



Efficacy of Intranasal Steroid Spray in the Treatment of Post Adenoidectomy Recurrence

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ABSTRACT

Objectives: To evaluate the efficacy of intranasal steroid in the treatment of recurrent subjective and objective upper airway obstruction secondary to adenoid hypertrophy following adenoidectomy

Materials and methods: 60 children aged 5-12 years, diagnosed with nasopharyngeal obstruction secondary to recurrent adenoid hypertrophy (symptoms > 3 months) post-adenoidectomy, confirmed radiographically, were included in a prospective comparative study. Nasal obstructive symptoms and radiographic adenoid size were scored. All patients received intranasal mometasone furoate spray for 12 weeks. Follow up was done at the end of 2, 12 weeks, 6, 12 months. Symptoms were recorded, scored and totalled on all visits. Lateral nasopharynx X-ray was repeated at 12 weeks and 1 year.

Results: 33 males (55%) and 27 females (45%) met the inclusion criteria, with a male: female ratio of 1:1. Mean age of the study patients was 7.85 ± 2.36 years. Mean total symptom score prior to initiation with intranasal steroid therapy and at 2 weeks, 12 weeks, 6 months and 1 year following initiation of steroid therapy was 7.11, 6.73, 0.91, 0.92 and 0.93 respectively, with a decrease in individual mean snoring, nasal obstruction and rhinorrhoea scores. 64.5% of patients showed a mild, statistically insignificant decrease in symptoms at 2 weeks. 88% showed a statistically significant ($p < 0.001$) decrease in symptoms and radiographic adenoid size at 12 weeks, which was sustained over the remainder of the study period. Correlation was seen between symptoms and radiographic adenoid size. No complications were seen following intranasal steroid therapy in any of the patients.

Conclusion: Intranasal steroid spray is safe and effective in the treatment of recurrent nasal obstruction secondary to adenoid hypertrophy following adenoidectomy. Maintenance therapy at low doses can be safely continued over long periods.

Keywords: Adenoidectomy; Adenoids; Anti-Inflammatory Agents; Nasal Obstruction; Nasopharynx/radiography

INTRODUCTION

Adenoid is one of the important parts of the Waldeyer's ring that is located in the nasopharyngeal area. Because of its location near the posterior choanae and Eustachian tube, it can be the etiology of many health problems of childhood^[1].

Abnormally large adenoids have been attributed to recurrent acute upper respiratory tract infection, although it has been suggested that allergic episodes also result in adenoidal hypertrophy^[2]. Enlarged adenoids can cause harmful effects like nasal obstruction with snoring (obstructive sleep apnoea), otitis media with effusion, facial maldevelopment and failure to thrive^[1,2].

The definitive treatment of upper airway obstruction due to adenoid hypertrophy is surgical treatment with adenoidectomy, although it sometimes produces only short term benefits. The major difficulties associated with incomplete removal of adenoids surgically are due to the presence of lymphoid tissue in the pharyngeal recess and bulging adenoidal tissue into the posterior choanae^[3]. Adenoid "re-growth" is a poorly understood phenomenon, which may be the result of the same presumed causes for primary adenoid hyperplasia^[4]. Medical management of enlarged adenoids includes measures bringing about symptomatic relief, and intranasal steroid spray, as systemic absorption and thus side effects are less with the aerosol form than with oral steroid. However, nonsurgical treatment is considered only in non severe cases with lower degrees of airway obstruction^[3] and those in

whom surgery is contraindicated due to associated morbidity^[5].

Literature has shown that while numerous studies have been conducted on the role of intranasal steroids in the primary treatment of adenoid hypertrophy, little attention has been paid to their role secondarily in the treatment of recurrent nasal obstructive symptoms following surgical treatment of adenoid hypertrophy. Thus this study was undertaken with the aim of evaluating the efficacy of intranasal steroid in the treatment of recurrent subjective and objective upper airway obstruction secondary to adenoid hypertrophy following adenoidectomy.

MATERIALS AND METHODS

A prospective observational study was conducted from January 2011 to October 2014. 60 children of both sexes aged 5-12 years, diagnosed with nasopharyngeal obstruction secondary to recurrent adenoid hypertrophy post adenoidectomy (symptomatic for more than 3 months), confirmed radiographically, were included in the study following parental consent. Those with tonsillar hypertrophy, anatomical abnormality or other pathology causing nasal obstruction, allergic rhinitis, use of intranasal or systemic steroids within the last 1 year, use of any intranasal medication within the previous 2 weeks of entering the study, acute upper respiratory tract infection or inflammatory disease of the airway within 2 weeks of entering the study, history of epistaxis, history suggestive of gastro-oesophageal reflux disease, immunodeficiency disorders, hypersensitivity to mometasone furoate,

craniofacial, neuromuscular or genetic disorders and attrition to follow up were excluded from the study.

