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ORIGINAL RESEARCH

Retrospective Evaluation of Hypertrophy of Salpingopharyngeal Fold in OSA at a Tertiary Care Hospital

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ABSTRACT

Introduction: The most common cause of sleep disordered breathing is reported to be the obstructive sleep apnoea. It could possibly lead to serious physiological, social and few neurocognitive disorders. Since the reported prevalence rate is higher, OSA has often been misdiagnosed and unnoticed.

Materials and Methodology: This study was designed as a retrospective analysis of patients reported with the polysomnographic confirmation of obstructive sleep apnoea. Those patients included in this study had undergone Drug Induced Sleep Endoscopy (DISE) using flexible video rhino-laryngoscope under BISTM monitoring which is reported to be a valid bi-spectral index monitor in measuring the depth of sedation. Drugs like dexmedetomidine and midazolam was initially used in titrating doses in order to maintain BISTM score in the range of 60–80. The DISE video data of 189 patients was assessed by two ENT surgeons individually. Each of them was instructed to grade the hypertrophy of the salpingopharyngeal fold as Grade 0 having normal anatomy, Grade 1 being hypertrophy causing partial obstruction and Grade 2 being hypertrophy which is reportedly responsible for complete obstruction of lateral pharyngeal wall. Data of 160 patients of the 189 had 100% agreement between the two expert surgeons while grading the hypertrophy. Thus, the data of 160 patients was included in the study for further comparison and analyses. The patient's data was divided into two groups. Group 1 included 110 patients who had isolated palatal level obstruction whereas group 2 constituted 50 patients affected with sleep apnoea due to obstruction at multiple levels BMI of the patient was calculated. There was no significant difference between the two groups for age ($p<0.05$), AHI ($p<0.05$) and BMI ($p<0.05$) Shapiro Wilks test of Normality was applied to check for the distribution of the data, non-normal distribution the data was subjected to non-parametric analyses.

Results: It was observed that 80/160 patients had hypertrophy of the SPF, as reported by the experts, amounting to 50%. Out of the 80 patients, 36% of the patients had grade 1 hypertrophy of the salpingopharyngeal fold while 14% had grade 2. Mann Whiteny U test showed that there was no significant difference between the two groups for AHI for grade 0 hypertrophy ($Z = 1.7$, $p = 0.13$), for grade 1 hypertrophy ($Z = 1.41$, $p = 0.19$) and grade 2 hypertrophy ($Z = 0.84$, $p = 0.49$), thus the data of the two groups were

combined to find the effect of hypertrophy on AHI. Table 1 shows the AHI values of the patients with SPF hypertrophy across groups. Kruskals Wallis H test showed that there occurred significant increase in the AHI scores as the hypertrophy increased (X^2 = 12.27, p = 0.005). Mann whitney U test showed that patient with no hypertrophy (grade 0) had significantly lesser AHI values as compared to patients with grade 1 (Z = 3.008 p = 0.004) and grade 2 hypertrophy (Z = 2.655 p = 0.009). Out of 160 patients, 67 patients had BMI > 30 and 93 of them had BMI < 30. Mann whitney U test showed that patients with high BMI values had higher AHI as compared to those having low BMI (Z = 3.52, p = 0.00). Spearman's correlation showed that there was significant increase in the AHI with increase in BMI (rs = 0.299 p = 0.000).

Conclusion: Hence, we would like to conclude that though vestigial / non-functional, hypertrophy of SPF significantly increases AHI in patients with OSA. The presence of SPF hypertrophy could significantly be attributed to the severity of the obstruction, attributing to lateral collapse at the upper retropalatal level and also significantly increases AHI values. Therefore, it is advisable to reconsider the management of the SPF hypertrophy while surgically dealing with OSA.

Keywords: OSA, Salpingopharyngeal Fold, Hypertrophy, BMI.

