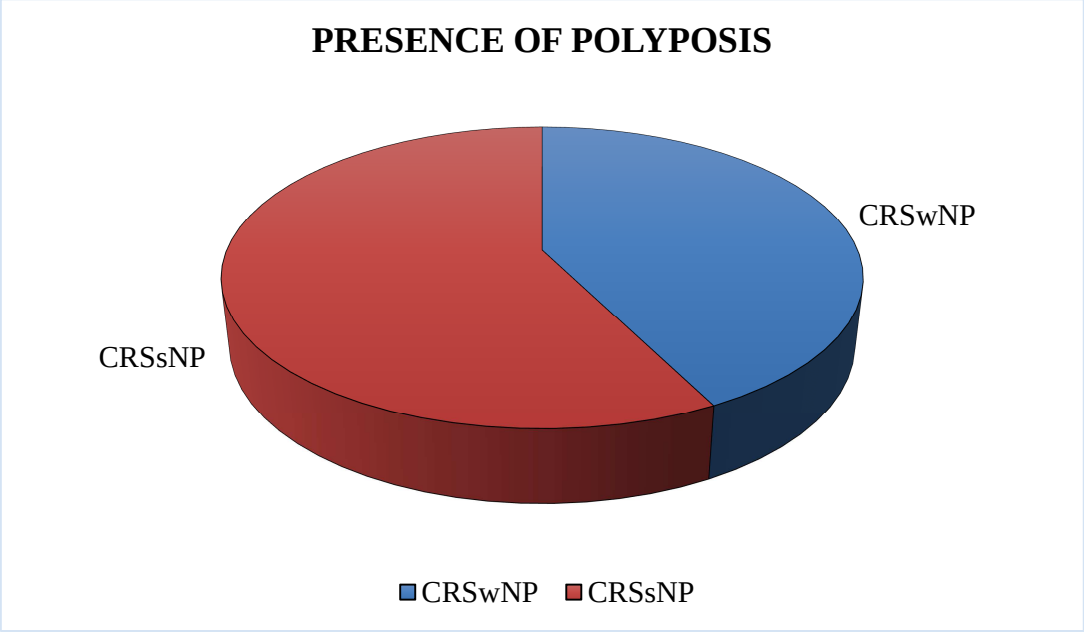
In the test group, mean duration of symptoms was 4.95±6.68 years with minimum being 3 months, maximum being 25 years.

	MEAN	**S.D.**	**MIN**	**MAX**
CONTROL GROUP	3.37	4.83	0.25	20
TEST GROUP	4.95	6.68	0.25	25

Table 3: ***Duration of Symptoms***

- **PRESENCE OF POLYPOSIS**

Presence of polyposis was noted in 42.5% of patients, 15% being in the control group while 27.5% being in the test group.



Graph 3: ***Presence of Polyposis***

	CONTROL GROUP		TEST GROUP	
PRESENCE OF POLYPOSIS	**NUMBER**	**%**	**NUMBER**	**%**
POSITIVE	6	30	11	**55**
NEGATIVE	14	70	9	45
TOTAL	20	100	20	100

Table 4: ***Presence of Polyposis***

- **LUND KENNEDY ENDOSCOPIC SCORING**

Before Treatment:

In the control group,

- mean Lund Kennedy Endoscopic Score of right nasal cavity was 2.75±1.48 with minimum score being 1, maximum being 6.
- mean Lund Kennedy Endoscopic Score of left nasal cavity was 2.60±1.19 with minimum score being 1, maximum being 6.
- mean total Lund Kennedy Endoscopic Score before treatment was 5.35±2.43 with minimum score being 3, maximum score being 11.

	CONTROL GROUP			
	MEAN	**S.D.**	**MIN**	**MAX**
RIGHT SIDE	2.75	1.48	1	6
LEFT SIDE	2.60	1.19	1	6
TOTAL	5.35	2.43	3	11

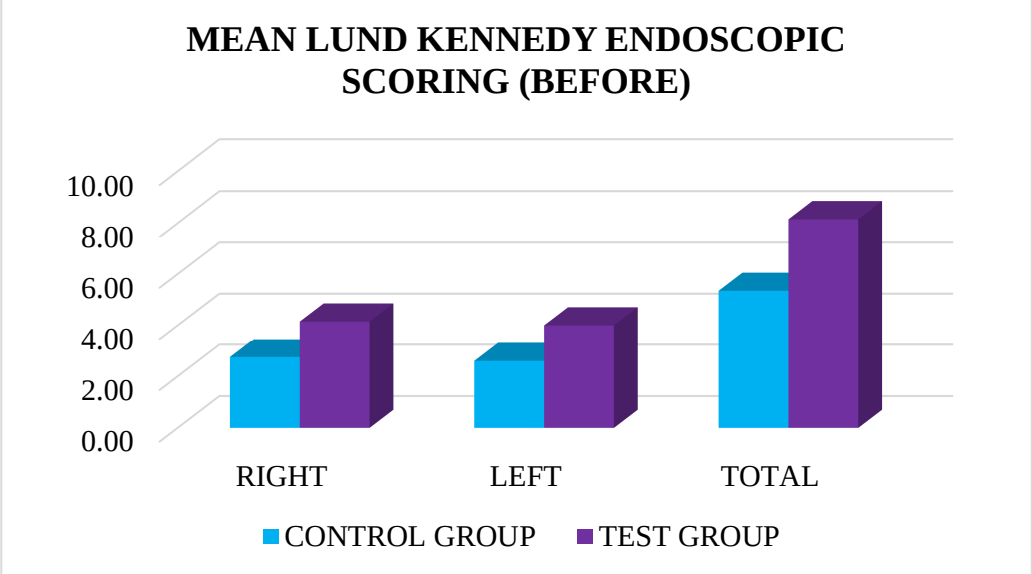
Table 5a: ***Lund-Kennedy Endoscopic Scoring, before treatment- Control group***

In the test group,

- mean Lund Kennedy Endoscopic Score of right nasal cavity was 4.15±1.35 with minimum score being 2, maximum being 7.
- mean Lund Kennedy Endoscopic Score of left nasal cavity was 4.00±1.49 with minimum score being 2, maximum being 7.
- mean total Lund Kennedy Endoscopic Score before treatment was 8.15±2.62 with minimum score being 5, maximum score being 14.

	TEST GROUP			
	MEAN	**S.D.**	**MIN**	**MAX**
RIGHT SIDE	4.15	1.35	2	7
LEFT SIDE	4.00	1.49	2	7
TOTAL	8.15	2.62	5	14

Table 5b: ***Lund-Kennedy Endoscopic Scoring, before treatment- Test group***



Graph 4a: ***Mean Lund-Kennedy Endoscopic Scoring- Before treatment***

For right nasal cavity, p value was 0.0034.