Detailed history was obtained followed by general and ENT examination. Obstructive symptoms were scored as follows: Snoring (0 = absent, 1 = 1–2 night(s) per week, 2 = 3–5 nights per week, 3 = 6–7 nights per week), nasal obstruction/chronic mouth breathing (0 = absent, 1 = one fourth to one half of a day, 2 = one half to three fourths of a day, 3 = three fourths to all of the day), nasal discharge (0 = absent, 1 = mild, 2 = moderate, 3 = severe). All scores were summed to obtain an overall symptom score for each patient. Lateral radiograph of the nasopharynx was performed in all cases. Adenoid thickness compared with the rest of the airway was defined as the perpendicular distance from the pharyngeal tubercle to the highest convexity of the adenoid tissue. The ratio of airway to adenoid thickness was calculated and the amount of obstruction was scored as follows: 0 = 0–25%, 1 = 25–50%, 2 = 50–75%, and 3 = 75–100%.

All patients were started on intranasal mometasone furoate spray, 1 puff (50 mcg) in each nostril every morning for 12 weeks. Follow up was done at the end of 2 weeks, 12 weeks, 6 months and 1 year. Symptoms were recorded, scored and totalled on all visits. Lateral X-ray of the nasopharynx was repeated at 12 weeks and 1 year.

Data was analysed using descriptive and inferential statistical methods. Results on continuous measurements are presented on Mean \pm SD (Min-Max) and results on categorical

measurements are presented in Number (%). Significance was assessed at 5% level of significance. Student t test (two tailed, dependent) has been used to find the significance of study parameters on a continuous scale within each group. Chi-square/ Fisher Exact test has been used to find the significance of study parameters on categorical scale between two or more groups. Statistical software SPSS 15.0 was used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc.

RESULTS

33 males (55%) and 27 females(45%) met the inclusion criteria, with a male:female ratio of 1:1. Mean age of the study patients was 7.85 ± 2.36 years. The mean time interval between adenoidectomy and presentation with nasal obstructive symptoms was 4.61 ± 1.2 years.

Prior to initiation with intranasal steroid therapy, all participants suffered from all symptoms. Mean snoring, nasal obstruction and rhinorrhoea scores were 2.48, 2.46 and 2.16 respectively. After 2 weeks of steroid therapy, a decrease in symptoms was seen in 64.5% of cases and the mean symptom scores for snoring, nasal obstruction and rhinorrhoea were 2.36, 2.35 and 2.02 respectively. However this decrease was not statistically significant.

At the end of 12 weeks, immediately following cessation of intranasal spray, improvement in symptoms was seen in 88.3% of cases with a significant ($p < 0.001$) reduction in mean snoring, nasal obstruction and rhinorrhoea scores to 0.32, 0.33 and 0.27 respectively. At the end of 6 months

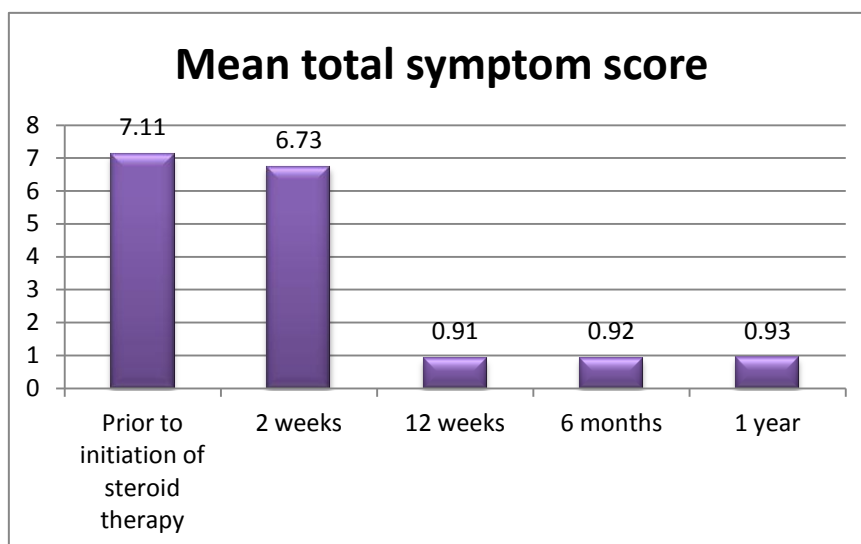
and 1 year, no further significant difference was noted as 87.5% of cases remained relieved of symptoms with a mean snoring, nasal obstruction and rhinorrhoea score of 0.33, 0.34 and 0.29 respectively on both visits. However symptoms remained statistically significantly ($p < 0.001$) better than prior to initiation with steroid therapy. (Graph 1)

Correlation was seen between symptoms and adenoidal size on lateral radiograph of the nasopharynx. Moderate to severe obstruction of