INTRODUCTION

Obstructive Sleep Apnoea (OSA) is the most common cause of sleep disordered breathing causing sleep disorder[1], which lead to a scope ofserious physiological, social and neurocognitive disorders. Though the prevalence rate is higher, it is often unknown and misdiagnosed.[1]According to the epidemiological study it is been reported that a prevalence rate of 2–9% in case of women and 4–24% in case of men and higher prevalence rate of 40% for overweight people.[2, 3] This conditionis found to impairs the quality of life.[4] and lead to greater number of motor vehicle accidents by 2–3 folds.[5]OSA is associated with other systemic illness like hypertension, diabetes mellitus, coronary artery disease, stroke and mortality.[6] The management of OSA is either non-surgical or surgical, which renders a vital to improve the physiological complications and alleviate the quality of life of patient. Symptoms like snoring and sleep apnoea are examined with the help of overnight polysomnography by Drug Induced Sleep Endoscopy (DISE) which has the added advantage of mimicking the upper airway collapse which occurs during physiological sleep[7] and mayprovides an opportunity to notice directly and define the pathophysiology. Recently, DISE has proved to be the most reliable standard procedurein determining the level, number and severity of the obstruction site in sleep apnoea patients[8]and has effectively proven to be safe, diagnostic and influential in decision making before surgery.[9] Non-surgical treatment mainly comprises of the use of Continuous Positive Airway Pressure (CPAP). CPAP therapy, has made itsimpact in improving clinical symptoms, quality of life and potential consequences associated with OSA, the effectiveness of same is strongly associated to patient compliance.[10] Recent studies have observed CPAP compliance of only 35.3% over a period 12 months of usage.[11]The first description of uvulo-palato-pharyngoplasty (UPPP) by *Fujita* et al[12], surgical options and techniques for obstructive sleep apnoea keep on upgrading constantly. The currently available surgical treatment choices include surgery of nose, palate, base of tongue, epiglottis, maxilla and mandible. Though there is presence of wide range of armamentarium, dissatisfactory results have been reported frequently.[13,14] Special mention regarding our observation about the presence and need for the management of SPF hypertrophy is postulated to aid as an alternative procedure in reducing the failure rates effectively. Hence, the present study scopes at reporting the incidence of SPF hypertrophy on DISE in patients affected with OSA.

MATERIALS AND METHODOLOGY

This study was designed as a retrospective analysis of patients reported with the polysomnographic confirmation of obstructive sleep apnoea. Those patients included in this study had undergone Drug Induced Sleep Endoscopy (DISE) using flexible video rhino-laryngoscope under BISTM monitoring which is reported to be a valid bi-spectral index monitor in measuring the depth of sedation.

Drugs like dexmedetomidine and midazolam was initially used in titrating doses in order to maintain BISTM score in the range of 60–80. The DISE video data of 189 patients was assessed by two ENT surgeons individually. Each of them was instructed to grade the hypertrophy of the salpingopharyngeal fold as Grade 0 having normal anatomy, Grade 1 being hypertrophy causing partial obstruction and Grade 2 being hypertrophy which is reportedly responsible for complete obstruction of lateral pharyngeal wall. Data of 160 patients of the 189 had 100% agreement between the two expert surgeons while grading the hypertrophy. Thus,the data of 160 patientswas included in the study for further comparison and analyses. The patient's data was divided into two groups. Group 1 included 110 patients who had isolated palatal level obstruction whereas group 2 constituted 50 patients affected with sleep apnoea due to obstruction at multiple levels BMI of the patient was calculated. There was no significant difference between the two groups for age ($p<0.05$), AHI ($p <0.05$) and BMI ($p<0.05$) Shapiro Wilks test of Normality was applied to check for the distribution of the data, non-normal distribution the data was subjected to non-parametric analyses.

RESULTS

It was observed that 80/160 patients had hypertrophy of the SPF, as reported by the experts, amounting to 50%. Out of the 80 patients, 36% of the patients had grade 1 hypertrophy of the salpingopharyngeal fold while 14% had grade 2.

Mann Whiteny U test showed that there was no significant difference between the two groups for AHI for grade 0 hypertrophy ($Z = 1.7$, $p = 0.13$), for grade 1 hypertrophy ($Z = 1.41$, $p = 0.19$) and grade 2 hypertrophy ($Z = 0.84$, $p = 0.49$), thus the data of the two groups were combined to find the effect of hypertrophy on AHI.

Table 1 shows the AHI values of the patients with SPF hypertrophy across groups. Kruskals Wallis H test showed that there occurred significant increase in the AHI scores as the hypertrophy increased ($X^2 = 12.27$, $p = 0.005$). Mann whitney U test showed that patient with no hypertrophy (grade 0) had significantly lesser AHI values as compared to patients with grade 1 ($Z = 3.008$ $p = 0.004$) and grade 2 hypertrophy ($Z = 2.655$ $p = 0.009$).