For left nasal cavity, p value was 0.0022.

For total score, p value was 0.0012.

After Treatment

In the control group,

- mean Lund Kennedy Endoscopic Score of right nasal cavity was 2.00±1.30 with minimum score being 1, maximum being 5.
- mean Lund Kennedy Endoscopic Score of left nasal cavity was 1.70±0.92 with minimum score being 0, maximum being 4.
- mean total Lund Kennedy Endoscopic Score after treatment was 3.70±1.95 with minimum score being 2, maximum score being 8.

	CONTROL GROUP			
	MEAN	**S.D.**	**MIN**	**MAX**
RIGHT SIDE	2.00	1.30	1	5
LEFT SIDE	1.70	0.92	0	4
TOTAL	3.70	1.95	2	8

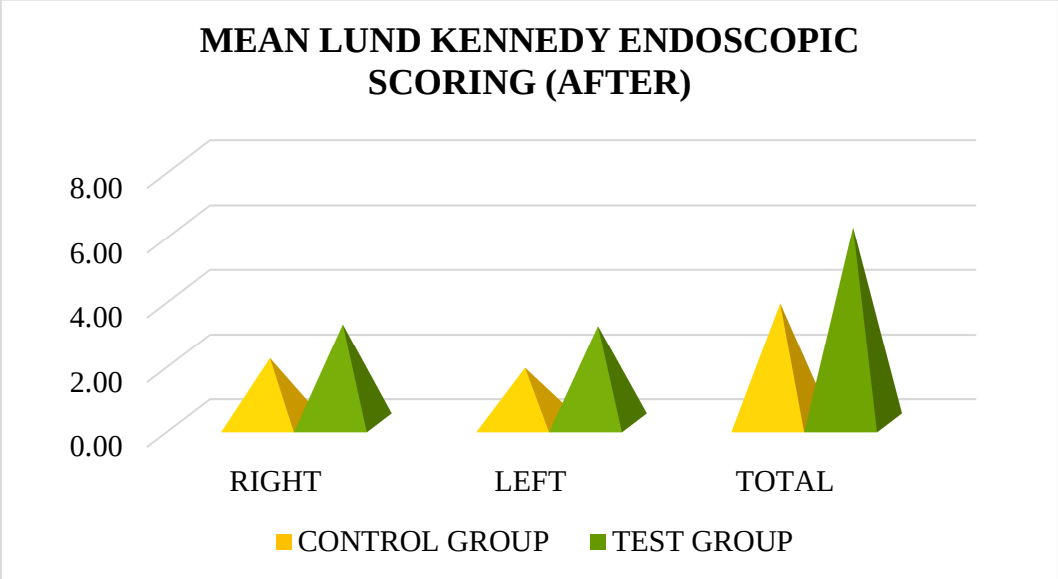
Table 6a: ***Lund-Kennedy Endoscopic Scoring, after treatment- Control group***

In the test group,

- mean Lund Kennedy Endoscopic Score of right nasal cavity was 3.05±1.10 with minimum score being 1, maximum being 5.
- mean Lund Kennedy Endoscopic Score of left nasal cavity was 3.00±1.12 with minimum score being 1, maximum being 5.
- mean total Lund Kennedy Endoscopic Score after treatment was 6.05±2.04 with minimum score being 3, maximum score being 10.

	TEST GROUP			
	MEAN	**S.D.**	**MIN**	**MAX**
RIGHT SIDE	3.05	1.10	1	5
LEFT SIDE	3.00	1.12	1	5
TOTAL	6.05	2.04	3	10

Table 6b: ***Lund-Kennedy Endoscopic Scoring, after treatment- Test group***



Graph 4b: ***Mean Lund-Kennedy Endoscopic Scoring- After treatment***

For right nasal cavity, p value was 0.0088.

For left nasal cavity, p value was 0.0003.

For total score, p value was 0.0006.

INTRA-GROUP COMPARISION:

In the following tables, p value is calculated using Student's Paired t Test.

For control group,

For right side, mean LKES before treatment was 2.75±1.48, minimum score being 1 and maximum score being 6; whereas after treatment the score was 2.00±1.30, minimum score being 1 and maximum score 5. The p-value was 0.0484.

For left side, mean LKES before treatment was 2.60±1.19, minimum score being 1 and maximum score being 6; whereas after treatment the score was 1.70±0.92, minimum score being 0 and maximum score being 4. The p-value was 0.0055.

Overall, mean LKES before treatment was 5.35±2.43, minimum score being 3 and maximum score being 11; whereas after treatment the score was 3.70±1.95, minimum score being 2 and maximum score being 8. The p-value was 0.0116.

	BEFORE				**AFTER**				**p VALUE**
	MEAN	**S.D.**	**MIN**	**MAX**	**MEAN**	**S.D.**	**MIN**	**MAX**	
RIGHT SIDE	2.75	1.48	1	6	2.00	1.30	1	5	0.0484
LEFT SIDE	2.60	1.19	1	6	1.70	0.92	0	4	0.0055
TOTAL	5.35	2.43	3	11	3.70	1.95	2	8	0.0116

Table 7a: ***Lund-Kennedy Endoscopic Scoring, intra-group comparison- Control group***

For test group,

For right side, mean LKES before treatment was 4.15±1.35, minimum score being 2 and maximum score being 7; whereas after treatment the score was 3.05±1.10, minimum score being 1 and maximum score 5. The p-value was 0.0037.

For left side, mean LKES before treatment was 4.00±1.49, minimum score being 2 and maximum score being 7; whereas after treatment the score was 3.00±1.12, minimum score being 1 and maximum score being 5. The p-value was 0.0107.

Overall, mean LKES before treatment was 8.15±2.62, minimum score being 5 and maximum score being 14; whereas after treatment the score was 6.05±2.04, minimum score being 3 and maximum score being 10. The p-value was 0.0037.

	BEFORE				**AFTER**				**p VALUE**
	MEAN	**S.D.**	**MIN**	**MAX**	**MEAN**	**S.D.**	**MIN**	**MAX**	
RIGHT SIDE	4.15	1.35	2	7	3.05	1.10	1	5	0.0037
LEFT SIDE	4.00	1.49	2	7	3.00	1.12	1	5	0.0107
TOTAL	8.15	2.62	5	14	6.05	2.04	3	10	0.0037

Table 7b: ***Lund-Kennedy Endoscopic Scoring, intra-group comparison- Test group***

- **SNOT-22 SCORING**

In the following tables, p value is calculated using Student's Paired t Test.

For control group,

Mean score before the treatment was 38.90±12.01 with minimum score being 21, maximum score being 57.

Mean score after the treatment was 25.70±9.21 with minimum score being 12, maximum score being 43.

The p value was 0.0002.