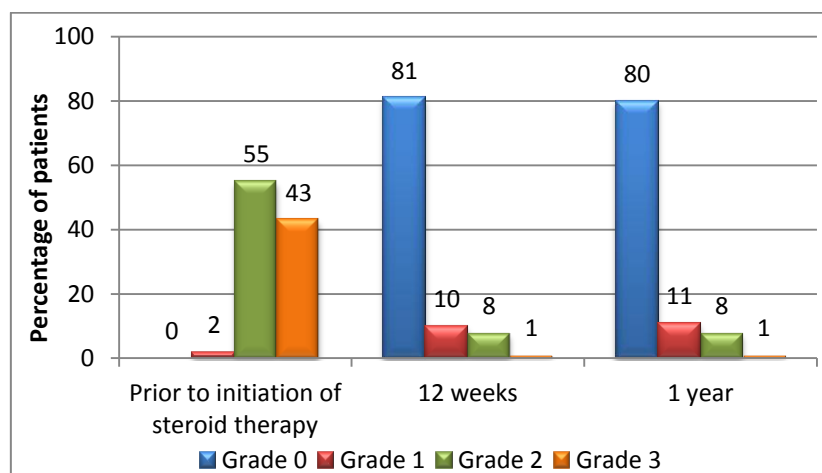
the nasopharyngeal airway was seen on lateral radiograph of the nasopharynx in all patients preoperatively. A significant ($p < 0.001$) reduction in adenoid size was seen in 88% of cases at the end of 12 weeks. At the end of the study period, at 1 year no further significant change in tissue mass was seen; however reduction in adenoid tissue mass remained significantly ($p < 0.001$) better than at the time of enrolment in the study. (Graph 2)

No complications were seen following the use of intranasal steroid in any of the patients.

Graph 1 – Analysis of mean total symptom score



Graph 2 – Analysis of adenoid size on lateral radiograph of nasopharynx



DISCUSSION

In our study, only those with a radiographically demonstrable adenoid pad to which symptoms of nasal obstruction were attributable, were included. A study by Buchinsky et.al failed to demonstrate a new obstructing adenoid pad after adenoidectomy^[3], while, on the contrary, Joshua et.al reported the presence of adenoid “re-growth” after adenoidectomy that causes nasal obstruction which accounts for 3% of patients with persistent post-adenoidectomy symptoms^[6]. However, the incidence of revision adenoidectomy remains low at 0.55 to 0.8%. Inadequate removal of adenoid tissue at adenoidectomy and failure to address the underlying cause for adenoid hyperplasia are reasons proposed for possible adenoid re-growth^[4].

Studies by Monroy et.al^[4] and Emerick et.al^[7] found that symptoms of adenoid regrowth was observed at a mean age of about 7.8 years and 7.2 years respectively, which was similar to the mean age of patients in our study i.e 7.85 ± 2.36 years. The mean time interval between adenoidectomy and presentation with nasal obstructive symptoms in our study was 4.61 years, which was comparable to the mean interval of 4.2 years between primary adenoidectomy and revision adenoidectomy in the study by Monroy et.al^[4].

Successful use of intranasal steroid treatment in children with adenoid hypertrophy was introduced by Demain and Goetz. Although it is not yet clear by which mechanisms steroids reduce the nasal airway obstruction, there are some theories such as the anti-inflammatory effect of steroids that

help to reduce adenoidal and nasopharyngeal inflammation^[8].

Significant efficacy of various nasal steroids such as mometasone, beclomethasone, flunisolide in improving nasal obstruction symptoms and reducing adenoid size has been demonstrated^[9]. Our study aimed to evaluate the effectiveness of intranasal mometasone furoate spray in the treatment of recurrent subjective and objective nasal obstruction secondary to adenoid hypertrophy following adenoidectomy. Mometasone was chosen as this drug had been reported previously not to cause any adverse effects on growth and hypothalamic pituitary adrenal axis. Also, the systemic availability of the drug after topical administration is lower than that of other steroids^[10].

In our study, the duration of steroid therapy was 12 weeks. The duration of treatment with intranasal steroids in previous studies varied from 8 to 24 weeks. None of these trials established the optimal duration of treatment in children. The effects are expected after 2 weeks of the initiation of the treatment as described by Criscuoli et.al^[11], as was seen in our study with 64.5% of patients showing a mild, statistically insignificant improvement in symptoms. As noted in previous studies, our study as well did not show any adverse effects with steroid therapy such as epistaxis, nasal burning sensation or allergy.

In our study, it was observed that 88% of cases showed significant improvement in both symptoms and adenoid size on X-ray of the nasopharynx after 12 weeks of treatment with intranasal steroid spray, and this effect remained

sustained over the entire 1 year follow up on both subjective assessment of symptoms and objective evaluation of adenoid size.

Berlucchi et.al. in a study used mometasone furoate nasal spray for over 15 months in children with adenoid hypertrophy without any complications and with an improvement in symptoms in 77.7% of patients^[12]. In contrast, Mohebbi et.al in a study assessing the effect of mometasone furoate on children with adenoid hypertrophy, failed to demonstrate any significant improvement in symptoms following 3 months of steroid therapy^[5]. Similarly, Lepcha et.al, did not find any significant efficacy of intranasal steroids in improving nasal blockage, nasal discharge, or snoring, although a fivefold reduction in adenoid size was observed in intranasal steroid group when compared with the placebo group. However, this difference did not reach a statistical significance^[2]. Chadha et.al concluded that long term maintenance therapy with low dose steroid is needed if symptom relief is to persist^[9].

CONCLUSION

Intranasal steroid spray is safe and effective in the treatment of recurrent nasal obstruction secondary to adenoid hypertrophy following adenoidectomy. Maintenance therapy at low doses can be safely continued over long periods.

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