Out of 160 patients, 67 patients had BMI>30 and 93 of them had BMI <30. Mann whitney U test showed that patients with high BMI values had higher AHI as compared to those having low BMI ($Z = 3.52$, $p = 0.00$). Spearman's correlation showed that there was significant increase in the AHI with increase in BMI ($rs = 0.299$ $p = 0.000$).

Kruskal Wallis H test showed that there was no significant difference for the BMI across patients having different grade of SPF hypertrophy [$X^2(2) = 2.77$, $p = 0.46$]. There was no significant relation between BMI and SPF (rs- = 0.021, p [0.06).

Kruskal Wallis H test showed that there was no significant difference for the BMI across patients having different grade of SPF hypertrophy [v2 (2) = 2.15, p = 0.89]. There was no noticeable significant relation between BMI and SPF (r_s- = 0.039, p [0.05).

Table 1: AHI score of patients with SPF hypertrophy (Agrawals grading) across groups Grade – 0: Group 1 (110) and Group 2(50)

X	M	SD
37.72	40 (n=61)	25.23
34.65	38.2 (n=21)	21.65

Grade – 1: Group 1(110) and Group 2(50)

X	M	SD
49.35	52 (n=34)	25.23
46.6	51 (n=25)	25.95

Grade – 2: Group 1(110) and Group 2(50)

X	M	SD
55.62	60.45 (n=15)	25.13
55.34	57.24 (n=4)	28.32

0, normal anatomy; 1, moderate hypertrophy; 2, severe hypertrophy; X, mean; M, median; SD, standard deviation

DISCUSSION

Anatomically, the salpingopharyngeal muscle is a slender one which has its origin from the medial and inferior borders of the tubal cartilage runs through slips of muscular and tendinous fibres. The muscle then takes the course posteroinferiorly to join as a fan-shaped insertion into the palatopharyngeus muscle located at the junction of the velum and lateral pharyngeal wall.[15] Salpingopharyngeal fold is formed by a raised ridge of mucous membrane extending from the lower end of the tubal elevation along the wall of the pharynx which overlies the salpingopharyngeus muscle.[15]With the data available in the present literature, the major function assigned to the salpingo-pharyngeusmuscle, is bounded to closing the eustachian tube when open,[16] opening of the Eustachian tube when closed,[17] elevating the lateral wall of the oropharynx[18] and elevating the velum[27]making it vestigial, under developed and probably non-functional.[19,20] Various studies done over the past decades have reported SPF with minimal function.

Huge array of professionals are often in dilemmatic state when it comes in treating OSA, owing to its complexity in its clinical presentation and various surgical options available nowadays.[21] Retropalatal obstruction is observed as the most frequent site of obstruction in patients with OSA.[14,22] It can be seen as an anteroposterior collapse because of the soft palate, lateral collapse by the lateral pharyngeal wall and circular as a combined chance. A missing entity in this could be noticeably the SPF which is prevalent but often unrecognised. SPF plays a major role in being additive to the obstruction due to the lateral wall collapse in patients with OSA. A number of treatment options have been designed for the treatment which aims at the managing the palate and not the retropalatal region.[14]Since dealing with isolated palatal obstruction with the neglection of the retropalatal SPF hypertrophy is partial treatment that couldpossibly leads to the failure or incomplete surgery with patient could have apnoea and hypopneas as observedin number of studies.[23]

The AHI recorded a significant increase with the increase in hypertrophy as well. This provides a fact to the possible role of SPF hypertrophy in patients affected with OSA. The increased AHI and hypertrophy wereobserved in patients having multiple level obstruction too. Hence it has been quoted that BMI has norelation with the presence of hypertrophy which indicates its value as an independent contributor to increase in AHI and the severity of the OSA. Thus, the study and research on the role of SPF hypertrophy needs to be paid more attention than it is usually given. Till now as per our understanding, there has been no study which have reported structural changes of SPF in patients with OSA however in a study presented by the *Agrawal* V et al[24], it was proposed that channelling of the SPF in grade 1 and 2 with coblation plasma wand has proved significantly for the better post-operative outcomes. Hence the management of these patients must be consideredfor the role of SPF in the development of OSA and its following channelling if required must be sorted out.

CONCLUSION

Hence, we would like to conclude that though vestigial / non-functional, hypertrophy of SPF significantly increases AHI in patients with OSA. The presence of SPF hypertrophy could significantlybe attributed to the severity of the obstruction, attributing to lateral collapse at the upper retropalatal level and also significantly increases AHI values. Therefore, it is advisable to reconsider the management of the SPF hypertrophy while surgically dealing with OSA.

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