BEFORE				AFTER				p
MEAN	**S.D.**	**MIN**	**MAX**	**MEAN**	**S.D.**	**MIN**	**MAX**	**VALUE**
38.90	12.01	21	57	25.70	9.21	12	43	0.0002

Table 8a: ***SNOT -22 Scoring- Control group***

For test group,

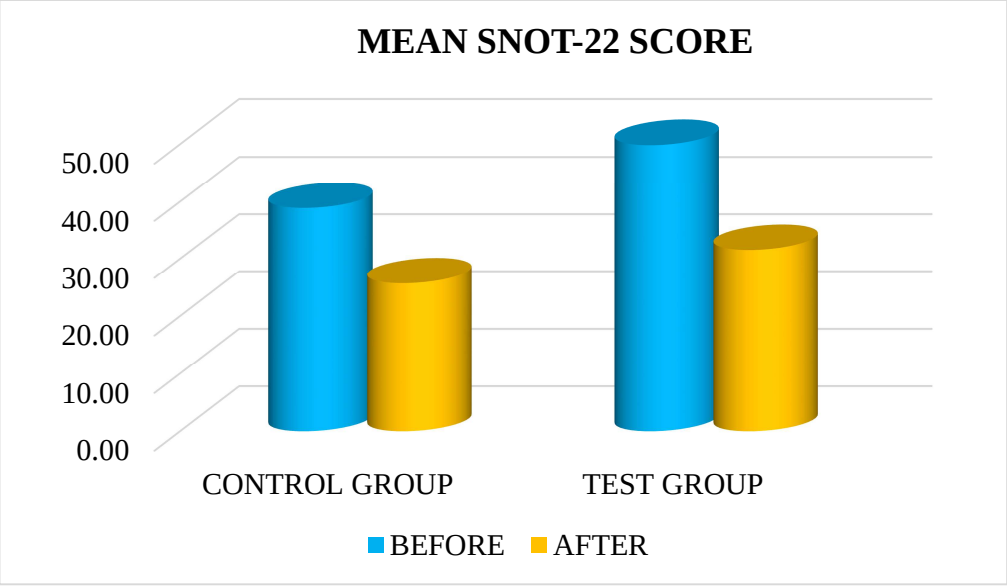
Mean score before the treatment was 49.85±11.38 with minimum score being 30, maximum score being 67.

Mean score after the treatment was 31.55±9.91 with minimum score being 14, maximum score being 51.

The p value was <0.0001.

BEFORE				AFTER				p
MEAN	S.D.	MIN	MAX	MEAN	S.D.	MIN	MAX	VALUE
49.85	11.38	30	67	31.55	9.91	14	51	< 0.0001

Table 8b: ***SNOT -22 Scoring- Test group***



Graph 5: ***Mean SNOT-22 Score***

DISCUSSION

CRS is defined as an inflammatory condition of the nasal cavity and paranasal sinuses that is considered to be the result of mucosal inflammation, which results in swelling and blockage at the sinus ostium. Due to mucus stasis caused by this, bacterial superinfection may follow. The aim of our study was to evaluate the efficacy of supplementing normal saline nasal spray to medical line of treatment for chronic rhinosinusitis.

Saline nasal irrigation enhances the function of the nasal mucosa through a number of physiological processes, including direct mucus cleansing, antigen removal, biofilm removal, removal of inflammatory mediators, and a better mucociliary function.

In our study, 40 CRS patients were included out of which 19 (47.5%) patients were females and 21 (52.5%) patients were males. Male predominance was seen.

In the present study, CRS patients within the age group 18-65 years were included, majority of patients belonging to the age group 25-34 years (35%) and 34-44 years (25%), overall mean age being 35 years. Presence of polyposis was noted in 42.5% of patients.

Our findings showed that each patient had improved post-treatment to some extent in both the groups, more so in the test group. For control group, mean SNOT-22 score before the treatment was 38.90±12.01 whereas after the treatment score was 25.70±9.21 (p value was 0.0002). For test group, mean score before the treatment was 49.85±11.38 whereas mean score after the treatment was 31.55±9.91 (p value was <0.0001).

The majority of symptom improvements were seen in patients with the highest symptom scores, highlighting the significance of the overall SNOT-22 score.. This corresponds with a previous study by Smith TL et al., which has mentioned how those patients who were more seriously impacted showed greater improvement.[73] It's crucial to note that improvements were seen even in the individuals with the lowest pre-operative symptom levels.

Objective confirmation of sino-nasal mucosal inflammation is necessary utilizing nasal endoscopy or diagnostic imaging due to clinical overlap with other frequent diseases. The most popular method for diagnosing medical issues affecting the nose and sinuses at the moment is nasal endoscopy which is a minimally invasive procedure. The LKES method was created in 1995 and is still the most widely used endoscopic scoring system.[74] Despite being created with postsurgical patients in mind, it is still often used to evaluate people who haven't had sinus surgery. According to our findings, all patients in both groups presented with some degree of improvement post-treatment, although a greater improvement was noted in the test group. For control group, mean Lund Kennedy Endoscopic Score before the treatment was 5.35±2.43 whereas after the treatment score was 3.70±1.95 (p value was 0.0116). For test group, mean Lund Kennedy Endoscopic Score before the treatment was 8.15±2.62 whereas mean score after the treatment was 6.05±2.04 (p value was 0.0037).

Since there is evidence that many different factors, such as biofilms, osteitis, allergies, superantigens from Staphylococcus aureus, fungi, etc., contribute to CRS, medical management of CRS includes antimicrobial and anti-inflammatory drugs, steroid nasal sprays as well as oral steroids, nasal irrigations, decongestants,

leukotriene antagonists, management of allergy, and other therapies that are frequently used in the field before going for surgical management.

Nasal saline irrigation (NSI) is a helpful, low-risk therapy that aids in the treatment of CRS when used in conjunction with other methods like surgery and medicine. It is thought to work by thinning mucus, enhancing mucociliary clearance, reducing edema, and lessening the amount of antigens present in the nose and sinus cavities.[75] For these reasons, saline is beneficial in treating rhinitis, acute and chronic sinusitis, and other upper respiratory tract illnesses brought on by inflammatory mediators. Our study concludes that normal saline irrigation reduces symptoms of CRS. It also demonstrates high patient compliance with no reported side effects.

Temperature, added minerals, oligo-elements, and sodium chloride tonicity all have an impact on the composition of nasal saline. Studies show that In CRS or postoperative FESS patients, there is little advantage to utilizing hypertonic saline (HS) over isotonic saline (IS; 0.9%).[76] Additionally, HS is linked to increased patient intolerance and discomfort.[77] When compared to dilute IS saltwater and regular water, an in-vitro investigation found that non-dilute IS seawater solution more efficiently increased ciliary beat frequency and wound repair speed.[78] In a different investigation, Woods et al. evaluated the effects of IS, HS, and low-salt solution on the antibacterial activity of nasal secretions and found that IS caused the antimicrobial activity to decline more significantly.[79] There is no additional benefit to heating the solution, according to Nimsakul et al's analysis of the effect of NSI temperature on mucociliary clearance and nasal patency.[80]

CONCLUSION

Normal saline is beneficial in rhinosinusitis patients by acting locally on the nasal mucosa. It is helpful in clearance of mucous, infected crusts and pro-inflammatory agents via mucous thinning, enhanced mucociliary evacuation from sinuses, reduced edema, and decreased antigen load in the nasal and sinus cavities, hence reducing CRS symptoms.

Hence, from the observations in our study, we conclude that addition of normal saline nasal spray to medical management of chronic rhinosinusitis is efficacious. It is evident through endoscopic as well as symptomatic improvement in patients.

SUMMARY

Supplementation of normal saline nasal spray to medical treatment of chronic rhinosinusitis is beneficial. It supplements in clearance of mucous from nasal cavity and sinuses by reducing local edema and thinning of mucous, hence improving mucociliary clearance which is also aids in reducing antigen load.

BIBLIOGRAPHY

1) Watkinson J., Clarke R. Scott-Brown's Otorhinolaryngology, Head & Neck Surgery. 8th ed. Vol 1. CRC Press; 2018:1025-1069.

2) Smith TL, Kern R, Palmer JN, Schlosser R, Chandra RK, Chiu AG, Conley D, Mace JC, Fu RF, Stankiewicz J. Medical therapy vs surgery for chronic rhinosinusitis: a prospective, multi-institutional study with 1-year follow-up. Int Forum Allergy Rhinol. 2013 Jan;3(1):4-9.

3) DeConde AS, Mace JC, Ramakrishnan VR, Alt JA, Smith TL. Analysis of factors associated with electing endoscopic sinus surgery. Laryngoscope. 2018 Feb;128(2):304-310.

4) Hauptmann G, Ryan MW. The effect of saline solutions on nasal patency and mucociliary clearance in rhinosinusitis patients. Otolaryngol Head Neck Surg 2007; 137:815–821.

5) Paventhan K, Pradeep KR, Ramyashree C. Comparison of nasal douching with isotonic saline versus Ringer lactate in chronic rhinosinusitis: a randomized controlled trial. Int J Otorhinolaryngol Head Neck Surg 2020; 6:1064-8.

6) Huang A, Govindaraj S. Topical therapy in the management of chronic rhinosinusitis. Curr Opin Otolaryngol Head Neck Surg. 2013 Feb;21(1):31-8.

7) Low TH, Woods CM, Ullah S, Carney AS. A double-blind randomized controlled trial of normal saline, lactated Ringer's and hypertonic saline nasal irrigation solution after endoscopic sinus surgery. Am J Rhinol Allergy. 2014;28(3):225-31.

8) Chong LY, Head K, Hopkins C, Philpott C, Glew S, Scadding G, Burton MJ, Schilder AG. Saline irrigation for chronic rhinosinusitis. Cochrane Database Syst Rev. 2016 Apr 26;4(4):CD011995.

9) Fokkens WJ, Lund VJ, Mullol J, Bachert C, Alobid I, Baroody F, et al. EPOS 2012: European position paper on rhinosinusitis and nasal polyps 2012. A summary of Otorhinolaryngologists. Rhinology. 2012;50:1-12.

10) Jain, D., 2017. Comparison of Outcomes of Management of Chronic Rhinosinusitis by Conservative Approach Vs Endoscopic Sinus Surgery with Review of Literature. Journal of Medical Science and Clinical Research, 05(03), pp.19369-19378.

11) Baroody FM, Mucha SM, Detineo M, Naclerio RM. Nasal challenge with allergen leads to maxillary sinus inflammation. J Allergy Clin Immunol. 2008; 121:1126–32.

12) Baroody FM, Mucha SM, de Tineo M, Naclerio RM. Evidence of maxillary sinus inflammation in seasonal allergic rhinitis. Otolaryngol Head Neck Surg : Off J Am Acad Otolaryngol Head Neck Surg. 2012; 146:880–6.

13) Hulse KE, Norton JE, Suh L, et al. Chronic rhinosinusitis with nasal polyps is characterized by B-cell inflammation and EBV-induced protein 2 expression. J Allergy Clinical Immunol. 2013; 131:1075–1083. 1083, e1071–1077.

14) Munoz DelCastillo F, Jurado-Ramos A, Fernandez-Conde BL, et al. Allergenic profile of nasal polyposis. J Investig Allergol Clin Immunol. 2009; 19:110–6.

15) Pearlman AN, Chandra RK, Chang D, et al. Relationships between severity of chronic rhinosinusitis and nasal polyposis, asthma, and atopy. Am J Rhinol Allergy. 2009; 23:145–8.

16) Robinson S, Douglas R, Wormald PJ. The relationship between atopy and chronic rhinosinusitis. Am J Rhinol. 2006; 20:625–8.

17) Lam, K., Schleimer, R. & Kern, R.C. The Etiology and Pathogenesis of Chronic Rhinosinusitis: a Review of Current Hypotheses. *Curr Allergy Asthma Rep* **15**, 41 (2015).

18) Ebbens FA, Fokkens WJ. The mold conundrum in chronic rhinosinusitis: where do we stand today? Curr Allergy Asthma Rep. 2008; 8:93–101.

19) Shin SH, Lee SH, Jeong HS, Kita H. The effect of nasal polyp epithelial cells on eosinophil activation. Laryngoscope. 2003; 113:1374–7.

20) Shin SH, Lee YH, Jeon CH. Protease-dependent activation of nasal polyp epithelial cells by airborne fungi leads to migration of eosinophils and neutrophils. Acta Otolaryngol. 2006; 126:1286–94.

21) Rudack C, Steinhoff M, Mooren F, et al. PAR-2 activation regulates IL-8 and GRO-alpha synthesis by NF-kappaB, but not RANTES, IL-6, eotaxin or TARC expression in nasal epithelium. Clin Exp Allergy : J Br Soc Allergy Clin Immunol. 2007; 37:1009–22.

22) Matsuwaki Y, Wada K, White T, et al. Alternaria fungus induces the production of GM-CSF, interleukin-6 and interleukin-8 and calcium signaling in human airway epithelium through protease-activated receptor 2. Int Arch Allergy Immunol. 2012; 158(Suppl 1):19–29.

23) Bhushan B, Homma T, Norton JE, et al. Suppression of epithelial STAT1 activation by extracts of Aspergillus fumigatus. Am J Respir Cell Mol Biol. 2014

24) Ryan MW. Allergic fungal rhinosinusitis. Otolaryngol Clin N Am. 2011; 44:697–710.

25) Lee CG, Da Silva CA, Dela Cruz CS, et al. Role of chitin and chitinase/chitinase-like proteins in inflammation, tissue remodeling, and injury. Annu Rev Physiol. 2011; 73:479–501.

26) Zhu Z, Zheng T, Homer RJ, et al. Acidic mammalian chitinase in asthmatic Th2 inflammation and IL-13 pathway activation. Science. 2004; 304:1678–82.

27) Ramanathan M Jr, Lee WK, Lane AP. Increased expression of acidic mammalian chitinase in chronic rhinosinusitis with nasal polyps. Am J Rhinol. 2006; 20:330–5.

28) Brook I. The role of bacteria in chronic rhinosinusitis. Otolaryngol Clin N Am. 2005; 38:1171–92.

29) Larson DA, Han JK. Microbiology of sinusitis: does allergy or endoscopic sinus surgery affect the microbiologic flora? Curr Opin Otolaryngol Head Neck Surg. 2011; 19:199–203.

30) Corriveau MN, Zhang N, Holtappels G, et al. Detection of Staphylococcus aureus in nasal tissue with peptide nucleic acid-fluorescence in situ hybridization. Am J Rhinol Allergy. 2009; 23:461–5.

31) Sachse F, Becker K, von Eiff C, et al. Staphylococcus aureus invades the epithelium in nasal polyposis and induces IL-6 in nasal epithelial cells in vitro. Allergy. 2010; 65:1430–7.

32) Tan NC, Foreman A, Jardeleza C, et al. Intracellular Staphylococcus aureus: the Trojan horse of recalcitrant chronic rhinosinusitis? Int Forum Allergy Rhinol. 2013; 3:261–6.

33) Bachert C, Gevaert P, Holtappels G, et al. Total and specific IgE in nasal polyps is related to local eosinophilic inflammation. J Allergy Clin Immunol. 2001; 107:607–14.

34) Bachert C, Zhang N, Patou J, et al. Role of staphylococcal superantigens in upper airway disease. Curr Opin Allergy Clin Immunol. 2008; 8:34–8.

35) Seiberling KA, Grammer L, Kern RC. Chronic rhinosinusitis and superantigens. Otolaryngol Clin N Am. 2005; 38:1215–36.

36) Seiberling KA, Conley DB, Tripathi A, et al. Superantigens and chronic rhinosinusitis: detection of staphylococcal exotoxins in nasal polyps. Laryngoscope. 2005; 115:1580–5.

37) Bernstein JM, Ballow M, Schlievert PM, et al. A superantigen hypothesis for the pathogenesis of chronic hyperplastic sinusitis with massive nasal polyposis. Am J Rhinol. 2003; 17:321–6.

38) Conley DB, Tripathi A, Seiberling KA, et al. Superantigens and chronic rhinosinusitis: skewing of T-cell receptor V beta-distributions in polyp-derived CD4+ and CD8+ T cells. Am J Rhinol. 2006; 20:534–9.

39) Conley DB, Tripathi A, Seiberling KA, et al. Superantigens and chronic rhinosinusitis II: analysis of T-cell receptor V beta domains in nasal polyps. Am J Rhinol. 2006; 20:451–5.

40) Tripathi A, Conley DB, Grammer LC, et al. Immunoglobulin E to staphylococcal and streptococcal toxins in patients with chronic sinusitis/nasal polyposis. Laryngoscope. 2004; 114:1822–6.

41) Wang M, Shi P, Yue Z, et al. Superantigens and the expression of T-cell receptor repertoire in chronic rhinosinusitis with nasal polyps. Acta Otolaryngol. 2008; 128:901–8.

42) Patou J, Gevaert P, Van Zele T, et al. Staphylococcus aureus enterotoxin B, protein A, and lipoteichoic acid stimulations in nasal polyps. J Allergy Clin Immunol. 2008; 121:110–5.

43) Perez Novo CA, Jedrzejczak-Czechowicz M, Lewandowska-Polak A, et al. T cell inflammatory response, Foxp3 and TNFRS18-L regulation of peripheral blood mononuclear cells from patients with nasal polyps-asthma after staphylococcal superantigen stimulation. Clin Exp Allergy : J Br Soc Allergy Clin Immunol. 2010; 40:1323–32.

44) Langier S, Landsberg R, Sade K, Kivity S. Anti-IL-5 immunomodulates the effect of Staphylococcus aureus enterotoxin on T cell response in nasal polyps. Rhinology. 2011; 49:570–6.

45) Van Crombruggen K, Zhang N, Gevaert P, et al. Pathogenesis of chronic rhinosinusitis: inflammation. J Allergy Clin Immunol. 2011; 128:728–32.

46) Foreman A, Jervis-Bardy J, Wormald PJ. Do biofilms contribute to the initiation and recalcitrance of chronic rhinosinusitis? Laryngoscope. 2011; 121:1085–91.

47) Dorrestein PC, Mazmanian SK, Knight R. Finding the missing links among metabolites, microbes, and the host. Immunity. 2014; 40:824–32.

48) Gallo RL. S. epidermidis influence on host immunity: more than skin deep. Cell Host Microbe. 2015; 17:143–4.

49) Borody TJ, Khoruts A. Fecal microbiota transplantation and emerging applications. Nat Rev Gastroenterol Hepatol. 2012; 9:88–96.

50) Abreu NA, Nagalingam NA, Song Y, et al. Sinus microbiome diversity depletion and Corynebacterium tuberculostearicum enrichment mediates rhinosinusitis. Sci Transl Med. 2012; 4 151ra124

51) Roca-Ferrer J, Garcia-Garcia FJ, Pereda J, et al. Reduced expression of COXs and production of prostaglandin E(2) in patients with nasal polyps with or without aspirin-intolerant asthma. J Allergy Clin Immunol. 2011; 128:66–72.

52) Perez-Novo CA, Waeytens A, Claeys C, et al. Staphylococcus aureus enterotoxin B regulates prostaglandin E2 synthesis, growth, and migration in nasal tissue fibroblasts. J Infect Dis. 2008; 197:1036–43.

53) Okano M, Fujiwara T, Haruna T, et al. Prostaglandin E(2) suppresses staphylococcal enterotoxininduced eosinophilia-associated cellular responses dominantly through an E-prostanoid 2-mediated pathway in nasal polyps. J Allergy Clin Immunol. 2009; 123:868–74.

54) Tan BK, Schleimer RP, Kern RC. Perspectives on the etiology of chronic rhinosinusitis. Curr Opin Otolaryngol Head Neck Surg. 2010; 18:21–6.

55) Kern RC, Conley DB, Walsh W, et al. Perspectives on the etiology of chronic rhinosinusitis: an immune barrier hypothesis. Am J Rhinol. 2008; 22:549–59.

56) Wang X, Moylan B, Leopold DA, et al. Mutation in the gene responsible for cystic fibrosis and predisposition to chronic rhinosinusitis in the general population. JAMA. 2000; 284:1814–9.

57) Wang X, Kim J, McWilliams R, Cutting GR. Increased prevalence of chronic rhinosinusitis in carriers of a cystic fibrosis mutation. Arch Otolaryngol Head Neck Surg. 2005; 131:237–40.

58) Antunes MB, Gudis DA, Cohen NA. Epithelium, cilia, and mucus: their importance in chronic rhinosinusitis. Immunol Allergy Clin N Am. 2009; 29:631–43.

59) Chen B, Antunes MB, Claire SE, et al. Reversal of chronic rhinosinusitis-associated sinonasal ciliary dysfunction. Am J Rhinol. 2007; 21:346–53.

60) Zuckerman JD, Lee WY, DelGaudio JM, et al. Pathophysiology of nasal polyposis: the role of desmosomal junctions. Am J Rhinol. 2008; 22:589–97.

61) Rogers GA, Den Beste K, Parkos CA, et al. Epithelial tight junction alterations in nasal polyposis. Int Forum Allergy Rhinol. 2011; 1:50–4.

62) Richer SL, Truong-Tran AQ, Conley DB, et al. Epithelial genes in chronic rhinosinusitis with and without nasal polyps. Am J Rhinol. 2008; 22:228–34.

63) Soyka MB, Wawrzyniak P, Eiwegger T, et al. Defective epithelial barrier in chronic rhinosinusitis: the regulation of tight junctions by IFN-gamma and IL-4. J Allergy Clin Immunol. 2012; 130:1087–96.

64) Den Beste KA, Hoddeson EK, Parkos CA, et al. Epithelial permeability alterations in an in vitro air-liquid interface model of allergic fungal rhinosinusitis. Int Forum Allergy Rhinol. 2013; 3:19– 25.

65) Pothoven KL, Norton JE, Hulse KE, et al. Oncostatin M promotes mucosal epithelial barrier dysfunction, and its expression is increased in patients with eosinophilic mucosal disease. J Allergy Clin Immunol. 2015.

66) Chandra RK, Lin D, Tan B, et al. Chronic rhinosinusitis in the setting of other chronic inflammatory diseases. Am J Otolaryngol. 2011; 32:388–91.

67) Tan BK, Chandra RK, Pollak J, et al. Incidence and associated premorbid diagnoses of patients with chronic rhinosinusitis. J Allergy Clin Immunol. 2013; 131:1350–60.

68) Larsen PL, Tos M. Origin of nasal polyps: an endoscopic autopsy study. Laryngoscope. 2004 Apr;114(4):710-9.

69) Brown CL, Graham SM. Nasal irrigations: good or bad? Curr Opin Otolaryngol Head Neck Surg. 2004 Feb;12(1):9-13.

70) Hopkins C, Gillett S, Slack R, Lund VJ, Browne JP. Psychometric validity of the 22-item Sinonasal Outcome Test. Clin Otolaryngol. 2009 Oct;34(5):447-54

71) Levy JM, Mace JC, Rudmik L, Soler ZM, Smith TL. Low 22-item sinonasal outcome test scores in chronic rhinosinusitis: Why do patients seek treatment? Laryngoscope. 2017 Jan;127(1):22-28.

72) Lund VJ, Kennedy DW. Quantification for staging sinusitis. The Staging and Therapy Group. Ann Otol Rhinol Laryngol Suppl. 1995 Oct;167:17-21.

73) Smith TL, Litvack JR, Hwang PH, Loehrl TA, Mace JC, Fong KJ, et al. Determinants of outcomes of sinus surgery: a multi-institutional prospective cohort study. Otolaryngology--head and neck surgery:official journal of American Academy of Otolaryngology-Head and Neck Surgery. 2010; 142: 55–63.

74) Psaltis AJ, Li G, Vaezeafshar R, Cho KS, Hwang PH. Modification of the Lund-Kennedy endoscopic scoring system improves its reliability and correlation with patient-reported outcome measures. *Laryngoscope*. 2014;124(10):2216-2223.

75) Orlandi RR, Kingdom TT, Hwang PH. International Consensus Statement on Allergy and Rhinology: rhinosinusitis executive summary. Int Forum Allergy Rhinol. 2016;6(suppl 1):S3-S21.

76) Chong LY, Head K, Hopkins C, et al. Saline irrigation for chronic rhinosinusitis. Cochrane Database Syst Rev. 2016;4:CD011995.

77) Kanjanawasee D, Seresirikachorn K, Chitsuthipakorn W, Snidvongs K. Hypertonic saline versus isotonic saline nasal irrigation: systematic review and meta-analysis. Am J Rhinol Allergy. 2018;32:269-279.

78) Bonnomet A, Luczka E, Coraux C, de Gabory L. Nondiluted seawater enhances nasal ciliary beat frequency and wound repair speed compared to diluted seawater and normal saline. Int Forum Allergy Rhinol. 2016;6: 1062-1068.

79) Woods CM, Tan S, Ullah S, Frauenfelder C, Ooi EH, Carney AS. The effect of nasal irrigation formulation on the antimicrobial activity of nasal secretions. Int Forum Allergy Rhinol. 2015;5:1104-1110.

80) Nimsakul S, Ruxrungtham S, Chusakul S, Kanjanaumporn J, Aeumjaturapat S, Snidvongs K. Does heating up saline for nasal irrigation improve mucociliary function in chronic rhinosinusitis? Am J Rhinol Allergy. 2018;32:106-111.

ANNEXURE I

INFORMED CONSENT

"EFFICACY OF NORMAL SALINE NASAL SPRAY ADDED TO STANDARD TREATMENT REGIMEN OF CHRONIC RHINOSINUSITIS: A RANDOMIZED CONTROL TRIAL"

PRINCIPAL INVESTIGATOR: **Dr.**
Post Graduate student
Department of Otorhinolaryngology & HNS

CO-INVESTIGATOR: **Dr.**
Professor
Department of Otorhinolaryngology & HNS

INTRODUCTION AND PURPOSE: The present study is conducted among patients who are undergoing Diagnostic Nasal Endoscopy in Otorhinolaryngology & HNS department in KLE's Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum for symptoms suggesting chronic rhinosinusitis.

PROCEDURE: If you agree to participate in this study, the relevant data will be collected as per the proforma and the final diagnosis will be confirmed.

After getting enrolled in the study, you will be required to fill out a questionnaire addressing symptoms defined for chronic rhinosinusitis and evaluated with Diagnostic Nasal Endoscopy for evaluation of severity of the disease. This will be followed by medical treatment for a period of 3 weeks at the end of which you will be again required to fill out the questionnaire and undergo Diagnostic Nasal Endoscopy.

BENEFITS: The study will help evaluate the efficacy of normal saline nasal spray in conservative treatment of chronic rhinosinusitis.

RISKS: Methods applied to do the study are safe. Any untoward complications will be taken care of by the investigator

COST OF PARTICIPATION: The cost of the Investigation will be borne by the participant.

PRIVACY AND CONFIDENTIALITY: The results of the study may be published in journals for scientific purposes. However your identity will not be revealed. All information collected will be coded so that no one other than the investigator will know your identity.

WITHDRAWAL FROM THE STUDY: You can withdraw from the study at any time if you wish to do so.

AUTHORIZATION TO PUBLISH THE RESULTS: The researcher may use the information gathered from this study for presentation in scientific meetings. However your identity will not be revealed.

QUERIES AND CONTACT: If you have any queries regarding the study, you can contact Dr. _____ without any hesitation on Mobile no: _____and guide Dr. _____ on Mobile no: _____

If you have any query about rights as a research participant you can contact Dr. Harsha Hegde, Chairperson, Jawaharlal Nehru Medical College, IEC & Scientist D, ICMR, National Institute of Traditional Medicine on Mobile no. 9480422500.

CONSENT SUMMARY:

I have been explained all the contents of this consent form in my local language and having understood and clarified all my queries about the study to the best of my knowledge, I hereby give my voluntary consent for participation in the study. I do sign the informed consent form in front of an eyewitness whom I recognize.

Name and Signature/ left thumb impression of the participant:

Name and Signature of the interviewer:

Name and Signature/ left thumb impression of the eyewitness (Relative):

Signature of the guide:

Date:

ANNEXURE II

PROFORMA

EFFICACY OF NORMAL SALINE NASAL SPRAY ADDED TO STANDARD TREATMENT REGIMEN OF CHRONIC RHINOSINUSITIS: A RANDOMIZED CONTROL TRIAL

Date:

O.P. No: IP No:

Name: Age:

Sex: Occupation:

Address: Phone No:

D.O.A D.O.D:

CLINICAL PROFILE:

Chief Complaint:

History of Present Illness:

Past History:

Personal History:

Family History:

Physical Examination:

I) General Physical Examination -

Vital signs:

Pulse-

Blood pressure-

Respiratory Rate-

Pallor

Icterus

Clubbing

Cyanosis

Lymphadenopathy

Oedema

II) ENT Examination

1. **NOSE EXAMINATION**

-External Examination

- Root
- Bridge
- Dorsum
- Alae
- Tip
- Columella
- Vestibule

-Cold spatula test

-Tip Elevation test

-Anterior Rhinoscopy: **RIGHT** **LEFT**

- Mucosa
- Septum
- Floor
- Inferior meatus
- Inferior turbinate

- Middle meatus
- Middle turbinate

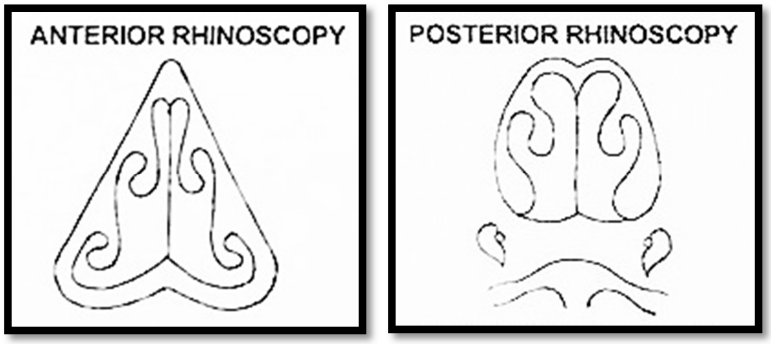
- Cottle's test

-Posterior Rhinoscopy

- Posterior end of septum
- Posterior ends of:

  middle turbinate

  inferior turbinate
- Eustachian tube opening



-Paranasal Sinus Examination

-Smell test

2. EAR EXAMINATION **Right ear** **Left ear**

Pinna

Preauricular area

Post auricular area

External auditory canal

Tympanic membrane

Tuning Fork Test: **Right ear** **Left ear**

-Rinne's test:

-Weber's test:

-Absolute Bone Conduction test:

3. **THROAT EXAMINATION –**

-ORAL CAVITY and OROPHARYNX:

4. **NECK EXAMINATION**

DIAGNOSIS

DIAGNOSTIC NASAL ENDOSCOPY FINDINGS

TREATMENT RECEIVED

Sino-Nasal Outcome Test-22 Questionnaire v4

Below you will find a list of symptoms and social/emotional consequences of your nasal disorder. We would like to know more about these problems and would appreciate you answering the following question to the best of your ability. There are no right or wrong answers, and only you can provide us with this information. Please rate your problems, as they have been over the past two weeks. Thank you for your participation.

Considering how severe the problem is when you experience it and how frequently it happens, please rate each item below on how 'bad' it is by circling the number that corresponds with how you feel using this scale ➔	**No problem**	**Very mild problem**	**Mild or slight problem**	**Moderate problem**	**Severe problem**	**Problem as bad as it can be**
1. Need to blow nose	0	1	2	3	4	5
2. Sneezing	0	1	2	3	4	5
3. Runny nose	0	1	2	3	4	5
4. Cough	0	1	2	3	4	5
5. Post nasal discharge (dripping at the back of your nose)	0	1	2	3	4	5
6. Thick nasal discharge	0	1	2	3	4	5
7. Ear fullness	0	1	2	3	4	5
8. Dizziness	0	1	2	3	4	5
9. Ear pain/pressure	0	1	2	3	4	5
10. Facial pain/pressure	0	1	2	3	4	5
11. Difficulty falling asleep	0	1	2	3	4	5
12. Waking up at night	0	1	2	3	4	5
13. Lack of a good night's sleep	0	1	2	3	4	5
14. Waking up tired	0	1	2	3	4	5
15. Fatigue during the day	0	1	2	3	4	5
16. Reduced productivity	0	1	2	3	4	5
17. Reduced concentration	0	1	2	3	4	5
18. Frustrated/restless/irritable	0	1	2	3	4	5
19. Sad	0	1	2	3	4	5
20. Embarrassed	0	1	2	3	4	5
21. Sense of taste/smell	0	1	2	3	4	5
22. Blockage/congestion of nose	0	1	2	3	4	5

ANNEXURE III

PHOTOGRAPHS

Sino-Nasal Outcome Test-22 Questionnaire v4

Below you will find a list of symptoms and social/emotional consequences of your nasal disorder. We would like to know more about these problems and would appreciate you answering the following question to the best of your ability. There are no right or wrong answers, and only you can provide us with this information. Please rate your problems, as they have been over the past two weeks. Thank you for your participation.

Considering how severe the problem is when you experience it and how frequently it happens, please rate each item below on how 'bad' it is by circling the number that corresponds with how you feel using this scale →	No problem	Very mild problem	Mild or slight problem	Moderate problem	Severe problem	Problem as bad as it can be
1. Need to blow nose	0	1	2	3	4	5
2. Sneezing	0	1	2	3	4	5
3. Runny nose	0	1	2	3	4	5
4. Cough	0	1	2	3	4	5
5. Post nasal discharge (dripping at the back of your nose)	0	1	2	3	4	5
6. Thick nasal discharge	0	1	2	3	4	5
7. Ear fullness	0	1	2	3	4	5
8. Dizziness	0	1	2	3	4	5
9. Ear pain/pressure	0	1	2	3	4	5
10. Facial pain/pressure	0	1	2	3	4	5
11. Difficulty falling asleep	0	1	2	3	4	5
12. Waking up at night	0	1	2	3	4	5
13. Lack of a good night's sleep	0	1	2	3	4	5
14. Waking up tired	0	1	2	3	4	5
15. Fatigue during the day	0	1	2	3	4	5
16. Reduced productivity	0	1	2	3	4	5
17. Reduced concentration	0	1	2	3	4	5
18. Frustrated/restless/irritable	0	1	2	3	4	5
19. Sad	0	1	2	3	4	5
20. Embarrassed	0	1	2	3	4	5
21. Sense of taste/smell	0	1	2	3	4	5
22. Blockage/congestion of nose	0	1	2	3	4	5

Fig. 1 : ***Sin-Nasal Outcome Test-22 Questionnaire***

Criteria of Assessment	Scores		
	0	1	2
Polyps in middle meatus	Absent	Restricted to middle meatus	Beyond middle meatus
Discharge in middle meatus	Absent	Thin and clear discharge	Thick and purulent discharge
Edema of the middle meatus	Absent	Mild-moderate	Moderate-severe
Scarring in middle meatus	Absent	Mild-moderate	Moderate-severe
Crusting in middle meatus	Absent	Mild-moderate	Moderate-severe

Fig. 2: ***Lund-Kennedy Endoscopic Scoring***

ANNEXURE IV

KEY TO MASTERCHART

GLOSSARY	ABBREVIATIONS
-	Absent
+	Present
C	Control group
T	Test group
LKES	Lund Kennedy Endoscopic Scoring
R	Right nasal cavity
L	Left side nasal cavity
T	Total
SNOT-22	Sino-Nasal Outcome Test-22

ANNEXURE V

MASTERCHART

S. no	Name	Age	Sex	Symptom duration	Presence of Polyposis	Study Group (C/T)	LKES						SNOT 22	
							Before			After				
							R	L	T	R	L	T	Before	After
1.	Parvati J	61	F	3 months	-	C	1	2	3	1	1	2	33	22
2.	Pratibha Soloni	23	F	7 months	-	C	1	2	3	1	1	2	37	27
3.	Akshata Kelaginamani	30	F	5 years	+	T	4	2	6	3	2	5	60	30
4.	Manjunath Sagarad	29	M	4 years	+	T	3	3	6	3	2	5	31	14
5.	Preeti Sinkad	33	F	5 months	-	C	2	2	4	1	1	2	21	13
6.	Basavanand Bagi	40	M	3 months	-	C	2	2	4	1	1	2	24	12
7.	Annapurna Yattur	45	F	4 months	+	C	2	3	5	2	2	4	31	17
8.	Priyanka Nikam	26	F	6 months	+	T	2	3	5	1	3	4	45	28
9.	Nagappa Yallatti	30	M	20 years	+	T	3	2	5	2	2	4	53	27
10	Pavitra Jadappagol	22	F	3 months	+	C	3	5	8	2	3	5	40	26
11	Gururaj Khanapu	23	M	7 years	+	C	2	1	3	2	0	2	27	19
12	Manisha Patil	28	F	2 years	+	C	4	3	7	3	2	5	51	30
13	Raziya Devadi	38	F	3 years	+	T	3	3	6	3	3	6	62	51
14	Beauti Dey	27	F	2 years	-	C	2	2	4	1	2	3	55	40
15	Shivaprasad Huddar	19	M	2 years	-	T	4	3	7	2	3	5	44	27
16	Lagamavva Patil	48	F	9 months	-	C	2	2	4	1	2	3	30	22
17	Anil Sutar	52	M	3 years	+	T	6	6	12	5	4	9	55	38
18	Suresh Talwar	59	M	20 years	+	C	6	3	9	5	2	7	54	43
19	Kousar Kalibhai	41	F	10 years	-	T	4	3	7	2	1	3	52	35
20	Bhimangouda Patil	63	M	10 years	+	T	7	7	14	5	5	10	50	38
21	Mallesh Shegunashi	19	M	4 months	-	T	4	4	8	2	2	4	41	22

22	Reshma Gawade	25	F	3 years	-	C	3	2	5	2	1	3	42	31
23	Kaveri Patil	24	F	8 years	+	C	6	4	10	5	3	8	52	32
24	Pankaj Mathapati	40	M	3 months	-	T	3	4	7	2	2	4	37	23
25	Bhimangouda Paoguda	32	M	3 years	-	T	5	5	10	3	3	6	55	42
26	Basangouda Patil	39	M	4 years	-	C	2	2	4	1	2	3	57	38
27	Laxmi Deshpande	37	F	2 years	-	C	2	2	4	1	1	2	41	20
28	Appasab Davane	35	M	25 years	+	T	6	6	12	4	5	9	60	37
29	Pavankumar Kadakol	29	M	5 months	-	T	5	2	7	4	2	6	67	36
30	Nilofer Ankalgi	28	F	1 year	-	C	2	2	4	2	1	3	23	17
31	Ravichandra Kadam	40	M	1 year	+	T	5	5	10	4	4	8	64	46
32	Jayprakash Ghorpade	35	M	10 years	-	C	5	6	11	3	4	7	52	32
33	Basavaraj Ganvi	28	M	1 year	-	T	2	3	5	2	2	4	30	16
34	Suhas Navajekar	22	M	4 years	-	C	2	2	4	1	1	2	33	21
35	Shobha Huddar	39	F	1 year	-	C	4	3	7	3	2	5	49	36
36	Maruti Gundlur	50	M	5 years	-	T	5	6	11	4	4	8	60	40
37	Jaya Vaval	34	F	3 years	+	T	5	4	9	4	4	8	57	36
38	Suresh Kokatanur	22	M	2 years	+	T	4	5	9	3	4	7	39	23
39	Savita Lokale	30	F	6 months	-	C	2	2	4	1	1	2	26	16
40	Ramkrishna Yalashetti	62	M	5 months	-	T	3	4	7	3	3	6	35	22